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PATHOLOGY OF DIRECT AIR-BLAST INJURY

Thomas L. Chiffelle

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Technical Progress Report
on
Contract No. DA-49-146-XZ-055

This work, an aspect of investigations dealing with the Biological Effects of Blast from Bombs, was supported by the Defense Atomic Support Agency of the Department of Defense.

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Lovelace Foundation for Medical Education and Research
Albuquerque, New Mexico
April 1966

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PREFACE

This manuscript was written from lecture notes and illustrative material prepared and presented to the Allied Command – Atlantic Medical Officers' Symposium, Biomedical Nuclear Weapons Effects Briefings, October 29-30, 1963, held at the National Naval Medical Center, Bethesda, Maryland, and sponsored by the Defense Atomic Support Agency. The lecture, for the most part, dealt with the pathologic anatomy of blast injury. Brief discussions of important points of clinical and physiologic observations in blast injury were included for practical correlation with anatomical findings. The final discussion of sequelae of blast injury has been expanded slightly from that of the original lecture for purposes of clarity and completeness.

The source of information was derived principally from the published studies of blast injury effects by the English and German investigations during World War II, from personal experience of the author in field studies of nuclear weapons effects (supported by Civil Effects Branch of the Division of Biology and Medicine of the U. S. Atomic Energy Commission under Contract No. AT(29-1)-1242, and from continuing studies of blast biological effects at the Lovelace Foundation, supported by the Defense Atomic Support Agency of the Department of Defense under Contract No. DA-49-146-XZ-055. The table of references is not a complete survey of the literature on blast effects, but is intended to document the manuscript material and serve as a guide to the more important experimental work in the field of blast biologic effects and to previous published reviews.

The anatomic illustrations were selected from the author's material. Most of the charts, graphs, tables, and diagrams were generously contributed by my colleagues, particularly Drs. C. S. White and D. R. Richmond, for support and clarification of the subject. These have been presented and published elsewhere, although not in other papers of this symposium. Where these have been used, reference is indicated to the original publications.

ABSTRACT

Blast injury is a complex and very hazardous phenomenon to the biologic target. Together with effects of thermal radiations from modern nuclear weapons, blast injury (direct and indirect) appears to be accountable for the vast bulk of early deaths and casualties in nuclear explosions. This article has attempted to summarize the important clinical, physiologic, and pathologic information concerning the effects of direct air-blast injury on the biologic subject. Certain features have been emphasized in order to assist the clinical medical officer towards proper management of casualties. A brief description of pulmonary sequelae of blast injury is included for completeness.

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PATHOLOGY OF DIRECT AIR-BLAST INJURY

Thomas L. Chiffelle

Introduction

Direct or primary blast injury is a term which has superseded the older designation of "air concussion" and has become accepted as an expression of pathophysiologic events resulting from exposure of an animal organism to a high compression, high velocity shock wave, usually emanating from a detonating explosive in air. In its pure form, direct blast injury is characterized by lesions appearing in various internal organs, particularly the air-containing structures, without signs of external injury. In the usual catastrophic event, however, the clinical picture is often compounded by additional trauma caused by flying objects of varying size, by rapid bodily displacement, and by effects of heat, hot gases, dust, and sometimes by ionizing radiations. The physical nature and behavior of the blast force and the biologic response of several species of animals to various overpressure patterns have been adequately discussed in previous publications and by Dr. Donald R. Richmond in a presentation before this group.[32] Such information is not repeated in this report, but attention should be drawn to their importance for a more complete understanding of the scope and effects of blast injury.

It is rather likely that blast injury, even under a variety of synonyms, has existed ever since man made explosive devices or made conditions under which sudden increases in environmental pressure could take place. The fact that sudden changes in pressure could incapacitate or kill people, without any external signs of injury, has long been known and details of early reports and studies can be found in excellent reviews.[2, 5, 6, 35, 41, 42] From these one can conclude that prior to the onset of World War II, except for the little known studies of Hooker in 1919 and 1924,[16, 17] the manner in which the shock wave affected an animal organism was clouded in considerable mystery. It is surprising how little was contributed to the understanding of blast injury from the events of World War I. As Zuckerman (1941)[52] stated, blast was something that "blew a man to pieces or threw him violently." Ruska (1914), quoted by Clemedson,[6] appeared to be one of a few who appreciated the predominance of pulmonary lesions as an index of blast injury. Clinical studies of survivors generally laid great stress upon symptoms and signs from the central nervous system[23, 24, 25] and significance of effects in other organs and tissues tended to be minimized. One may but recall the popular term "shell shock," so frequently referred to in the casualty period, 1915-1920. This entity must have included unrecognized primary blast effects intermingled with cases of cerebral concussion, nervous exhaustion, fear, and other major or minor psychiatric disorders.

Hooker[16, 17] in 1919-1924 systematically studied the effects of blast injury on dogs and cats exposed to the muzzle flash of large guns, emphasizing the dominant role of lung lesions. He correctly visualized the injury as a result of "direct shock" action, but his efforts apparently received little attention until 1940, when subsequent investigators[2, 9, 12, 19, 22, 26, 27, 35, 47] added support to his original concepts.

The events of the Spanish Civil War, with the higher yield explosives, gave an entirely different dimension to the meaning of "blast injury." It was now noted that men could be killed or disabled at considerable distance without obvious external signs of injury. Benzinger[2] very aptly compared this to a "shot without a bullet" and "a slash without a sword." With the onset of World War II, studies and discussions of blast effects were resumed[13, 20, 21, 38] and three prevalent theoretical proposals emerged to explain the nature of blast injury and were summarized by Clemedson:[5]

1. That of Logan (1939), which held that the hemorrhagic lung injury was due to the action of the follow-up "suction" wave which caused or induced capillary distension and rupture.

2. That of Barcroft (1939), which contended that the pulmonary injury was brought about by sudden distension of the lungs by air driven in through the upper respiratory passages.

3. That of Hooker (1924), based on experimental studies, which suggested that nearly all the biologic effects of blast could be explained by the direct impact effect of the shock wave on the trunk. It was this theory which was championed subsequently by Zuckerman and his associates.

We are particularly indebted to the efforts of the British group (Krohn, Fisher, Zuckerman, and Whitteridge)[5, 10, 11, 22, 50, 51, 52] and of the German investigators (Desaga, Benzinger, and Rössle)[2, 9, 35] and to the Swedish investigations (Clemedson)[5, 6, 7] for establishing the fundamentals of blast injury and for much of the physiological alterations resulting therefrom.

Clinical and Physiologic Summary

A rather comprehensive literature exists concerning the clinical aspects and many physiologic studies of blast injury, and the reader should refer to the many excellent original reports or reviews of these phases of the subject.[5, 6, 9, 18, 19] What follows is intended as a summation of the pertinent medical features of blast injury, together with significant highlights of physiologic studies and their clinical interpretation. The classification of primary, secondary, and tertiary effects of blast have been alluded to in a previous presentation[32] and will not be discussed further here. It should be remembered, however, that in

any explosive situation, the clinical picture of casualties will be subject to considerable variation, depending on the severity of the blast and to "indirect" effects previously referred to as secondary and tertiary effects.

The man or animal may be killed outright, without external signs of injury, but often with blood-tinged froth or frank blood appearing in the nose and mouth. Survivors have the same more or less negative external appearance, but may suffer from air hunger, with rapid, shallow respirations, and are usually quiet, apathetic, or even lethargic. Some of these may die in times varying from a few minutes to 24 hours. A few may succumb at longer intervals of a few days. Central nervous system signs are quite variable and, when injury is severe, may include disturbances in equilibrium, convulsions, and even paralysis. The patients may complain of tightness across the chest and varying degrees of chest or abdominal pain. Eardrum perforation (which may be overlooked as a diagnostic aid) is exceedingly common due to the rather low threshold for tympanic membrane rupture under pressure loading. In casualty studies,[38] deafness and tympanic membrane rupture occurred in as high as 80 per cent of the cases; many of these had actual hemorrhagic blebs on the eardrum which would account for some serosanguineous oozing from the external ear.

Coughing will occur, but not usually in the beginning. Depending on the severity of the exposure, positive x-ray findings of "cotton ball" densities in the lungs will usually divulge the bilateral nature of the process. Hemoptysis may appear, often well within an hour and tends to be repeated. With any degree of physical exertion, the pulmonary hemorrhage tends to be progressive, lending considerable support to the therapeutic argument for early and complete immobilization. The dimensions of the hemorrhagic process in the lungs can often be roughly related to survivorship, which is another way of correlating overpressure, extent of injury, and mortality. A rise in venous pressure and a conspicuous fall in arterial pressure, associated with bradycardia, usually completes the clinical picture.

Most of the survivors, if carefully cared for and pneumonia and abdominal injuries do not occur, appear to get well soon and the hemorrhages resolve quite rapidly. However, sequelae do occur and may be serious. Among these are pulmonary edema, bronchopneumonia, and even lung abscess. Hemothorax, pneumothorax, and empyema may present serious problems, as well as perforation within the abdominal cavity with subsequent peritonitis.

It is of interest that Hooker,[16, 17] in his early experience with muzzle-blast effects, rather well described the entire clinical picture, including not only the respiratory difficulties, but fatigue, lethargy, and occasional paralysis. He also noted a drop in the blood pressure which often occurred almost immediately. In addition, he noted that a high percentage of fatally injured animals usually died within a period of one hour.

In the light of physiologic studies, the sequence of clinical events may be explained as follows:

With respect to the respiratory system, pulmonary hemorrhage will lead to airway obstruction varying in degree with the extent of the injury, which, in turn, increases the rate of respiration. There seems to be little or no evidence for a central nervous system role in this process unless there is evidence of associated head injury or when cerebral air emboli may exist. Cassen[4] has suggested that there is a natural vasoconstrictive mechanism in the lung which, after blast injury, becomes manifest over approximately 15 to 20 minutes, tending to limit the hemorrhage from smaller vascular disruptions and promote clotting in the torn ends of the blood vessels. There seems to be little doubt, in some individuals, that virtual suffocation takes place when continued pulmonary bleeding with subsequent development of pulmonary edema obliterates the available air-exchange surfaces, decreasing oxygen saturation and ultimately resulting in a state of generalized anoxia. However, it should be borne in mind that a tremendous amount of pulmonary hemorrhage can exist without jeopardizing the small amount of lung tissue necessary to survive.

Secondly, from the standpoint of the cardiovascular system, the distinctive fall in arterial blood pressure, associated with bradycardia, is distinguishable from other forms of vascular shock. Several factors may be considered operative in this particular complex:

a. Stimulation of vagal reflex mechanism, with the effector organ as the heart, can result in bradycardia, reduced activity of the heart, and subsequent hypotension. It has been shown that these effects can be minimized by vagotomy. The sensing level is obscure, but it might arise from vagal centers in the central nervous system or local ischemia of the central centers could be produced by air embolism. It can also arise from pulmonary receptors, from increased pressure in pulmonary arteries, and from the heart itself in an anoxic state.

b. The mechanical blocking of the pulmonary circuit, induced by hemorrhage, edema, and vasoconstriction, could induce cardiac rate reduction and hypotension.

c. Direct cardiac ischemia, produced by massive coronary air embolism, may, and no doubt commonly does, yield a situation of right or left heart failure occurring rapidly in a matter of a few minutes.

d. Direct violence to the myocardium may produce a phenomenon of "commotio cordis," wherein a disturbance in the conduction system apparently occurs with rhythm changes, fibrillation, and heart block. In the worst cases of blast injury, myocardial contusion, hemorrhage, and even disruption of fibers have been observed. Benzinger,[2] however, has expressed belief that commotio cordis is a rather uncommon cause of cardiac failure by itself.

Thirdly, the effects of arterial air emboli arising in the lung is now known to be one of the most significant causes of death, particularly in those cases which succumb shortly after injury. Following the disruption of pulmonary vessels, quantities of air are introduced into the pulmonary venous circuit and appear in the left side of the heart, from whence they are distributed to the general circulation. The most significant distribution points, of course, are in the coronary and cerebral circuits. This very important phenomenon was not generally recognized until emphasized by the studies of Benzinger,[2] who found the emboli in a high percentage of his experimental animals. At the risk of oversimplification, he stated that air embolism solved much of the mystery of the rapidity, uniformity, and precision of death by blast, and also would account for the varied cerebral signs observed clinically. He contended that up to 30 minutes after injury, air emboli dominated the cause of death. At intervals greater than 30 minutes, death was probably caused by suffocation. Still later, death could be attributed to the complications of the primary injury. From these studies and similar studies in our own laboratories,[31] it is difficult to escape the conclusion that coronary air emboli are, indeed, the principal cause of rapid heart failure in blast injury. As will be discussed in more detail later, the incidence of air emboli increases with increasing severity of lung injury until it attains a proportion of 85 per cent at the near 100 per cent lethality mark.

Summary of Investigations During 1939-1945

At this point, because of the several conflicting views of the nature of blast injury prior to 1940, it is well to consider certain facts emerging from the studies of Hooker[16, 17] and of the British and German investigators.[2, 9, 22, 35, 52] These scientists systematically and intensively studied problems, using all practical, available instruments and methods, including recordings of environmental changes with pressure-time recording devices; photographic recordings with cameras; measurements of respiratory movement and pulmonary function; blood-pressure effects; biochemical studies of blood oxygen saturation, alkali reserve, and carbon monoxide ; x-ray studies; electrocardiograms; electroencephalograms; and detailed pathologic examinations of the tissues and organs of the experimental animals. The findings tended to dispel some of the earlier misconceptions and generally cleared the mystery which surrounded the nature and cause of blast injury.

1. First, and probably most important, was the fact that the impact of a high-velocity pressure pulse upon the trunk of an animal or man was absolutely essential for the production of direct blast injury, and, for the time interval involved (duration of the pressure pulse), enormous compressive forces were brought to bear which completely enveloped all but the larger animals.

2. Transmission of the pressure pulse through the upper respiratory passages was found to be negligible and not responsible for pulmonary injury.

3. The negative or suction phase of the shock front, once suspected of causing injury, was found not to be contributory or significant in the nature of the injury.

4. Blast injury and its attending cerebral signs could not be produced by the impact of the shock front on the cranium when the trunk was shielded from the pulse and the head was prevented from wind displacement.

5. The presence of air within or near an organ or tissue component was a major factor in the bulk of blast injuries. For example, the lungs, ears, sinuses, and portions of the gastrointestinal tract were the most vulnerable locations and the most common sites of injury.

6. The degree of blast injury could be directly correlated with the rate, magnitude, and character of the pressure rise and fall; the duration of the pulse; and the size and species of animal involved. Some of these relationships were first suspected from studies of casualty effects at various distances from the point source of the explosion.

7. In studies with various species of animals, using the P_{50} index, biologic tolerance was also related to the size and weight of the animal. With short-duration, rapidly rising pressures, the larger species were found to tolerate much greater overpressures. More recent studies have shown that these species differences in tolerance dwindled considerably with overpressures of very long duration.[31]

8. Benzinger[2] seems to have been the first to have suggested that the rate of pressure rise could be a decisive factor in morbidity, by pointing out that very high pressures were tolerable when the rise time is relatively slow. The truth of this has been clearly shown in the recent studies of Richmond and White.[31]

9. As previously described, the occurrence of air emboli is to be considered the principal cause of death in early fatalities. Although occasionally suspected as a factor, the credit for its emphasis belongs to the German school of Benzinger, Rössle, and Desaga.[2, 9, 35]

10. On the basis of certain known physical data concerning the transmission of pressure pulses through solid and liquid objects, it has been postulated that certain other factors related to the transmission of a pressure pulse might be of significance in the better understanding of the pattern of blast injuries.[37] These were listed by Schardin[37] as follows:

a. Spalling effects, which have reference to the disintegrative forces that occur at interfaces when a pressure pulse of high velocity progresses through structures of different densities.

b. Inertia effects, referring to the stresses or shearing forces that develop between two intimately related masses of different densities when subjected to pressure forces. Different acceleration rates develop between the different types of tissue, the lighter structures being accelerated more rapidly than the heavier. Shearing forces would develop between the two, resulting in disruption and separation.

c. Implosion effects. Such phenomena are observed in fluid media. Assuming the presence of microscopically tiny gas bubbles, it has been shown that a passing high-velocity pressure pulse will compress them, resulting in marked internal pressure and temperature increase. When the shock wave has passed by, these compressed and heated bubbles expand with near explosive force, creating new shock fronts emanating from their centers.

Model Studies

In order to visualize more clearly what is happening to the animal organism subjected to the onslaught of a high-velocity pressure pulse, we have found it particularly useful and instructive to employ simple mathematical models to aid in visualizing the relationships between the pressure variations of the environment and those which might be expected internally in the gaseous and fluid phases of the lungs. By diagrammatic means, one can more easily illustrate the general configuration of masses and forces which may be involved in the sudden application of external pressure. The following figures were taken from the report of White and Richmond.[46]

As depicted in Figure 1, one can consider the animal trunk as a simple box-like structure divided across the center by a flexible muscular diaphragm into thoracic and abdominal compartments. Thus, the trunk is seen as consisting of a body of fluid and gas contained within an elastic skin, but complicated by structural materials of different densities, varying from hard bone to a spongy air-containing lung. At the top, the trachea is shown with a narrowing or constriction representing the bronchiolar resistance to air flow. In approximately the center of each main section, there is another box-like structure representing the fluid phase of the thorax and abdomen, including vascular components, with connecting passage between the two. In the lower half of the figure, there is a pressure-time graph reflecting the behavior of internal pressures as the external pressure is increased above ambient. In this first illustration, one may consider the external pressure rising at a rate sufficiently slow to permit internal adjustments. Under such circumstances, there is ample time for air to flow through the upper respiratory passage into the alveoli at a rate which will keep the external gas pressure apace with the pressure of the fluid phase in the thorax and abdomen. The internal pressure will lag slightly behind the external level, but this difference is not physiologically significant and no hazard would exist if the maximum pressure was not too high nor too prolonged. One can also imagine that, if the external pressure

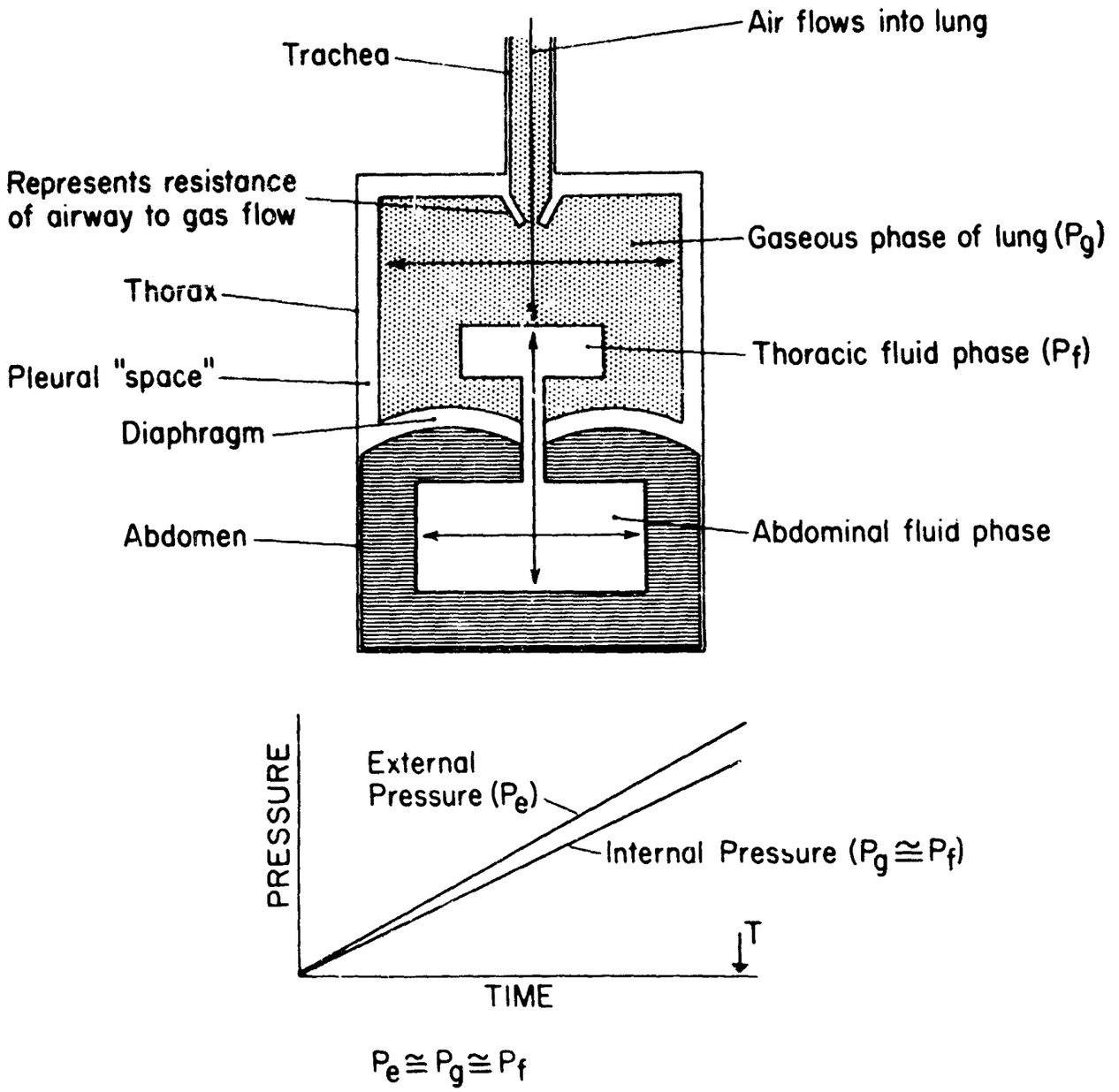

Figure 1 Diagram of Model Response to Slowly Rising Overpressure

were applied somewhat more swiftly, this lag in internal pressure adjustment would be greater, permitting a transfer of abdominal (and other) fluids into the thoracic compartment. It would also seem apparent that the faster the external pressure rise, the less competent becomes the constrictive orifice of the upper respiratory passage.

Figure 2 illustrates a situation in which the external pressure is applied quite rapidly and sustained for a period of time. This rate of pressure application is sufficient to deform both the chest and abdomen until the elastic limit of the walls is reached. There will be a marked difference in external and internal gaseous phase, permitting a large increase in fluid volume to appear in the chest cavity. Because of the incompressible nature of fluid, this transfer would take place quite rapidly. There would be some air flow through the upper respiratory passage, but at a rate insufficient to offset the rapid influx of fluid into the thorax. Thus, the pulmonary and vascular structure becomes overloaded and distended, with the likelihood of vascular rupture and resulting local areas of hemorrhage.

In Figure 3, we have a situation quite analogous to the effects of a high-explosive charge where the external pressure is of almost instantaneous rise but of relatively short duration. In the pressure-time graph, the external pressure rises and falls rapidly, for example, in a matter of a few milliseconds. There would be some deformation of the trunk walls, but the time period would be insufficient to overcome inertia for a maximum deformation, as depicted in the previous figure. The principal effects can be visualized as due to the transmission of a pressure pulse to the liquid phase of the lung before an equalizing pressure pulse can be transmitted through the air. (It should be remembered that a high-velocity pressure pulse travels several times faster in fluid media than in air.) Lung damage and hemorrhage would result from pressure differentials, together with implosion and spalling effects as discussed previously. Where delicate pulmonary veins are disrupted, it is now possible for alveolar air to be injected into the vascular circuit at the time of injury and with the resumption of respiratory cycles.

Finally, in Figure 4, a situation is illustrated in which there is a "fast"-rising overpressure which is sustained for a long period of time, and is analogous to the situation encountered with nuclear-type explosions. Because the pressure is sustained, the truck is maximally deformed and the momentum of the thoracic compression permits an overshooting of the internal pressure, producing an oscillatory or reverberation pattern of the internal gaseous phase (P_g), as seen in the pressure-time graph. It is of particular interest that such a mathematically predicted pattern has been found to occur in experimental animals when small transducers are appropriately placed to monitor the internal pressures. In fact, the measured pressure pattern almost exactly matches the oscillatory pattern predicted mathematically,[15] and is graphically illustrated in Figure 5. Whether this oscillatory pattern is of physiologic significance cannot be directly stated at this

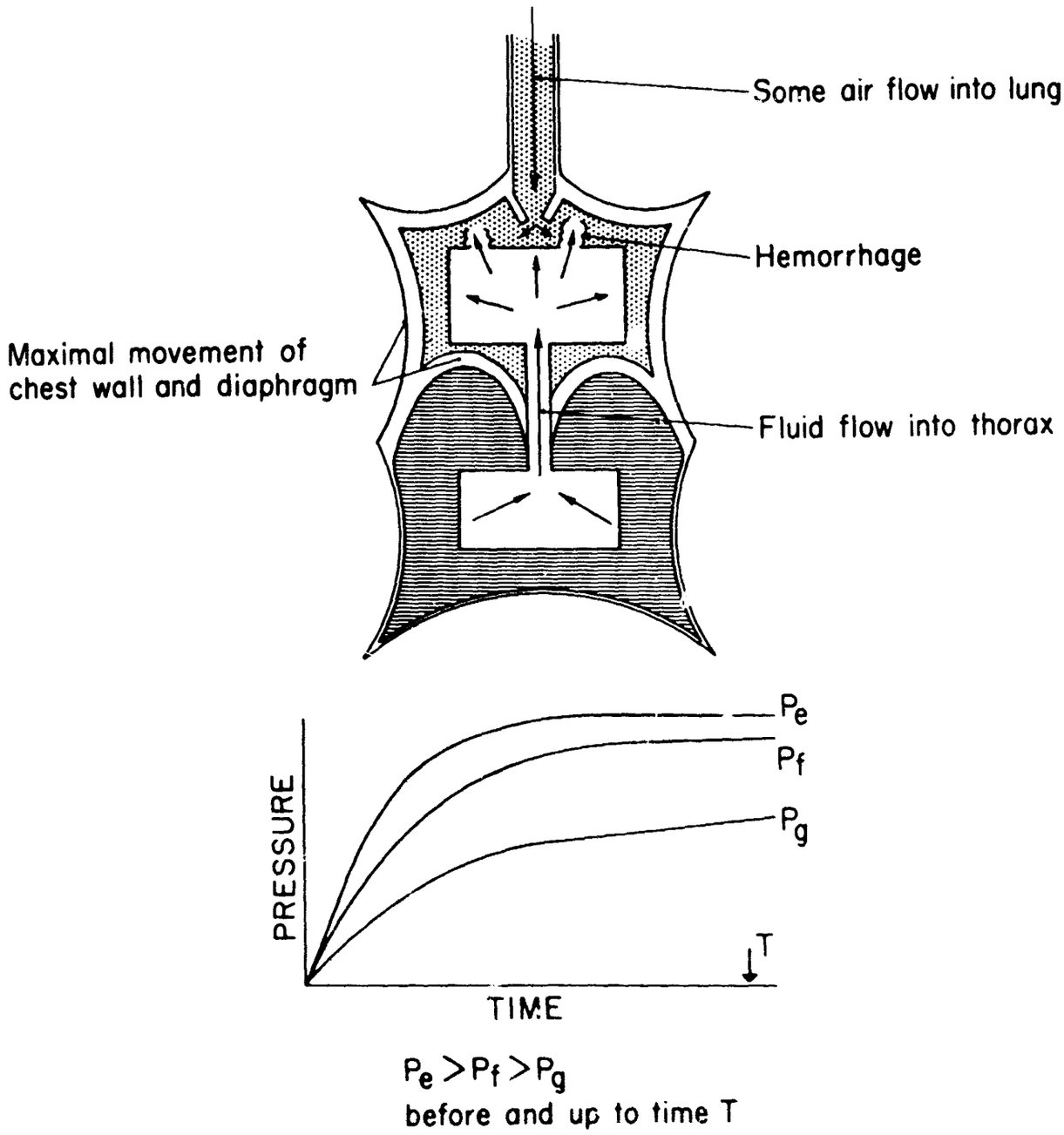

Figure 2 Diagram of Model Response to Moderately "Fast"-Rising Overpressure

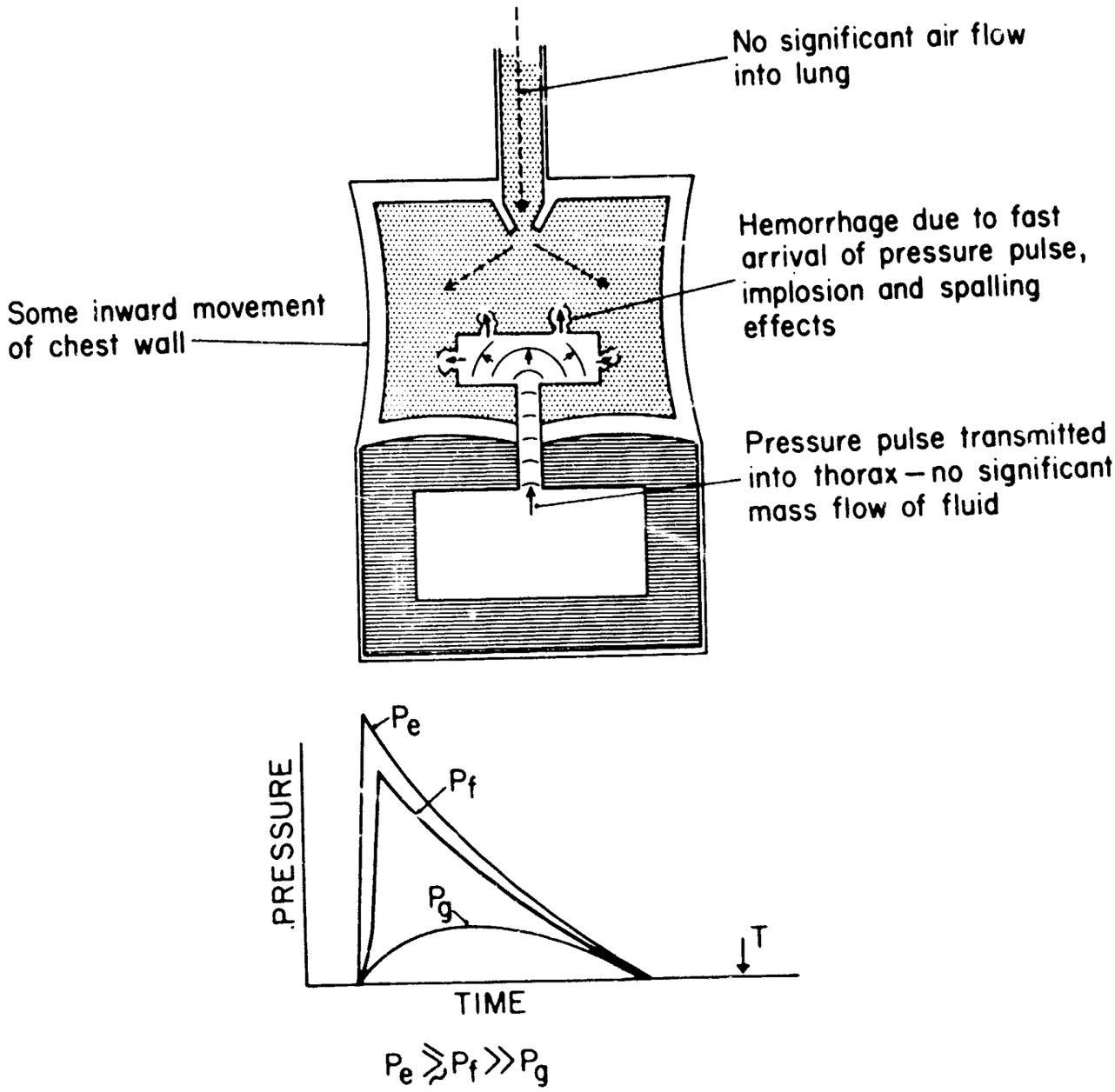

Figure 3 Diagram of Model Rsponse to "Fast"-Rising Overpressure of Short Duration

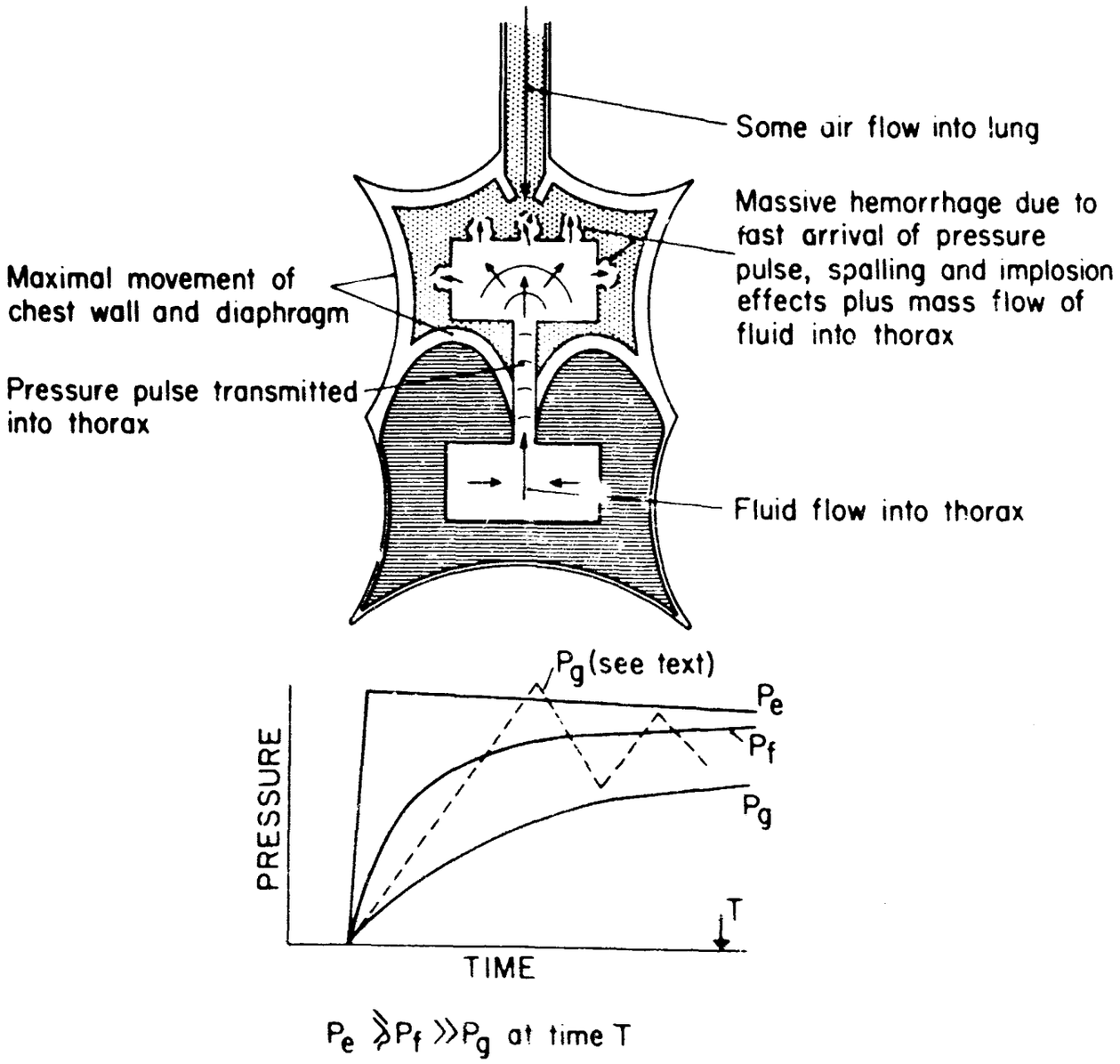

Figure 4 Diagram of Model Response to "Fast"-Rising Overpressure of Long Duration

Comparison of Observed with Simulated Lung Pressures

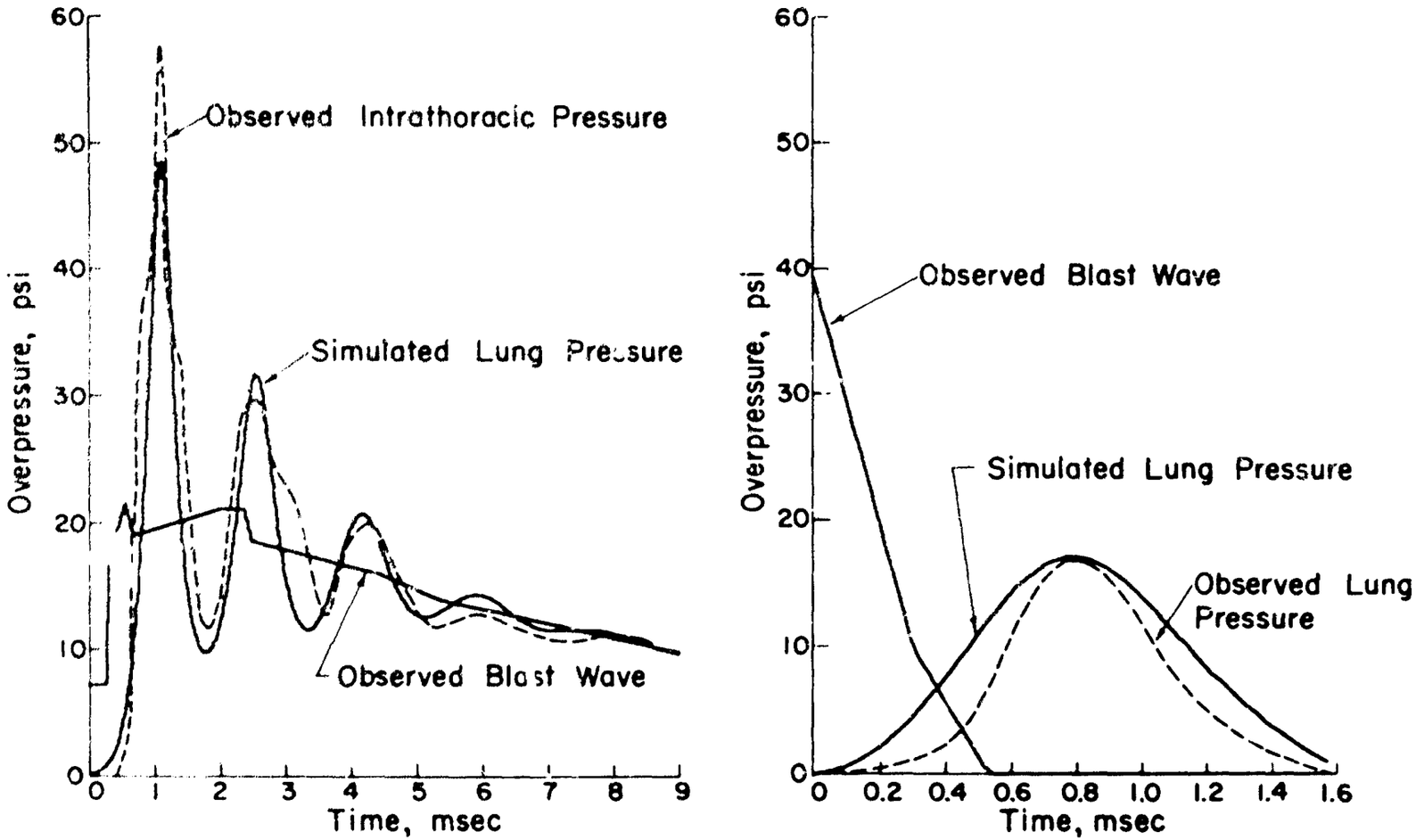

Figure 5 Comparison of Simulated and Recorded Internal Pressures. Left Figure: The Internal Pressure was Recorded in the Thoracic Portion of the Esophagus of a Rabbit Exposed to Shock-Tube Blast Overpressure.[15] Right Figure: The Internal Pressure was Recorded in the Rabbit Lung Facing a High Explosive Charge. (Clemedson and Jönsson, J. Appl. Physiol., 16: 426-430, 1961)

time. However, it raises the question of possible pathologic effects due to resonating qualities in different portions of the body and organs. There is, in addition, considerable movement of fluid into the chest, and local vascular disruption occurs because of pressure differentials, internal inertia differences, implosions, and spalling effects.

Lethality Data

The pattern of lethality observed in animals exposed to blast injury is remarkably and characteristically early. This consistency was first noted by Hooker,[17] who commented that nearly all of the fatally injured animals were dead by the end of the first 24 hours. Clinical observers of casualty situations have repeatedly noted that those who managed to survive the first few hours after blast injury were frequently saved. In recent years, Richmond and White[31] have been able to consolidate this observation by summarizing studies of a large series of animals (of various species) exposed to controlled overpressures of similar pressure-pattern characteristics. So regular is the rate of death in an individual species that the accumulated mortality (and degree of injury in survivors) of a series can be predicted fairly well for any given overpressure situation.

Figure 6 illustrates a typical cumulative per cent mortality of a series of guinea pigs exposed to overpressures in the range of 30 to 60 psi. The observation period was 30 days and detailed records were kept of the animal condition and the pathologic findings. All autopsies were performed within 10 minutes of death. The deaths were recorded to the nearest minute for the first 2 hours, to the nearest hour for a period of 2 to 24 hours, and daily for a period of 2 to 30 days. From the graph, it can be seen that over half the fatally injured animals will succumb within the first 30 minutes, and about 75 per cent of them will be dead within the first 2 hours. At the end of 24 hours, 90 per cent of the animals which are going to die will have succumbed and the few remaining are scattered at intervals over the subsequent 18 to 20 days. Within a given species range, this behavior pattern is very constant. When comparing one species group against another, as in Figure 7, the initial rate of dying would appear to be somewhat lower for the larger animals, although the overall features are essentially similar to that of the smaller animals. However, the lower initial rate, as reflected in depression of the curve for the series of larger animals, probably represents those animals which received a lower pressure exposure, resulting in a delayed death. This can be seen more easily in Figure 8, where the collected guinea-pig data of Figure 6 is separated into four curves representing the pressure levels at which the animals were exposed. Eighty per cent of the animals exposed to the highest average pressure (51.3 psi) died in 30 minutes, whereas 44 per cent of the animals exposed to the lowest average pressure (30.2 psi) died in 30 minutes. The remainder represent the delayed death over the 20-day period.

From the standpoint of gross pathology of these animals, it has been found that the mean lung weight for each group can be analyzed in terms of acute death, sacrifice at a later time, and controls. In general, as depicted in Figure 9, the lung weights of acute deaths are high, generally

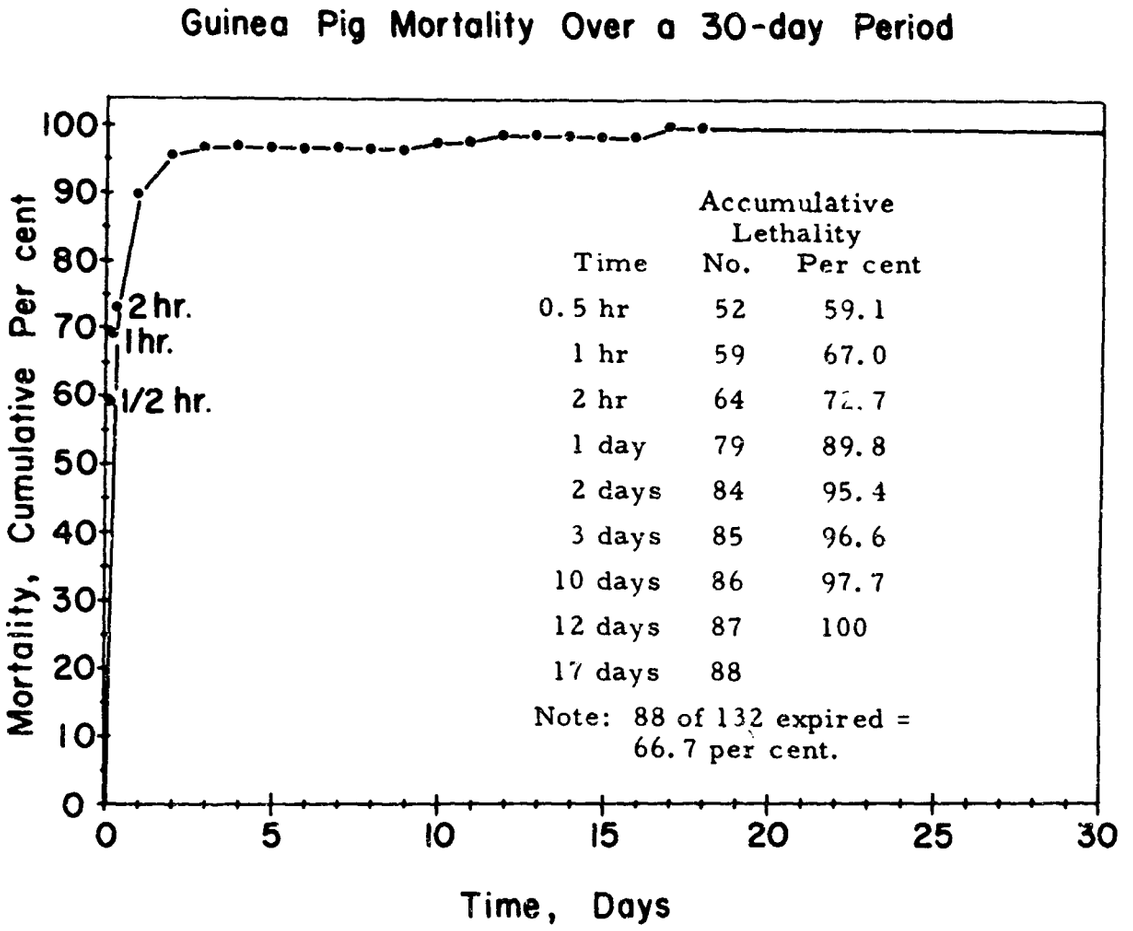

Figure 6 Cumulative Mortality for a Series of Guinea Pigs Exposed to Blast Overpressures. Time Period of Observation was 30 days.[43]

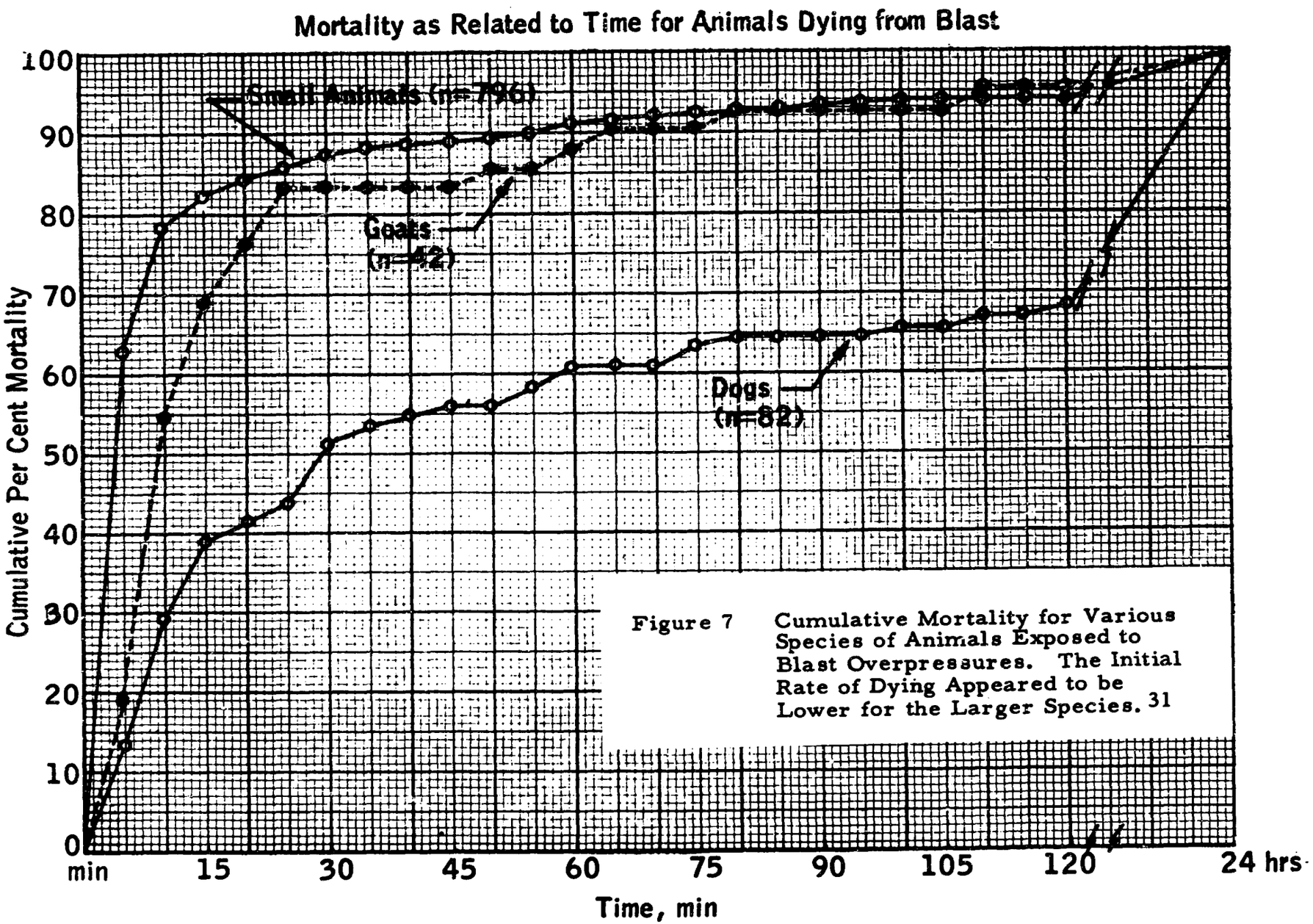

Figure 7 Cumulative Mortality for Various Species of Animals Exposed to Blast Overpressures. The Initial Rate of Dying Appeared to be Lower for the Larger Species.[31]

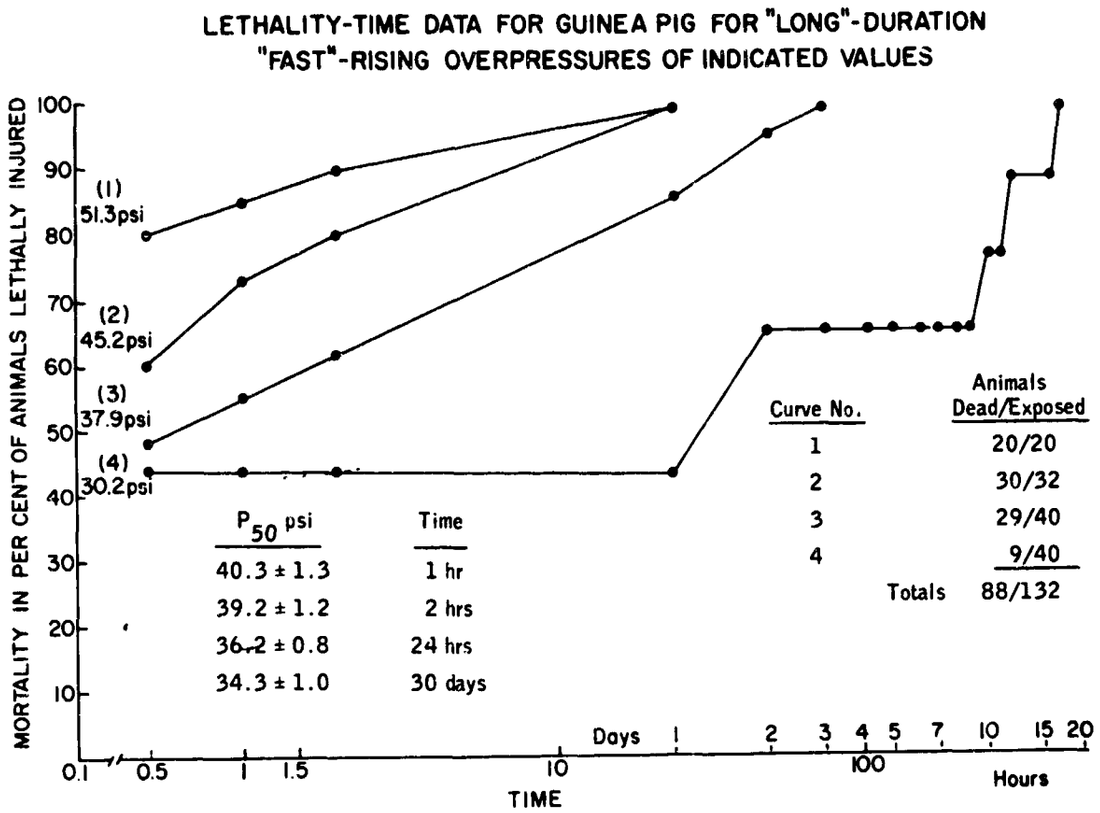

Figure 8 Reference: Clare et al., DASA Technical Report No. 1312, May 31, 1962.

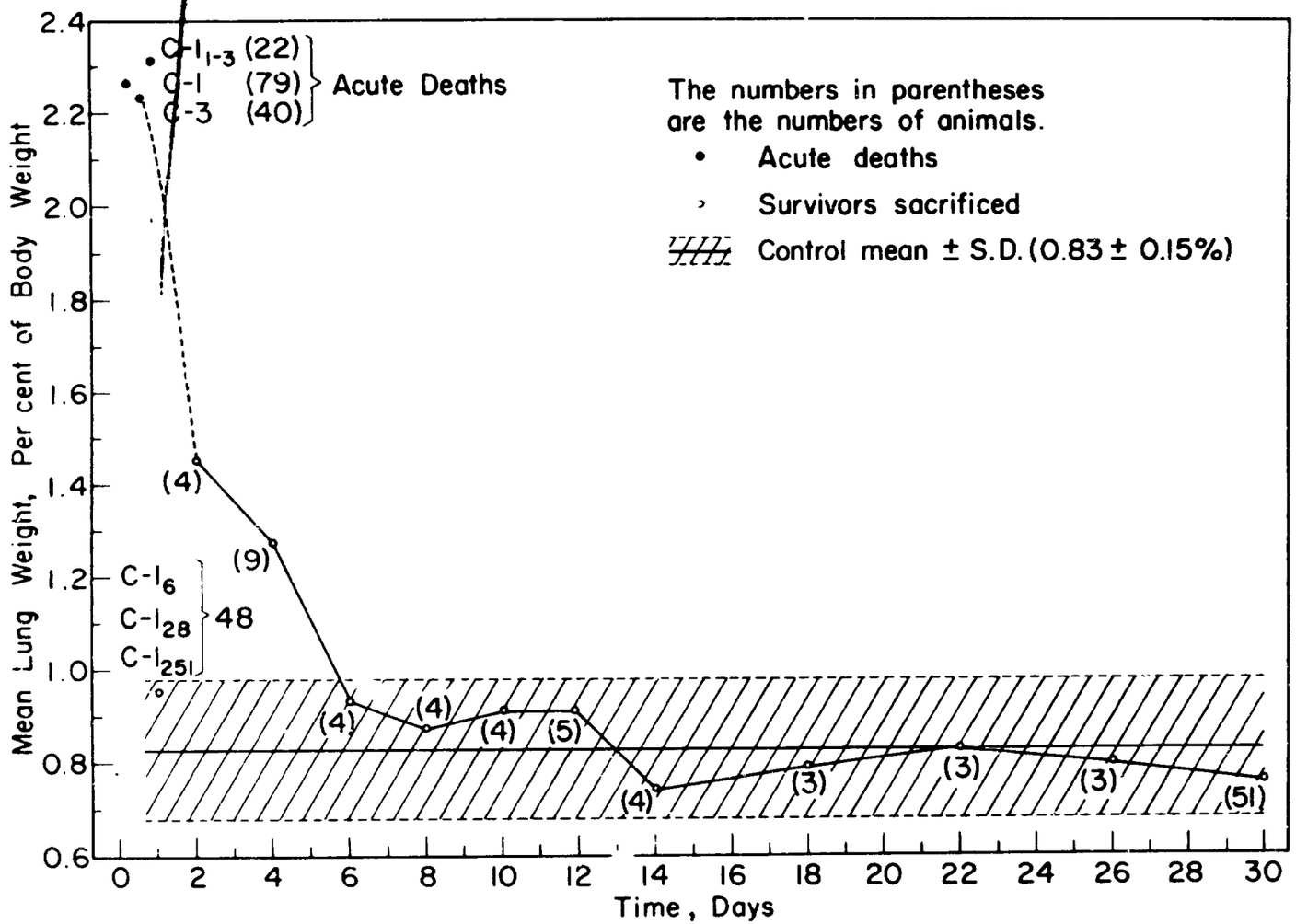

Figure 9 Comparison of Lung Weights of Animals Killed by Blast and Animals Sacrificed Subsequently. Reference: Clare et al., DASA Technical Report No. 1312, May 31, 1962.

averaging 2 per cent or more of their body weight. The ratio drops to near 1.5 per cent in the next 2 to 4 days, and with clearing of the hemorrhagic lesions, the ratio later approaches the normal average of 0.83 per cent. The findings would thus indicate that lung hemorrhage may be expected to disappear in approximately 7 to 10 days. In Figure 10, there is a compilation of mortality data (collected from several exposure series totaling 556 animals) relating lung weights (as a per cent of body weight) and incidence of air embolism in animals which died or survived blast injury in shock tubes. In this collection, the rise times of the pressure pulse were rapid, but the duration prolonged to 6 to 8 seconds. The vertical solid lines of the graph serve to divide the pulmonary injuries into arbitrary groups, depending upon the relative lung weight. Thus, the range extends from barely perceptible or essentially threshold values on the left to maximal possible involvement on the far right. The parallel relationship of per cent lung weight to per cent mortality is readily apparent. The striking incidence of air emboli related to the degree of lung injury and mortality is also demonstrated. In the 12 per cent mortality group, the incidence of air emboli was 27 per cent, but in the near-100 per cent mortality situation, the incidence of air emboli was 85 per cent. Since there are some practical difficulties in establishing the existence of air emboli because of their transient nature, rapid diffusibility, and the small volume of coronary circulation available for observation, it is likely that the quoted incidence of air emboli is in the low order and the true incidence must be higher. Under some circumstances, it is not always possible to conduct postmortem examination in the very short interval (15 to 30 minutes) after death that is often necessary to ascertain the existence of such.

Pathologic Findings in Acute Blast Injury

Various aspects of the gross and microscopic findings in blast injury studies, either from air or underwater sources, have been well presented in previous reports[3, 8, 17, 22, 35, 36, 39, 40, 47, 48, 50, 52] and are summarized in the following discussion. For purposes of convenience, the subject matter has been subdivided into systems and specific organs or tissues.

1. The Thorax

As stated previously, in the absence of trauma indirectly attributed to blast, as from bodily displacement, or by missiles, the human or animal organism generally exhibits no obvious external signs of injury, but may have an accumulation of frothy, blood-tinged fluid or frank blood in the nares or mouth. In general, the most conspicuous internal injuries are seen within or adjacent to air-containing structures. In severe cases pulmonary hemorrhage is the most regular and impressive finding. It is generally bilateral and occurs in patches showing a predilection for internal structures of the lung which are of greater density than that of the soft alveolar tissues. Thus, the hemorrhages are often seen in relation to the bronchovascular structures. However, on the

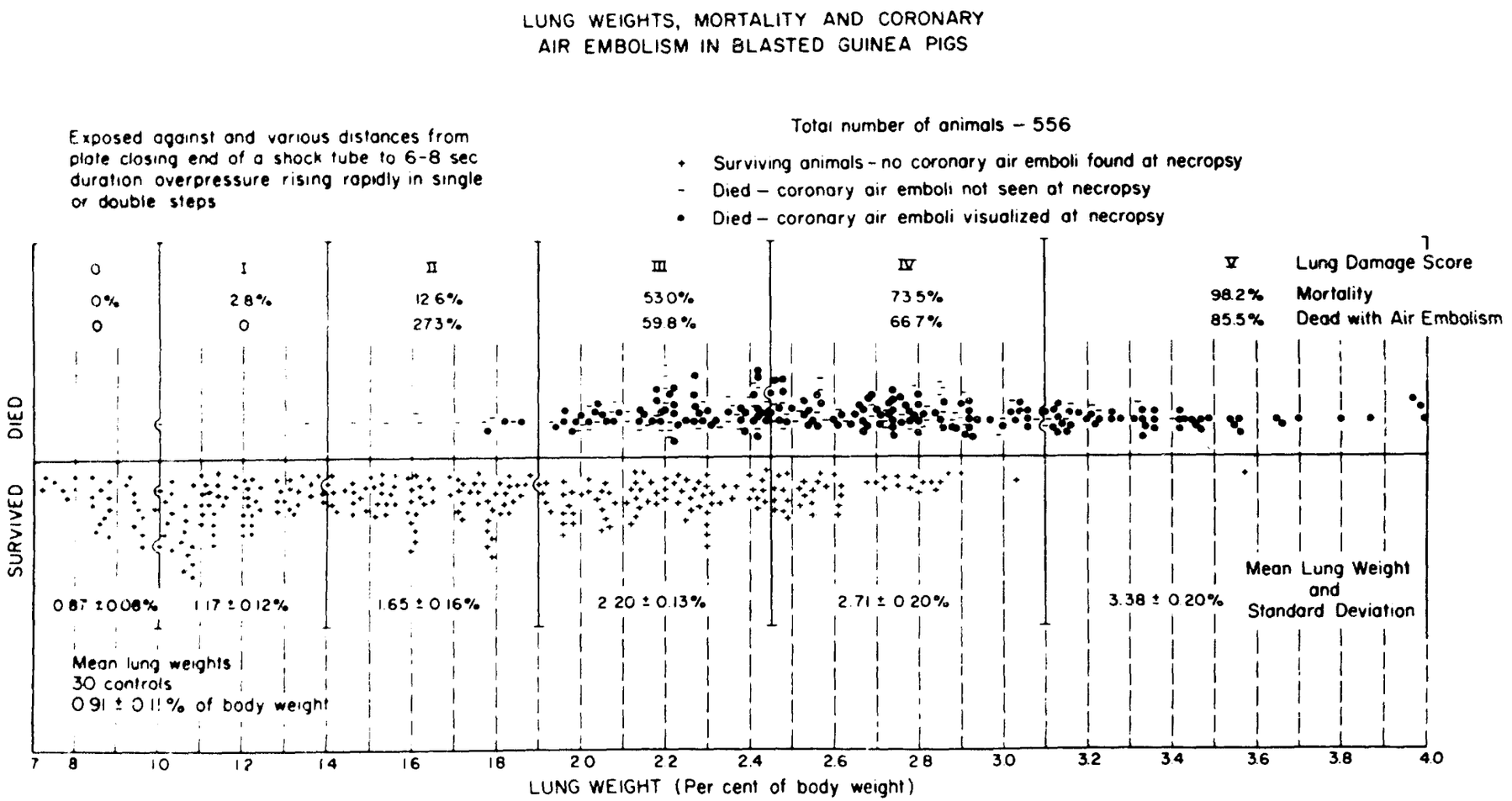

Figure 10 Compilation of Mortality Data and Gross Pathological Observations from Series of 556 Guinea Pigs Exposed to Blast Overpressures.[31]

surface of the lung, hemorrhagic markings are found in zones which normally are adjacent to hard, firm structures, such as the heart, the vertebral column, and the ribs. (See Figures 11 and 12.) Marginal areas of hemorrhage are often seen in such areas as the costophrenic angle, where compression pinching of the lung tissue occurs as the diaphragm is driven upwards. When the injury is very extensive and marked, the pulmonary lobe may be virtually consolidated with blood, and some of the finer details described above will have been obscured. Emphysematous blebs and interstitial emphysema are sometimes seen grossly. Small lacerations of the pulmonary parenchyma may be found by the examiner, even on the pleural surface, in which case some blood and air is present in the pleural space.

The lung weight is increased generally in proportion to the degree of hemorrhage. However, in some cases of delayed death and as cardiac stress progresses, pulmonary edema supervenes and contributes to the increased weight. Dissection of the bronchial tree will show accumulations of frothy and bloody fluid or strands of clotted blood which may, at times, form an actual cast of the airway. Part of this intrabronchial accumulation of blood is derived from alveoli which become overfilled and the excess wells up into the bronchioles. But, there is another source of the blood, and perhaps equally significant, derived from local areas of direct injury to the bronchial or bronchiolar wall and to the blood vessels therein. This finding has been reported previously by Cameron et al.,[3] but its significance has not generally been appreciated. It is important to note that, in many instances of rapid death, the total blood and observed diminution of respiratory surface in the lung is quite insufficient to account for death. This is in keeping with the oft expressed clinical opinion (in casualty studies) that the amount of blood seen in the lungs is frequently inadequate to explain death by suffocation alone. It should be noted, as discussed later, that coronary air emboli will account for rapid death from cardiac ischemia, and with early failure of the heart, there will be minimal lung hemorrhage and no significant increase in lung weight.

In Figures 13 to 17, a number of interesting features are seen. Figure 13 illustrates a rather typical appearance of the hemorrhagic areas in which large numbers of alveoli, alveolar ducts, and terminal portions of the bronchioles are choked and distended with blood. Various quantities of edema fluid may be present, although this is not usually seen at first and its presence denotes a more delayed form of death. Traumatic emphysema is conspicuous, even in relatively mild injuries. Overdistension of the alveoli is generally more apparent in the peripheral portions of the lung and in the vicinity of the hemorrhagic zones. Subpleural vesicles or blebs may be formed.

A distinctive microscopic feature which appears rather characteristic of blast injury is the wide separation of alveolar tissue from the denser and more rigid bronchovascular structures, as illustrated in Figure 14. This separation is quite conspicuous and is presumably

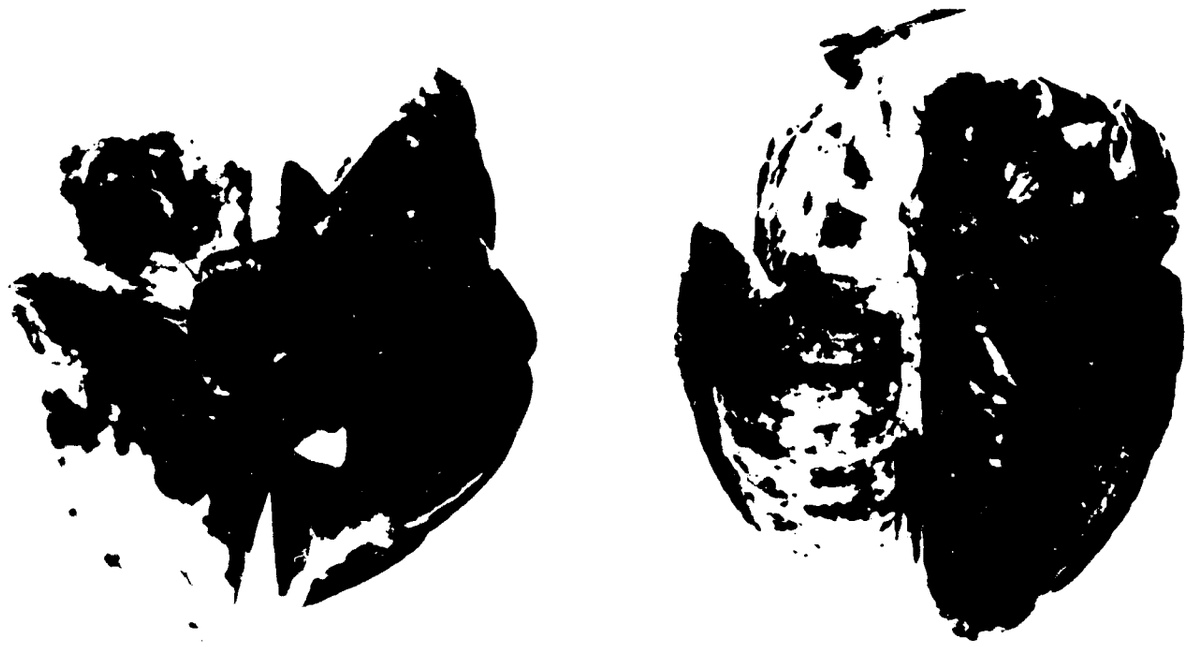

Figure 11 and Figure 12 Gross Appearance of Dog Lungs, Anterior (Left) and Posterior View Immediately after Exposure to Severe Blast Overpressure. Largest Areas Show Hemorrhagic Consolidation throughout the Lobes. Note the Transverse "Rib" Markings on the Posterior Surfaces (Right).

Figure 13 Typical Microscopic Appearance of Fresh Blast Hemorrhagic Zone in Dog Lung, with Alveoli, Alveolar Duct, and Terminal Portion of Bronchiole filled with Blood. Reproduced from Color Illustration. X 80

Figure 14 Dog Lung Exposed to Blast, Microscopic Section, Showing Typical Separation of Large Vascular Structure from Surrounding Alveolar Tissue. The Space is Partially Filled with Blood. Reproduced from Color Illustration. X 82

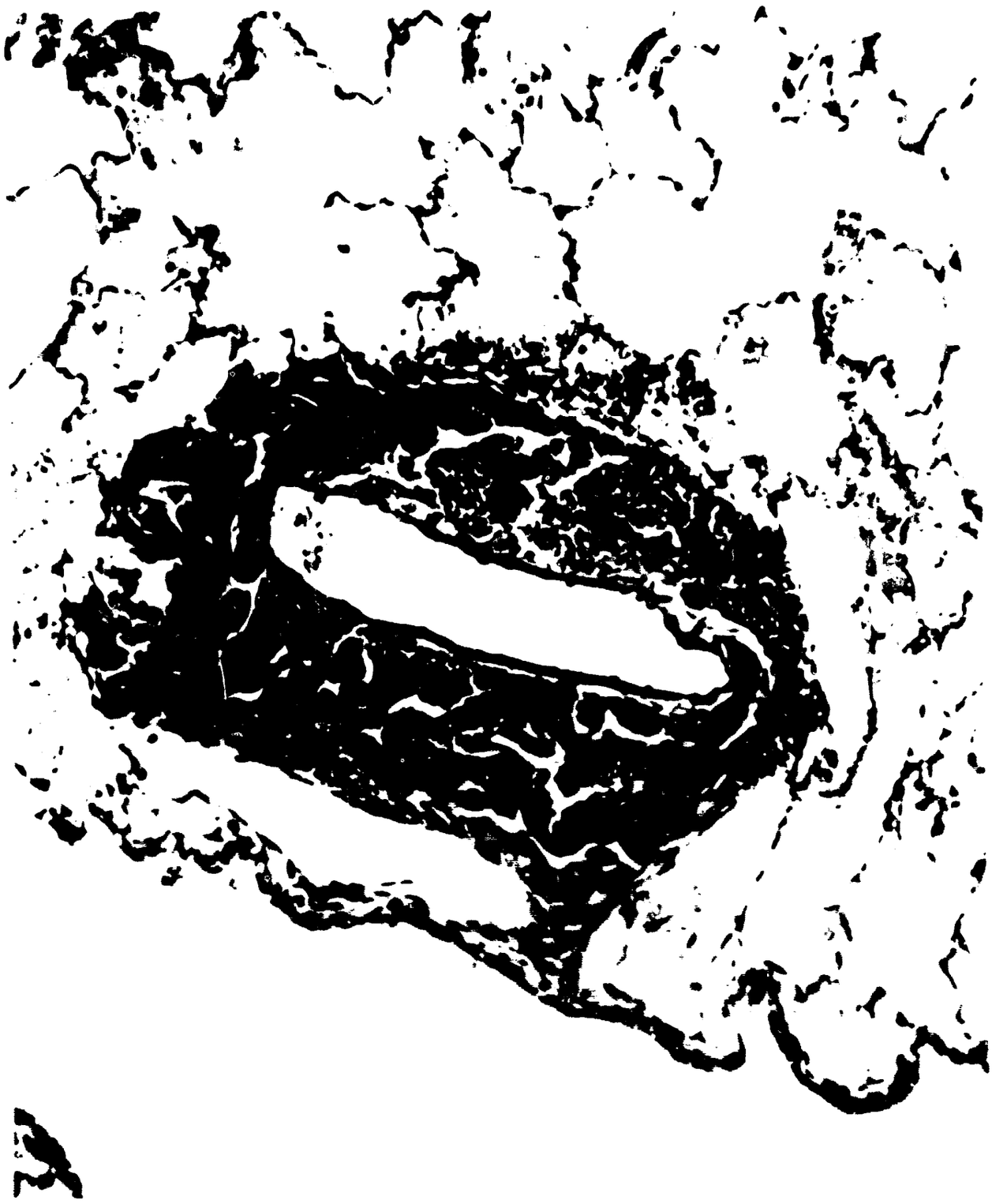

Figure 15 Dog Lung, Exposed to Blast, Microscopic Section, Showing "Ring" Hemorrhage Filling the Separation Gap Between Blood Vessel and Alveoli in Non-Hemorrhagic Zone. Reproduced from Color illustration. Z 82

Figure 16 Dog Lung, Blast Exposed, Illustrating Bronchial Hemorrhage and Injury to Lining Epithelium. Reproduced from Color Illustration. X 82

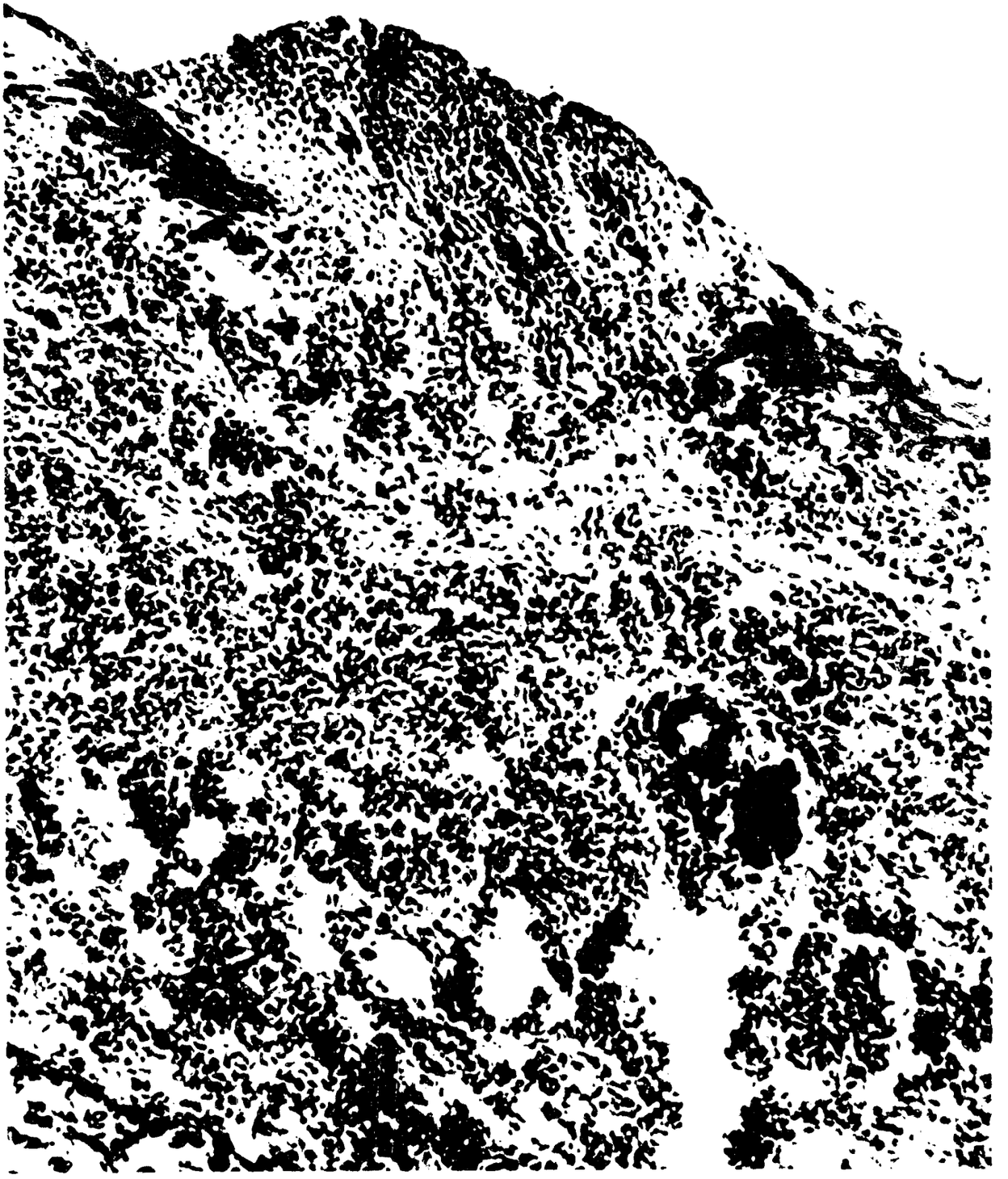

Figure 17 Dog Lung, Blast Exposed, Showing Segment of Hemorrhage and Disrupted Bronchial Wall. The Epithelial Lining has been Lost. Blood is Seen to Extrude through the Torn Portion of the Wall into the Lumen. Reproduced from Color Illustration. X 140

due partly to the application of severe shearing forces between structures of different densities, and possibly due to vast overdistension of the vascular system occurring at the time of injury when large amounts of blood are forcibly injected into the pulmonary vascular circuit. At any rate, the resulting "gap" between the two types of tissue is often quite wide, and disruption of small and medium sized blood vessels of the interstitial spaces may be found. Thus, the space may be filled with blood, edema fluid, lymph, air, or a combination of all four. This disruption is not necessarily associated with hemorrhagic zones, as seen in Figure 15, but may be found quite remote from them. Interstitial ring hemorrhages around blood vessels in an otherwise non-hemorrhagic zone of lung tissue are quite striking. If the surface of the lung has been lacerated, considerable air and blood may accumulate in the pleural cavity, and some degree of pulmonary collapse results.

Injury to the bronchial wall is generally quite marked, even in relatively mild degrees of injury. This feature has been pointed out by Cameron et al.,[3] Rössle,[35] and Cohen and Biskind.[8] This feature is easily overlooked in the vast hemorrhagic sea where it is most frequently found. Large areas of lining epithelium may literally be stripped from the surface of the bronchial wall, leaving only a bare basement membrane (Figures 16 and 17). Elsewhere, the epithelium may show little change, or it may have a flattened and frayed appearance, with loss of cilia. There also may be considerable hemorrhage in the various strata of the bronchial wall, occasional disruption of the muscular zone, and even local derangement of cartilage plates in the most severely injured cases. In such zones one occasionally may be fortunate enough to find a point where blood is being extruded into the lumen through a gap in the disrupted bronchial wall (Figure 17). Whether the epithelial stripping represents a reflection of spalling effect or of shearing forces disrupting the fine capillary blood supply is not known. Bronchial mucosal injury appears of sufficient frequency that one would surmise it would complicate the picture considerably, particularly with regard to recovery from blast injury. A significant loss of cilia would seem to seriously hamper the removal of secretions, enhance the growth of micro-organisms at the injured site, and delay reparative restoration.

2. The Upper Respiratory Passages

The mucosal lining of the trachea, larynx, and nasopharynx may have patchy areas of ecchymosis which, in the case of the trachea, often shows a transverse linear configuration corresponding to the underlying cartilaginous rings or plates. Some focal loss of epithelial lining may be found in microscopic sections of the hemorrhagic areas, not unlike that found in the bronchi. Hemorrhage into the paranasal sinuses is another frequent finding, and splotchy areas of hemorrhage are found in the mucosal lining of the nasal passages and of the turbinate bones.

3. The Auditory Apparatus

The high frequency of tympanic membrane rupture has been alluded to previously as one of the results of blast injury. It is probably the most common event of blast injury and, in casualty situations, may be of valuable diagnostic assistance, although often overlooked. White[41] has reviewed the problem of eardrum tolerance to pressure loading. Inspection of the data of Zalewski,[49] whose data were tabulated by White[41] as reproduced in Table 1, shows that the average pressure required to rupture the human tympanic membrane varied with age from about 33 psi for the first decade to near 20 psi for the older age groups. Minimum pressures for rupture ranged as low as 5.4 to 6.4 psi. It would seem rather likely that the eardrum would be less tolerant to dynamic rather than static pressure loading, but the point has not been proven as yet. Under field study conditions with dogs, the minimal pressure for rupture was 4.1 psi, whereas the highest overpressure without rupture was 66.6 psi.[44] It is to be noted that threshold levels are in the order of 4 - 6 psi, a value which is less than half the overpressure level necessary to produce minimal evidence of pulmonary injury (about 12 - 15 psi). Other findings are not infrequently seen, such as hemorrhagic blebs on the tympanic membrane, hemorrhage into the middle ear cavity, disruption of ossicles, and in some instances, hemorrhage in the adjacent mastoid bone cells.

4. The Orbit

A peculiar reproducible injury to the orbit has been found to occur in dogs that were exposed to long-duration, shock-tube overpressures, and has been well described and illustrated by Richmond et al.,[29] who refer to a previous description by Osborn.[28] This injury has been termed a "blow-out" fracture, in which portions of the orbital wall, medially, were displaced into the nasal fossa. The fractures involved portions of the frontal, sphenoid, and lacrymal bones, as illustrated in Figure 18. The lesion presumably results in the transmission of the pressure pulse by way of the fluid content of the orbit, sufficient to overcome the strength of the thin bony plate at this point. From Richmond's data, the lowest overpressure range associated with this injury was 141 to 160 psi, but the incidence decreased when the rise time of the pressure pulse was extended for any given pressure level. The concealed nature of the injury may well be overlooked unless associated retrobulbar hemorrhage produces proptosis of the eye. The location of the fracture is hazardous, since fracture lines can extend posteriorly into the cranial zone (as has happened in one of the experimental animals) and open a thoroughfare for bacterial invasion of the meninges.

5. The Eye

The ocular globe appears extraordinarily resistant to injury by blast, even under casualty conditions. When secondary effects of

TABLE 1

Pressures Required to Rupture the Eardrums of Human Cadavers and Dogs when Pressure was Applied Within 24 Hours After Death*

Species Studies	No. of Cases	Age group in years	Pressures required to rupture tympanic membranes in psi		
			minimum	maximum	mean
Human	19	1 - 10	20.9	43.2	33.1
	15	11 - 20	6.0	44.1	25.5
	15	21 - 30	15.3	29.6	20.5
	17	31 - 40	6.4	35.4	23.2
	11	41 - 50	16.8	31.5	20.7
	12	51 - 60	13.5	38.3	21.0
	14	61 - 70	5.4	31.5	18.8
	8	Above 70	16.2	26.5	20.7
Total	111	Average	5.4	44.1	22.9
Dogs	10	Not stated	9.1	22.8	14.9

*Zalewski, quoted by White[41]

Figure 18 Orbital "Blow-Out" Fracture in Dog, Exposed to Overpressure Levels of 141 - 160 Psi. Broken Fragments were Displaced Medially into Nasal Fossae.

blast were excluded,[47] as from flying debris, the occurrence of injuries to the globe in casualty situations are rare indeed. An occasional account of hemorrhage into the anteroposterior chamber may be found, as well as transient episodes of blindness which might well have been attributable to the presence of air emboli. It is interesting that Cordes, quoted by Richmond et al.,[29] reported conjunctival hemorrhage, photophobia, and night blindness to be the most common findings in blast injuries of the eyes. There are a few descriptions[29] of torn retinal or iris vessels, crescent tears of the optic disc, acute iritis, and acute secondary glaucoma among bomb blast victims, but it must be realized that under these circumstances, it is often difficult to exclude the possibilities of indirect blast effects with absolute certainty. In our own laboratories and in several field experiments, we have carefully examined the eyes of large numbers of animals under many conditions in extreme blast injury. Except for slight pitting of the conjunctival and corneal surfaces from flying dust particles, we have never observed any significant injury to the ocular globe.

6. The Abdomen

Intra-abdominal injuries, in general, appear to be related to air-containing structures or tissues immediately adjacent to them. They are known to be more prevalent in cases of immersion blast injury, but are certainly not exclusive to it. Scattered petechiae or small ecchymotic blotches on the serosa and mucosa of the gastrointestinal tract, occasionally with small lacerations of the latter, are perhaps the most commonly observed findings. Perforation has been known to occur, with subsequent accumulation of air in the abdominal cavity and resulting peritonitis. In spite of pressures exerted on the diaphragm, we have not seen disruption of this structure unless the subject was displaced and exposed to violent impact. Small hemorrhages have occasionally been seen in the substance of the pancreas, adrenal glands, and kidney. Subcapsular hemorrhages and small parenchymal hemorrhages have been found in the liver and spleen. In a few instances actual laceration of the liver has been found.

The fluid-filled structures of the abdominal cavity have generally been stated to be free of injury, although there are distinct exceptions to this. In our field experience we have found several instances of severe tears in the urinary bladder mucosa of dogs. It is of interest that Zuckerman[52] mentioned a similar urinary bladder disruption in one of his rabbits.

7. Cardiovascular System

Direct bruising or contusion to the heart is most often apparent as a zone of superficial small hemorrhage, but this may be of larger dimension and extend deep into the myocardium. Tiny petechiae are rather commonly found along the endocardial lining of the ventricular

chambers, papillary muscles, and occasionally along the lips of the cardiac valve leafs. We have rarely seen an actual small laceration of the myocardium, but clinical reports indicate that, at times, actual cardiac rupture, disruption of the aortic cusps, and laceration of the first part of the aorta may occur.[48, 34] Whether these injuries are related to contusion, or a transmission of the high-velocity pressure pulse in a fluid media, or both, is a matter of conjecture.

8. The Phenomena of Air Emboli

Benzinger,[2] Desaga,[9] and Rössle,[35] in the early 1940's, seem to have been the first to emphasize the importance of air emboli as a direct cause of death and to have documented the high incidence in both the coronary and cerebrovascular circuits. In their studies among the early fatalities, death and the occurrence of air emboli went almost hand-in-hand. In some animals, death appears to occur without this phenomenon, but the low negative incidence does not dispense with its existence as an assumption, since the emboli are quite difficult to find unless the examination is done shortly after death and with meticulous attention to technique. (Artefactual introduction of air into the vascular circuit during prosection is familiar to pathologists.) Air emboli are much more easily seen in the animal heart because of the paucity of fat surrounding the coronary arteries, but one must remember that only a small portion of the coronary system can actually be visualized. In our own experience we can only confirm the contention of Benzinger,[2] Desaga,[9] Rössle,[35] and Clemedson et al.,[7] that the incidence of air emboli is proportional to the degree of blast injury, and parallels the rate of death. White and Richmond have observed the occurrences of air emboli in all species studied locally.[45] Figure 19 illustrates rather clearly the appearance of numerous air bubbles in the coronary arterial circuit of one experimental animal. The relationship of air emboli occurrence, degree of blast injury, and death are graphically illustrated in Figure 10.

There seems to be little doubt that the production of air emboli is probably the most significant cause of death after blast injury and accounts for subsequent cardiac failure and the sometimes bizarre central nervous system effects. When one considers the microscopic structures of the lung, particularly the relationships of the air spaces and the delicate structure of some portions of the venous circuit, it is not difficult to perceive that, with a rupture of the thin venous walls, considerable quantities of air will be introduced into the vascular system during the overpressure phase and with subsequent respiratory cycles. Rarely, as shown in Figure 20, direct fistulas have been observed in histologic sections from animal material after diligent searching.

In retrospect, there is ample physiologic and clinical evidence to support the occurrence of air emboli, both in animals and

Figure 19 Air Emboli in Anterior Coronary Arterial Circuit of Dog Heart Exposed to Lethal Overpressure

Figure 20 Alveolar-Venous Fistula in Dog Lung

in human material by reason of altered EKG patterns consistent with sudden cardiac anoxia and the rapid appearance of cerebral symptoms and signs, both usually occurring within 2 to 4 minutes. The studies of Clemedson et al.,[7] and Benzinger[2] have shown that the effects of air emboli can be greatly alleviated and the mortality greatly reduced by compression after blast exposure.

9. The Central Nervous System

In the central nervous system, the anatomic findings are rather meager except for the appearance of air emboli in the basilar arterial circuit and in its principal tributaries. This, of course, excludes effects due to direct trauma as might result from bodily displacement or from flying missiles. Although we have examined hundreds of slides of serially blocked brain tissue, we have seen only a few random petechiae. This is in accord with the experience of British and German investigators.[2, 9, 22, 35, 52]

While central nervous system symptoms can be produced by a wide variety of means, including generalized anoxic states and metabolic toxicity disorders, the occurrence of air emboli seems the only logical reason for the sudden onset of CNS signs after blast injury. The signs are various but include lethargy, ataxia, and even paralysis, as noted by Hooker[16] and Benzinger.[2] Krohn et al.,[22] reported electroencephalographic signs of circulatory disturbances (cerebral hypoxia) in monkeys exposed to blast. Some idea of the clinical behavior after blast injury can be seen in the study of 55 dogs reported by Richmond and White,[31] and summarized by them in Table 2. The animals all appeared to be conscious until very near death, but most of them could not walk even when placed on their feet. Only 5 of the 55 fatally injured animals were able to walk immediately after blast injury, and 3 or these managed the task with difficulty (staggering). Of the remaining 50, only 5 maraged some form of locomotion in the next 40 minutes. Of the 90 dogs which survived for 24 hours, only 6 did not walk at all; 30 could not walk initially, but recovered the ability in an interval of 3-1/2 hours afterwards.

10. Fat Emboli

A survey of pathologic findings in a type of traumatic injury would hardly be complete without some mention of the subject of fat emboli. The appearance of intravascular fat in such structures as the lungs, kidney, and brain after traumatic injury is a familiar entity and particularly prevalent where injury involves portions of the skeletal system.[1] However, it is known to occur in non-traumatic conditions such as following ether anaesthesia, in some cases of thermal injury, in subjects with very fatty livers, and in some patients who have been severely shaken up but without bone fractures. Clinically, the occurrence of fat emboli has been associated with symptoms of dyspnea,

TABLE 2

IMMEDIATE POST-BLAST BEHAVIOR OF SERIES OF DOGS EXPOSED TO MODERATELY SEVERE OVERPRESSURES[31]

Remarks		Number of Animals	
		Survivors	Fatalities
Walked immediately postshot		54 (60.0%)	(9.1%)
Walked at:	2 min	1	-
	6 min	-	1
	10 min	12	1
	20 min	3	-
	25 min	-	1
	40 min	-	1
	60 min	3	-
	90 min	6	-
	120 min	1	-
	180 min	3	-
	210 min	1	-
Failed to walk		6* (6.7%)	46** (83.6%)
Total Number		90	55

*Between test and 24-hour sacrifice.
**Between test and death — mostly short-survival times.

restlessness, cyanosis, and with cerebral signs, including paralysis. On the other hand, it must be admitted that there may be considerable argument centered around the physiologic significance of fat emboli in less than massive amounts. It is not an infrequent finding in the lungs of postmortem material, and such aberrations may be difficult to correlate at all with clinical events.[14]

In 1941, Robb-Smith[33] emphasized the potential hazard of fat emboli in a series of accident cases, only one of which was a blast injury. Somewhat later, Wilson and Tunbridge[48] studied material from a series of blast fatalities occurring in a Malta cave and failed to find any such emboli. This was essentially the experience of Zuckerman and of the German investigators. It is rather likely that among casualties from an explosion where trauma is a mixed affair, logically one could expect a few instances of fat emboli probably related to the indirect effects of blast. In our studies with many animals, we have not observed fat emboli in lungs, kidney, or brain, although this should not preclude the possibility of a remote occurrence. Should some intravascular fat appear, it would seem likely that the fat would quickly be filtered out by the pulmonary vascular bed and be of relatively minor significance in comparison with other blast effects.

Complications and Sequelae of Blast Injury

From what has been discussed about the acute physiologic and pathologic results and from what has been seen as to biologic tolerance of various species of animals discussed elsewhere,[32] one can gain some appreciation for the serious nature of exposure to rapidly rising overpressures, particularly those of relatively long duration. Even excluding damages that would occur from indirect effects, simple air blast has become one of the most significant factors in the production of casualties, with its dominant hemorrhagic effects in the delicate and vulnerable respiratory system, the high incidence of metastatic injury to the heart and brain by distribution of air emboli, and rapid death rate when exposure conditions are severe. With modern high-yield explosives of nuclear type, such effects are apt to encompass far greater numbers of victims per unit device, not only for the greater ground area exposed to overpressure, but also for the markedly lower threshold for injury when environmental overpressure is sustained. Thus, there is a need for rapid and proper medical care if significant salvage of other fatally injured persons is to be expected.

While there is a natural pulmonary vasoconstrictive mechanism tending to limit hemorrhage in the beginning and thus promote clotting, hemorrhage may be repetitive due to exaggerated cyclic respiratory motion to compensate for some reduced air exchange, and from coughing and exercise. This picture may be complicated by the onset of pulmonary edema related to cardiac decompensation, more often than not related to myocardial ischemia due to air emboli. Any

marked degree of hemorrhagic process in the lungs, coupled with loss or damage to the ciliated bronchial epithelium, becomes an invitation for growth of micro-organisms with resulting lobular pneumonia and possibly lung abscesses. Laceration of the lung surface or disruption of pleural blebs lead to pneumothorax, hemothorax, and even empyema. Within the abdominal cavity, the hazards of ruptured liver, spleen, intestinal tract, urinary bladder, with subsequent peritonitis, are too well known to deserve additional discussion here.

Excluding direct trauma to the head and severe hypoxia due to cardiac failure, it now appears that all significant central nervous system effects after blast are to be attributed to air emboli. The seriousness of this can only be related to the amount of air introduced into the cerebral arterial circuit and to the specific areas of the brain to which the emboli are distributed. Since cerebral tissue is notoriously intolerant of anoxia, it is quite likely that air emboli lodged in vital centers would lead to rapid death. Distribution of air emboli into less sensitive centers would lead to transient signs of disturbance in survivors. The duration of air emboli in the vascular circuit, once the source has been cut off, is among other things a function of the diffusion coefficients of the gases involved which tend to diffuse into the liquid phase and disappear.

All of the above description does not intend to imply that all survivors of blast injury must look forward to such a stormy course. As a matter of fact, many who survive serious injury seem to recover very nicely and the hemorrhages "melt" away very quickly, in a matter of 2 to 3 weeks. An example of the rapidity of clearing of hemorrhagic areas can be seen in Figure 21 with four photographs depicting the rate of clearing over a period of 21 days. Thus, the first shows the situation at the end of 24 hours, the second at the end of 3 days, the third at the end of 7 days, and the last at the end of 21 days, in which all blood seems to have disappeared. While it is beyond the scope of this presentation to discuss the possible sequelae of blast injury, a few brief comments concerning such would seem appropriate here. Because of the known pulmonary tissue disruption and the injury to bronchial and bronchiolar structures, we have been concerned about the possible stigmata which might be left in the respiratory system after the hemorrhagic process has resolved. In spite of the good general appearance of the recovered animals, the important question seems to be whether the injury, as extensive as it sometimes is, would result in impaired pulmonary function, decreased resistance to respiratory infection, and reduced longevity.

Since a desired degree of pulmonary injury can now be produced with remarkable precision in a shock tube, Dr. D. R. Richmond exposed a series of healthy mature dogs to blast overpressures calculated to be just under the LD_{50}. The surviving animals were subsequently closely observed and serially sacrificed at periodic intervals. In spite of the rapid resolution of the hemorrhagic areas (within 3 weeks),

Clearance Rate of Hemorrhage from Dog Lungs Exposed to Precisely Measured Blast Overpressures Near the LD_{50} Mark. Top Left: Gross Lung Appearance at End of 24 Hours. Top Right: Lungs at End of 3 Days.

Lower Left: Marked Clearing at End of 7 Days. Lower Right: Appearance of Lung at End of 21 Days. Reproduced from Color Illustrations.

Figure 21

it was surprising to learn that, in addition to the expected emphysematous change, the lung tissue contained many fine scars, particularly around the alveolar ducts, as illustrated in Figure 22. The number and position of these would suggest that one could expect at least some decrease in pulmonary reserve under stress. The ultimate fate of these scars is not known at the moment, but they apparently persist unchanged beyond a period of 2 months.

Concluding Remarks

The attempt has been made in this report to present a general clinical and pathophysiologic summary of the effects and seriousness of air blast injury such as to be of some practical interest to those who may be faced with the problems of care and management of casualties. In compiling the material the author has drawn liberally upon the valuable experiences and studies of the many who, during World War II, contributed so much to the fundamental understanding of blast injury. In addition, much valuable knowledge has been added to our present information from the studies of Clemedson[6] and those of White and Richmond,[29,30,31,41,42,44,45,46] particularly in regard to the biophysical effects of overpressure patterns on the biologic target.

Even though only part of the casualty picture has been presented here, direct blast injury is a remarkable, complex, and very hazardous phenomenon. Today, the problems of blast injury are made even more significant, not only because of the tremendous increase in explosive yield, but also to the far-reaching thermal energy, and to irradiation effects, either initial or residual, produced by nuclear detonations. As to the immediate casualty problem, injury and death due to blast and thermal energy – except for yields less than a few kilotons – far outweigh the biologic effects of ionizing irradiations.

While the factors of blast are much better appreciated than in the past, much more needs yet be done to achieve a thorough understanding of the various facets of this type of injury and of the relationships to the indirect effects of modern explosions. Nevertheless, much of the mystery of blast has been unraveled and experiments have demonstrated that not only are the biologic effects quite constant and predictable under controlled conditons, but that the animal itself is a very sensitive indicator of environmental variations in pressure.

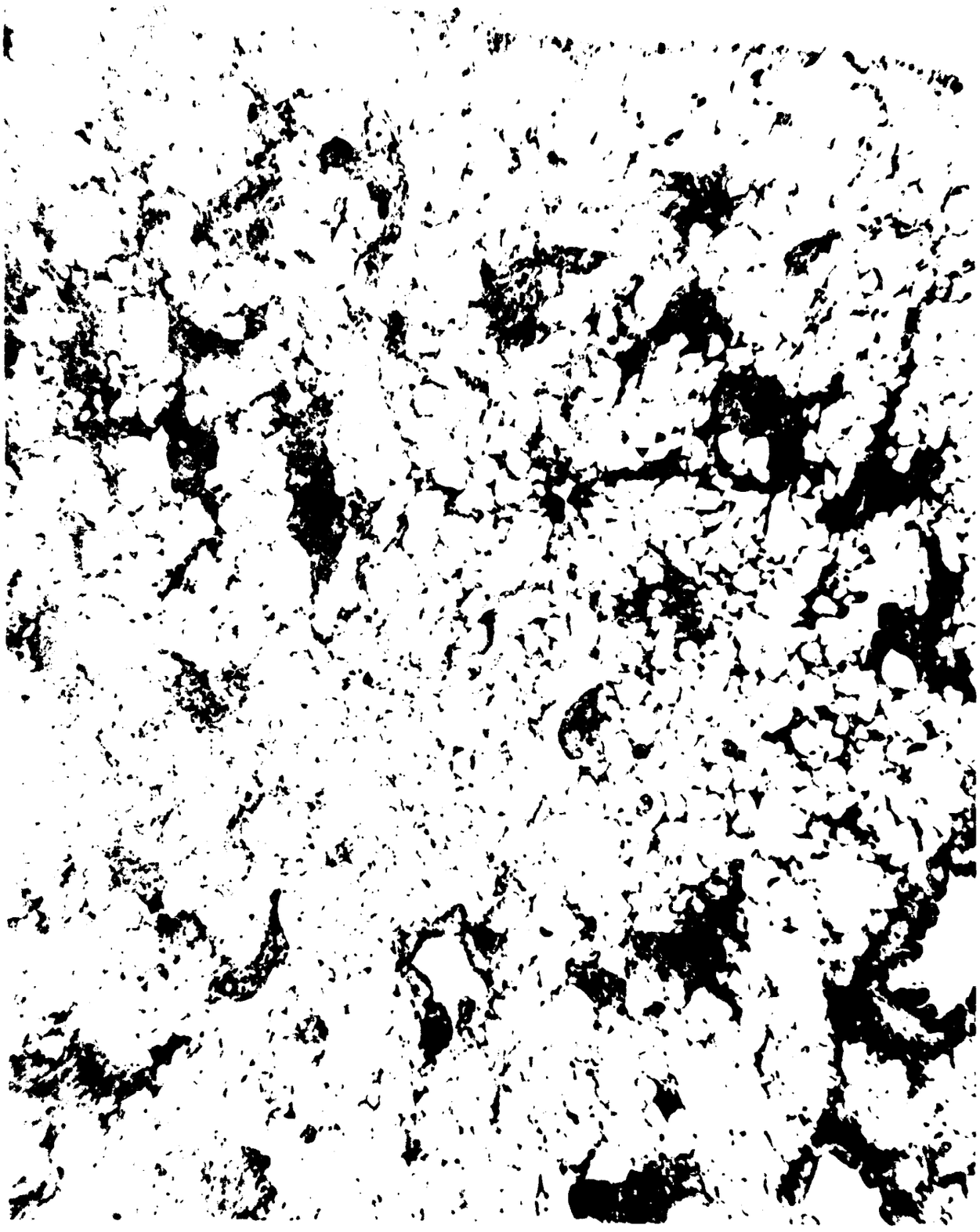

Figure 22 Sequelae of Blast Injury in Dog Lung. Multiple Fine Scars are Scattered about the Previously Injured Pulmonary Substance. X 25

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Blast injury is a complex and very hazardous phenomenon to the biologic target. Together with effects of thermal radiations from modern nuclear weapons, blast injury (direct and indirect) appears to be accountable for the vast bulk of early deaths and casualties in nuclear explosions. This article has attempted to summarize the important clinical, physiologic, and pathologic information concerning the effects of direct air-blast injury on the biologic subject. Certain features have been emphasized in order to assist the clinical medical officer towards proper management of casualties. A brief description of pulmonary sequelae of blast injury is included for completeness.

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