THE EFFECTS OF AMBIENT PRESSURE ON THE BIOLOGICAL RESPONSE OF MICE TO AIR BLAST

by

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ABSTRACT

Mice were mounted on the endplate of the expansion chamber of an air-driven shock tube and exposed to sharp-rising overpressures of "long" duration while under initial pre-blast pressures ranging from 7 to 42 psi absolute. The shock pressures were recorded by piezo-electric pressure transducers. A total of 672 mice were exposed to pressure changes in two series of experiments. In one series, the animals were held under the initial pressure for one hour following the blast before being returned to the ambient pressure level of the laboratory. In the second series, the pressure on the mice was returned to ambient immediately following blast exposure. In both series, animal tolerance, expressed as LD_{50} overpressures, increased linearly with increasing initial pressures.

The LD_{50}-1-hour gauge pressures in the first series were 20.3, 31.0, 44.5, 55.4, and 91.8 psi for initial pressures of 7, 12, 18, 24, and 42 psia, respectively. The LD_{50} pressures for the second series were below those of Series I for initial pressures above ambient and above them for initial pressures less than ambient. The values were 22.7, 37.9, 53.6, 61.3, and 68.4 psi for initial pressures of 7, 18, 30, 36, and 42 psia, respectively. These results indicated that pressurization of animals soon after blast exposure resulted in a decrease in the mortality whereas decompression resulted in increased lethality.

Practical and theoretical implications of the study were discussed. A promising but tentative procedure for scaling biological blast effects as a function of altitude was presented.

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CHAPTER 1

INTRODUCTION

The extensive use of high-energy fuels and the development of a wide variety of high explosives have led to increased accidental and wartime exposure of men to detonations. Furthermore, with the advent of the development of nuclear weapons, the need for a better understanding of the biological effects of blast has been greatly increased.

Explosions have three main effects leading to blast injuries. Injuries directly resulting from the pressure variations caused by a detonation are referred to as primary blast effects. The other two effects are injuries caused by debris thrown by a blast wave and those due to the translation of the body of an organism as a result of a detonation and are termed secondary and tertiary effects, respectively (White and Richmond, 1960).

For some 200 years, primary blast injuries have been documented and more recently extensively described in the medical literature (Clemedson, 1949). Yet, it was not until 1918 that Hooker first conducted experiments involving the exposure of animals to air blast in order to study the physiological effects of air concussion (Hooker, 1924). During World War II, Zuckerman (1940, 1941), Fisher, Krohn, and Zuckerman (1942), and Krohn, Whitteridge, and Zuckerman (1942) in England and Desaga (1950), Rössle (1950), and Benzinger (1950) in Germany conducted a number of investigations in blast biology. The

first extensive monograph on the subject was Clemedson's (1949) which presented an excellent comprehensive review of the literature. However, the rather extensive work of the German investigators, which was kept secret during the war, was not included in his review. Following World War II, Clemedson and his co-workers in Sweden began an intensive series of investigations in blast biology (Clemedson, 1949, 1954, 1954a, 1956, 1956a, 1957, 1958; Clemedson and Criborn, 1955, 1955a; Clemedson and Granström, 1950; Clemedson and Heilbronn, 1958; Clemedson and Hultman, 1957, 1958; Clemedson et al., 1954; Clemedson and Jönsson, 1961, 1961a, 1961b; Clemedson and Petterson, 1956; Carlsten, Clemedson, and Hultman, 1954; Celander et al., 1955, 1955a). With the advent of the testing of nuclear weapons, a broad program in the study of the biological effects of blast was initiated in the United States by White, Richmond, and their co-workers (White et al., 1957, 1961; White and Richmond, 1960; White, 1961; White, Bowen, and Richmond, 1962, 1964; Richmond et al., 1957, 1959, 1959a, 1961, 1961a, 1962; Richmond, Clare, and White, 1962; Richmond and White, 1962, 1964; Richmond, Pratt, and White, 1962; Clare et al., 1962).

In spite of the rather considerable attention devoted to the subject since World War II, there is at present no widely accepted, clear-cut concept of the biophysical mechanism of air-blast injury and many aspects of the problem are poorly understood.

Prominent among the physical processes which have been proposed as mechanisms of injury are the following: 1. Spalling effects - damage to a structure at an interface between two substances of different density which results from a negative reflection occurring at the surface of the denser medium when a shock wave travels from a

medium of greater density to one of less density (Schardin, 1950). 2. Inertial effects - structural shearing forces which arise as a result of differential acceleration, by the blast wave, of substances of different densities; 3. Implosion effect - the implosion and subsequent explosion of gas bubbles in a fluid through which a shock wave is traveling. As a shock wave travels through a fluid in which gas bubbles are present, the unequal pressures between the relatively non-compressible fluid and the compressible gas results in an implosion of the gas bubble. Due to the inertia of the moving fluid, as the pressure in the imploding gas bubble approaches that of the surrounding fluid, the kinetic energy of the inward moving fluid results in a continuation of the implosion until the pressure in the gas bubble is on the order of 100,000 atmospheres. The gas bubble then expands, acting, in effect, as the center of a small detonation. Schardin (1950) suggested damage could occur as a result of an implosion of the alveoli of an organism exposed to air blast. 4. Pressure Differential and the "squeeze effect" - this effect can best be understood if one considers the case of a pearl diver who descends, without diving gear, from sea level to increasing depths in water. If his lungs are filled to a total lung capacity of say 6000 cc when he starts his dive, then as he descends, the increasing water pressure causes a compensating volumetric decrease in the air in the lungs. At a depth of 33 ft, the water pressure would be two atmospheres and the volume of air in the lungs would be one-half of the volume at sea level, i.e., 3000 cc. If he descends an additional 66 ft, the water pressure would again be doubled and the volume of air in the lungs would again be reduced by one-half. Thus at a depth of approximately 100 ft, the pressure on the body would be

four atmospheres and the air in the lungs would be compressed into a volume of one-fourth of the original sea level volume, i.e., 1500 cc. This volume approximates the residual lung volume. Hence, due to the elastic resistance of the diaphragm and rib cage, if the diver descends to a greater depth, no further reduction in volume occurs. Consequently, the increasing water pressure is transmitted through the tissues and body fluids and there arises a pressure differential between the air pressure in the alveoli and the fluid pressure of the surrounding tissues. Fluids are therefore forced from other regions of the body into the thoracic region resulting in the occurrence of pain in the chest, lung hemorrhage, and edema. This phenomenon is known as the "squeeze syndrome" of divers. It has been suggested that the action of a "long" duration blast wave could produce comparable effects on an organism because the pressure is transmitted through the body much more rapidly than through the respiratory passageways (White, 1959).

Two of these four proposed mechanisms of injury involve pressure-volume relationships. Therefore, the ambient pressure, existing at the time of blast loading of a biological target, might be expected to affect animal tolerance to air blast (White, et al., 1957). For this reason the present investigation has been undertaken in order to facilitate definition of the mechanisms involved in blast injury.

An important purpose of this study was to develop shock-tube and related techniques for exposing animals to air blast at different ambient pressures and to explore the tolerance of mice to "sharp"-rising overpressures of "long" duration as related to pre-shot ambient pressures ranging from a fraction of an atmosphere to several atmospheres.

CHAPTER 2

SURVEY OF LITERATURE

The literature from which background information and theory have been drawn in the course of this investigation can be considered in five categories: (a) blast and shock tube physics; (b) relationships between blast parameters and biological response; (c) primary blast injuries; (d) air embolism; and (e) biological effects of pressurization and decompression. A brief summary of the pertinent information is presented below.

2.1 Blast and Shock Tube Physics

A nuclear or high-explosive detonation at the surface in air produces a hemispherical shock wave which expands radially from the charge (Glasstone, 1962; Schardin, 1950). This shock wave, before interaction with any obstacles is termed the incident shock. The variation of pressure with time occurring at some point on the surface, over which an incident shock wave travels, may be recorded by means of a pressure transducer mounted side-on to the advancing shock (Granath and Coulter, 1962). According to the pressure-time waveform, when the shock front passes over the gauge, the pressure rises almost instantaneously to a peak, decays exponentially with time back to the original ambient pressure level, dips below this level, and again levels off at ambient. The peak pressure of the incident shock wave is termed the incident or local static pressure, and is also referred to as the

shock overpressure. That portion of the wave during which the pressure is above ambient is referred to as the positive phase. The duration and magnitude of the positive phase of a blast wave are dependent upon the yield of the charge and the distance from it. For a given distance, the magnitude and duration of the overpressure increase with increasing yield. For a charge of a given yield, the duration increases and the overpressure decreases with increasing distance from the detonation (Glasstone, 1962).

The duration of the negative phase of the blast wave is approximately five times the positive phase, but the absolute value of the underpressure is always much less than the overpressure. Furthermore, as the underpressure can never exceed the value of the ambient pressure, it is a relatively unimportant phase of the blast wave as far as producing effects on materials is concerned.

A pressure transducer mounted face-on to the advancing shock wave generally records a higher peak pressure than a gauge mounted side-on because of the force exerted by the blast winds. This face-on pressure is called the stagnation pressure. The difference between stagnation pressure and incident pressure is termed dynamic pressure. The latter is a function of static overpressure and ambient pressure (Glasstone, 1962).

When an incident shock wave strikes a plane surface which is perpendicular to it, the shock reflects against the surface. The resultant pressure acting on the surface, referred to as the reflected overpressure, is much greater than the pressure of the incident shock. The peak pressure of the reflected shock may be two- to nine-fold the incident pressure depending on the magnitude of the latter (Glasstone, 1962).

Blast waves can be produced and controlled for use in experimental studies by means of shock tubes. A simple air-driven shock tube consists of a compression chamber and an expansion chamber which are separated by a frangible diaphragm. When the compression chamber is pressurized and the diaphragm is ruptured, a shock wave is produced and travels down the expansion chamber. The duration of the positive phase of the shock wave is dependent upon the length of the compression chamber compared to the distance down the expansion chamber at which the recording gauge is positioned (Bleakney, Weimer, and Fletcher, 1949).

If the expansion chamber is closed by means of an endplate, the shock wave will reflect against the endplate and pass back through itself. The duration of the reflected shock will therefore be a direct function of the duration of the incident wave (BRL Report No. 1390).

2.2 Relationships Between Blast Parameters and Biological Response

As early as 1924, the work of Hooker indicated the tolerance of animals to air blast could be related to both magnitude and the duration of the positive phase of the blast wave. Recent studies, however, have shown there exists for each species of small animals a "critical duration". Changes in the duration beyond this critical value have little if any effect on animal response. Animal tolerance, expressed as LD_{50} overpressures, rises sharply with decreases in the duration below the critical level (Richmond, 1962). Other studies have indicated the shape of the leading edge of the pressure-time waveform has a significant effect on animal tolerance to air blast. In general, blast waves of the "ideal" type, i.e., those in which the pressure rises almost

instantaneously to a maximal level, have a greater primary blast effect than such "non-ideal" types as those which exhibit a slow, smooth rise or reach a maximum in a series of steps (White et al., 1957; Richmond et al., 1957, 1959; Froboese and Wunsche, 1959; Clare et al., 1962; Richmond, Clare, and White, 1962).

White et al. (1957) analyzed the biological effects of step-rising pressures occurring inside protective shelters following a nuclear detonation and considered the possible relationship between biological effects and the pressure ratio, $\Delta P/P_i$ (where ΔP is the increase in pressure and P_i is the initial pressure from which the increase originated). It was pointed out that the effect of ambient pressure on animal tolerance to air blast had not been investigated and it was suggested that the local ambient pressure existing at the time an animal is exposed to air blast might have a significant effect on animal response.

2.3 Primary Blast Injuries

An organism which dies from primary blast may show no external signs of injury except for the presence of blood or bloody froth at the mouth or nostrils. Internal injuries observed at autopsy follow a typical pattern. The areas of greatest injury are those regions of the body where the greatest difference of density of tissues exist. The lungs and other gas-containing organs are especially susceptible to primary blast injury. Typically, massive lung hemorrhage occurs and frequently there is arterial air embolism. The air evidently gains access to the pulmonary circulation in the blast injured lung and passes via the pulmonary veins and left heart to the arterial system. A number of investigations conducted by Zuckerman and his

co-workers in England and Desaga and Benzinger in Germany during World War II have shown that the cause of lung injury cannot be attributed to the passage of the shock wave down the trachea to the lungs but rather to a transmission of the pressure from the blast wave through the body wall (Zuckerman, 1940, 1941; Benzinger, 1950; Desaga, 1950). The possibility of pulmonary capillary rupture resulting from the action of the suction wave through the upper respiratory passageways has also been eliminated by the results of experiments reported by these workers.

2.4 Air Embolism

Air embolism is a major cause of early lethality from blast injury (Benzinger, 1950; Clemedson, 1954). The literature on air embolism is extensive. Papers which were found especially helpful were those of Chase (1934), Ruckstinat and Lecount (1928), Birch (1950), Moore and Braselton (1940), Geoghegan and Lam (1953), Fine and Fischmann (1940), Oppenheimer *et al.* (1953), Durant (1935), and Durant *et al.* (1947, 1949, and 1954).

Prompt pressurization of animals following blast exposure would be expected to result in reductions in the volumes of any air emboli which might be present in the circulation. Their effectiveness as a potential cause of lethality might thus be reduced. The results of experiments reported by Benzinger (*op. cit.*), and Clemedson (*op. cit.*) supported this prediction.

Moore and Braselton (*op. cit.*) found that air emboli resulting from the experimental injection of air into the pulmonary veins of cats were eliminated from the circulatory systems of the surviving animals to the extent that they were no longer detectible within five

to twenty minutes from the time they were first observed in the coronary arteries.

According to Chase (op. cit.), there are two main effects of air emboli in rabbits: (1) mechanical obstruction and (2) an irritant or neurovascular effect leading to a localized constriction of the affected arteries or arterioles followed by perivascular hemorrhaging.

2.5 Biological Effects of Pressurization and Decompression

In the course of this investigation, it was necessary to compress and decompress animals in addition to exposing them to air blast at various experimental ambient pressures. In this regard, Behnke's (1951) statement that the body can be compressed to almost eighteen atmospheres or decompressed to pressures equivalent to altitudes of 50,000 feet without physiological change attributable to the pressure per se was of particular interest. On the other hand, a pressure differential of less than 50 mm Hg (1 lb/sq in.) between the tissues and the ambient atmosphere will result in alteration of the shape of the tissues and the induction of edema, hemorrhage, and pain (Ibid.).

Other papers of special interest in this category are those of Luft and Bancroft (1956), Haber and Clamann (1953), Kolder (1954 and 1957), Kolder and Wohlzogen (1957), Schaefer et al. (1958), Furry (1962), Vavala (1954), Wünsche (1963), Harvey (1950 and 1951), and Hitchock (1951).

CHAPTER 3

MATERIALS AND METHODS

3.1 Nomenclature

The following symbols were used in connection with the study and in the preparation of this paper:

P_o: The ambient air pressure at the laboratory.

P_i: The initial environmental air pressure, i.e., the experimental ambient pressure.

P_s: The overpressure of the incident shock wave.

P_f or ΔP: The overpressure of the reflected shock wave and hence the total explosive change in pressure to which the animals were subjected.

P_b: The absolute pressure in the shock tube after the pressure has stabilized immediately following the shot.

psia: Pounds per square inch, absolute pressure.

psig: Pounds per square inch, gauge pressure, i.e., overpressure.

LD_{50}-1-hr: Overpressure required to produce 50% lethality within 1 hour.

LD_{50}-24-hr: Overpressure required to produce 50% lethality within 24 hours.

3.2 Shock Tube

A conventional, cylindrical shock tube, 19 ft 6 in. long and 12 in. in diameter, was modified and used to expose mice to air blast at different ambient pressures. The tube had a wall thickness of

3/8 in. and, as shown diagrammatically in Figure 1, was divided by a frangible diaphragm into a compression chamber 2 ft 6 in. long and a 17-ft expansion chamber. The latter was closed with an endplate on which animal cages were mounted.

Appropriate pipes and valves, to allow pre- and post-shot control of the pressure inside the shock tube, were fitted to the expansion and compression chambers. Multiple layers of Dupont Mylar plastic were employed as a diaphragm. According to the results of tests conducted with this tube, the Mylar sheets exhibited a consistent bursting pressure. Therefore, the desired exposure pressures were achieved by varying the total thickness of the plastic and allowing each diaphragm to rupture spontaneously as the compression chamber was increasingly pressurized.

3.3 Pressure-Time Measurements

The techniques for measuring the shock overpressures and in the calibration of the blast gauges were described by Richmond *et al.* (1962), and Granath and Coulter (1962). On every test, the shock pressures were measured with piezoelectric gauges mounted side-on in the wall of the tube 6 in. upstream from the endplate (Figure 1). Occasionally, gauges were also located on the endplate to record the pressure-time wave form at the position of the animals. The piezoelectric transducers contained sensors of Lead Metaniobate (Model ST-2, Susquehanna Instruments, Bel Air, Maryland). Each signal from a pressure transducer was passed through a cathode follower and was displayed and photographically recorded on a cathode-ray oscilloscope. Typical pressure-time oscillograms obtained with the gauge mounted side-on in the wall of the tube are presented in Figure 1.

The approximate compression chamber pressures required to produce given shock overpressures at the various experimental ambient pressures were determined by means of the theoretical calibration curve shown in Figure 2. Also presented in Figure 2 is the empirical calibration curve constructed from the data obtained during the course of the study. The measured incident pressures were within 10% of those predicted by the theoretical relationships. These results, consistent with experience reported elsewhere (Bleakney, Weimer, and Fletcher, 1949; Lampson, 1950) as characteristic of air-driven conventional shock tubes, indicated the methods used to measure shock pressures were reliable at either reduced or elevated ambient pressures.

The overpressure in the expansion chamber before and after each blast was measured by a Bourdon-type dial pressure gauge (Heise Bourdon Tube Co., Newton, Connecticut). A mercury manometer gave the pressure levels when the expansion chamber was partly evacuated. The time required to increase or decrease the pressure in the expansion chamber was carefully measured with a stopwatch and also checked on oscillograms obtained with Quartz piezoelectric transducers (Model PZ-4, Kistler Instrument Corporation, North Tonawanda, New York).

3.4 Animal Exposure

In all, 672 female mice of the Webster strain were exposed to pressure changes. Their mean body weight was 19.7 g (standard error of the mean and range were ±0.84 and 16 - 24 g, respectively). Three animals were exposed per shot. Each animal was oriented right-side-on to the incident shock in an individual, cylindrical, wire-mesh cage mounted against the endplate. The diameter of the wire from which the cages were constructed was 1/16 in. and the inside diameter of

the squares of the mesh was 1/4 inch. The cages were arranged 2 in. apart, one above the other. Since the endplate of the tube was oriented normal to the incident shock wave, the animals were subjected to the incident and the reflected shock almost simultaneously. Consequently, the air-blast dose was taken to be the maximal overpressure in the reflected shock. Figure 3 presents a pressure-time wave typical of the reflected shock waves recorded by a pressure transducer mounted on the endplate of the shock tube. The duration of the positive phase of the primary blast wave was 16-20 msec, which is much longer than the "critical duration" for mice (Richmond et al., 1962).

Following the first positive wave, the animals were subjected to a series of decreasing secondary pressure pulses resulting from the reflection of the shock wave from one end of the tube to the other. Pressure-time record "a" in Figure 1 is a typical oscillogram showing these multiple reflections.

3.5 Experimental Procedure

To conduct the experiments with the available laboratory equipment, the procedure adopted involved the following four basic steps:

1. The environmental air pressure on the animals was changed to the desired level (P_i).
2. The animals were subjected to the air blast.
3. The pressure on the animals was returned to the initial pre-blast level (P_i).
4. The environmental pressure was then returned to the laboratory ambient pressure level (P_o) and the biological effects were assessed.

The assessment of the biological effects had to be made at ambient pressure. As it was considered that the change in pressure from P_i to

ambient, following the blast, might alter the biological response, the following two series of experiments were conducted:

1. In Series I, the animals were retained under the P_i pressure for one hour following the blast before they were returned to the ambient pressure level of the laboratory.
2. In Series II, the mice were returned to the laboratory ambient pressure level immediately following blast exposure.

In order to check for possible biological effects of the various slow pressure changes to which the experiemental animals were subjected, control animals were exposed to the same compression and decompression phases of the experimental sequence (minus the blast phase) as the experimental animals. Controls were run on both series of experiments.

3.6 Series I

Two hundred seventy mice were exposed to air blast while at initial pressures of 7, 12, 18, 24, and 42 psia. At each experimental ambient pressure, animals were exposed to three or more levels of blast overpressures. Fifteen or more mice were exposed at each overpressure level. Immediately after the blast, the pressure in the expansion chamber was quickly adjusted to the respective pre-shot level and then held for one hour before it was returned to the laboratory ambient level.

The five overall pressure-time profiles for Series I experiments are illustrated in Figures 4a - 4e. Indicated are the times required to increase or decrease the pressure on the animals before and after the blast. For instance, Figure 4c shows that 25 seconds (t_1) were

required to increase the pressure from the atmospheric ambient (P_o) of 12 psia to the initial pressure (P_i) of 18 psia in the expansion section. It was held for 78 seconds (t_2) before the blast. The duration of the blast wave itself was 0.016 seconds (t_3). After the blast, the pressure stayed at P_b (27 psia) for 2 seconds (t_4) before it could be reduced to the pre-shot level in 18 seconds (t_5). At the end of the 1-hour hold (t_6), the pressure was returned to ambient in 15 seconds (t_7).

Controls

Except for exposure to blast overpressures, 16 control animals were subjected to the pressure-time sequence illustrated in Figure 4e.

3.7 Series II

Two hundred eighty-five mice were exposed to air blast at initial pressures of 7, 18, 30, 36, and 42 psia following the general procedures used in Series I animals, except they were returned to the ambient pressure level of the laboratory immediately after blast exposure. The rates of pressure changes previous to and following the blast were kept similar to those in the Series I studies, except for the absence of the 1-hour hold period (t_6).

Controls

Fifteen Series II control animals were handled as Series I controls except they were not held for an hour at the pre-shot pressure (P_1) of 42 psia.

3.8 Analysis of Data

Probit analysis (Finney, 1952) was applied to the one-hour and 24-hour mortality data in order to obtain the value of the reflected

overpressure (P_f) associated with 50% lethality (LD_{50}) for each P_i. The analyses were performed by means of a Bendix G-15 computer which was programmed to provide probit regression equations relating per cent mortality in probit units to the log of the reflected pressure. The computer was also programmed to run parallel probit analyses on groups of the individual probit regressions, to determine whether the slopes of the dose-response curves were significantly different at the 95% confidence level, and to fit parallel probit regression lines to the data.

3.9 Autopsies

Animals surviving the blast exposure were sacrificed with chloroform on the day following the test. The animals killed by the blast and the survivors were autopsied. The gross pathological effects were recorded on a systematic protocol. The presence of blood or bloody froth at the mouth or nostrils, hemothorax, pneumothorax, hemoperitoneum, air embolism, and lesions on the intra-thoracic and intra-abdominal organs were noted.

The weight of the lungs of each animal was recorded. Clemedson's (1949) quotient of lung injury, W_1/W_o (W_1 = the weight of the blast-injured lung and W_o = the calculated weight of the normal lung for an animal of the same size as the blast-injured animal, obtained from a regression of lung weight on body weight based upon data from 116 control mice), was the criterion for determining the extent of lung injury. The quotient of lung injury is a measure of the increase in weight of the lung due to hemorrhage and edema. The data obtained from lungs having suppurative inflamation or other obvious infections were omitted.

CHAPTER 4

RESULTS

4.1 Mortality

Series I

Presented in Table I are the 1-hour and 24-hour lethality and the overpressure in the reflected shock for each P_i at which the animals were exposed to air blast in the Series I experiments. Figure 5 presents the dose-response curves obtained by probit analysis of the 1-hour mortality data. Statistical tests demonstrated the slopes of these five probit regressions were not different at the 95% confidence level. Therefore, they were adjusted parallel (Finney, 1952).

The LD_{50} reflected shock pressures with 95% confidence limits and the probit regression equations' constants are listed in Table 2 along with the respective number of animals. The reflected pressure required for 50% lethality rose as the initial pressures were increased. The LD_{50} pressures were 20.3, 31.0, 44.5, 55.3, and 91.8 psig for mice exposed at initial pressures of 7, 12, 18, 24, and 42 psia, respectively. Note that the LD_{50} values are gauge pressures and therefore are above the initial pressures. The value of each LD_{50} differed from the others at the 95% confidence level. A Bendix G-15 computer was programmed to fit a regression of the form, $\log y = a + b \log x$, to the data. Figure 6 presents the regression and a log-log plot of the data. Actually, the LD_{50} values increased linearly with increasing initial pressures.

Series II

Table 3 lists the 1-hour and 24-hour lethality and the associated reflected pressures for each of the five experimental ambient pressures used in Series II. The LD_{50}-1-hour reflected shock pressures and probit equation constants obtained from these data are presented in Table 4. The LD_{50} values were 22.7, 37.9, 53.6, 61.3, and 68.4 psig for initial pressures of 7, 18, 30, 36, and 42 psia, respectively. As illustrated in Figure 6, the LD_{50} values were below those of Series I at initial pressure greater than the laboratory ambient pressure (12 psia) and above them for initial pressures less than ambient.

4.2 Controls

Results of control experiments revealed that the most rigorous combinations of decompression or compression, hold, and release of pressure (without the blast) encountered in this study, by themselves, produced neither deaths nor noticeable injury in mice. For instance, groups of animals were compressed to 67 psia in 225 seconds, held at that level for two minutes. They were then returned to 42 psia and held for one hour after which the pressure was reduced to 12 psia in 34 seconds. In addition, mice were compressed to 67 psia in 225 seconds, held there for two minutes, and then returned to 12 psia in 56 seconds. The animals exhibited no detectible effects from these pressure changes.

4.3 Time of Death

Tables 5 and 6 present the time-mortality data for the two series of experiments. For animals that died within one hour in Series I, the exact time of death was not determined. They were retained within

the shock tube under the initial pressure, P_i, for one hour following the shot. Animals that showed signs of rigor mortis when first removed from the tube were recorded as 30-minute deaths and others that were dead when first observed were recorded as 1-hour deaths. The data are plotted in Figures 7 and 8 to show the cumulative per cent mortality as a function of time. For the higher P_i's, the mortality rose more rapidly in Series II than in Series I. Thus, decompression immediately following blast not only increased mortality, but also reduced the survival time of the mortally injured animals. For P_i = 42 psia in Series I (Figure 7), the sharp increase in mortality at 60 - 65 minutes post-shot (i.e., within five minutes following decompression) suggested that the decompression, even after 1-hour hold, was not entirely without effect.

4.4 Pathological Observations

Lung Injury

As expected, there was extensive lung damage in the animals which were killed or severely injured by the air blast. The data are presented in Tables 7 and 8. Listed in the tables are the quotients of lung injury (weight of the lungs divided by the expected, normal lung weight) for the survivors and the fatalities, the ratio of the reflected pressure, P_f, to the initial pressure, P_i, and the per cent mortality for each group. The quotients of lung injury were higher for the fatalities than for the survivors in all groups in both series with the exception of Group I, P_i = 7 psia, in Series II (Table 8). However, there was only one fatality in this group. It is not surprising that, at the higher P_i's, the survivors in Series II generally had lower quotients of lung injury than comparable groups in Series I.

For example, of 15 groups of survivors at P_i's of 18 and above in Series II, only one group had a mean quotient of lung injury greater than 1.50 (P_i = 18 psia, Group IV). On the other hand, in Series I, five of nine groups of survivors at P_i's of 18 and above, had mean quotients of lung injury greater than 1.50. Another way of expressing this is that the animals which were held at P_i for one hour following blast exposure before being decompressed to ambient pressure were able to survive with greater lung injury than those which were decompressed immediately after the air blast. It is also notable that the quotients of lung injury in the Series II survivors were generally lower at the higher P_i's than at the lower initial pressures. Thus the data indicated relationships between the ability of the animals to survive on the one hand, and the extent of lung injury, the magnitude of the decompression following blast, and the time at which the decompressiion occurred, on the other.

Hemothorax and Pneumothorax

Observations of the occurrence of hemothorax and pneumothorax in the fatally injured animals in the two series are summarized in Table 9. There was a higher incidence of both hemothorax and pneumothorax at the higher than at the lower P_i's. The volume of air involved in any given case of pneumothorax would of course be increased during decompression. Thus, at the higher P_i's, animals exhibiting pneumothorax frequently had so much intra-thoracic air that the diaphragm was greatly bulged into the abdominal cavity.

In the experiments in which P_i = 42 psia, the incidence of pneumothorax was considerably greater in Series I than in Series II (Table 9). There are two main factors which may share the responsibility for this difference. Animals in Series I were subjected to

higher overpressures (cf. Tables 1 and 3) than those in Series II. In addition to this, the animals in Series I were held at P_i for one hour post-blast before being decompressed to ambient. During this hold time, air passageways in the lungs of the fatally injured animals may have become effectively blocked as a result of blast-induced lung hemorrhage. Air trapped in localized areas of the lung may have then been forced through the lung wall into the pleural cavity at the time of decompression. The animals in Series II were decompressed immediately post-blast thus providing very little time for the blocking of air passageways by hemorrhage.

Coronary Air Embolism

In both series, the one-hour fatalities which had been subjected to air blast while under an initial pressure of 42 psia were assessed for the occurrence of coronary air embolism. Decompression from 42 psia to ambient (12 psia) resulted in an increase in the volumes of air emboli by a factor of 3.5. This greatly facilitated in the assessment for the occurrence of air embolism in these animals. Of the 25 one-hour fatalities in Series I, 15 (60 per cent) were observed to have coronary air emboli. In Series II, 20(66.7 per cent) of the 30 one-hour fatalities exhibited the occurrence of coronary air emboli. Thus, at this P_i, there was very little difference in the incidence of air embolism among the fatalities in the two series. It should be pointed out, however, that there are several factors which may have played a part in the occurrence and distribution of intravascular air in these animals. Some of these are discussed later.

CHAPTER 5

DISCUSSION

5.1 Comparison of the Two Series

This study was designed to explore the effects of ambient pressure on the tolerance of mice to air blast. The animals in both series exhibited unequivocal increases in resistance to overpressure with increases in the experimental ambient pressure. The LD_{50} reflected pressures increased linearly with increases in the pre-shot initial pressure in both the Series I and Series II experiments. The tolerance of animals held at the pre-shot ambient pressure level for one hour following blast-exposure before being returned to the ambient pressure level of the laboratory (Series I) rose fourfold with a sixfold increase in experimental ambient pressure. The mice which were returned to the ambient pressure level of the laboratory immediately after exposure to the air blast (Series II) showed only a threefold increase in tolerance associated with the same sixfold increase in experimental ambient pressure.

Figure 6 illustrates the differences in the results from the two series. These data indicated pressurization of the animals soon after blast-exposure resulted in a decrease in the lethality (Series II, P_i = 7 psia). On the other hand, decompression carried out soon after blast-exposure (viz. all of the Series II experiments in which P_i was greater than 12 psia), resulted in increased lethality.

These effects were not surprising since air embolism is known to be a major cause of early lethality in blast-injured animals. Post-shot pressure changes following soon after blast exposure would be expected to produce compensatory changes in the volumes of the air emboli, thus altering their effectiveness as a potential cause of death.

In the Series I animals, the air emboli had time to produce their biological effects or be largely eliminated from the circulation (Moore and Braselton, 1940), during the hold period, before the animals were subjected to any post-shot pressure changes. Therefore, the results of Series I are undoubtably a truer representation of the effects of ambient pressure on blast tolerance than those of Series II.

As noted in Chapter 4, three of the animals exposed to air blast in Series I, at an initial pressure of 42 psia, died soon after decompression at the end of the one hour hold. It is remarkable that one of these animals died with coronary air embolism two minutes after having been decompressed even though the animal had been held for one hour post-shot before being subjected to the decompression. There are at least two possible explanations. The first is that blast-induced air emboli had not been sufficiently eliminated from the circulation during the hold period so that their volumetric increase at the time of decompression produced fatal results. The other, and perhaps more likely explanation, is that the air was injected into the circulation from the blast-injured lung at the time of decompression. The latter might be expected to occur if air is trapped in the distal airways as a result of intra-bronchial hemorrhage.

5.2 Multiple Reflections and the Immediate Post-Shot Pressure

As mentioned in Chapter 3, following the initial shock wave, the animals were subjected to a series of pressure pulses resulting from the reflection of the shock from one end of the tube to the other. Following these pressure reflections, the pressure became stabilized at a level above that of the experimental ambient pressure. This sequence differs from the true blast situation in the open where the pressure rapidly returns to ambient after the shock wave has passed. In order to assess the possible effects of these immediate post-shot pressure events, the tolerance of the animals which were exposed at the laboratory ambient pressure level (12 psia) in this arrangement was compared to that for mice exposed in a shock tube arrangement that provided shock waves of 3 to 4 msec duration which were free of subsequent pressure reflections (Richmond, 1962). The values, expressed as LD_{50}-24-hour reflected overpressures, were 29.6 and 29.0 psig, respectively. This close agreement supports the view that the repetitive, decreasing pressure pulses and the stabilization of the pressure at a level above the experimental ambient pressure had little, if any, effect on the results of the experiments.

5.3 Theoretical Implications of the Study

The results of these experiments lend support to the concept that a major biophysical mechanism of primary blast injury involves implosion of the lungs. When an organism is subjected to a sharp-rising overpressure of "long" duration, a pressure wave is transmitted through the body tissues (White and Richmond, 1960). The pressure is readily transmitted through the tissues and body fluids but due to the compressibility of gases the pressure of the air in

the alveoli becomes equal to the pressure of the surrounding tissues and body fluid only after having been reduced in volume by implosion of the alveolar wall and surrounding capillaries. The pressure of the air in the alveoli will equal the pressure of the surrounding fluids and tissues when the volume of the alveolus has been reduced by a factor equivalent to the ratio of the pressure of the fluids (which depends upon the overpressure of the blast wave) to the original pressure of the air in the alveolus, i.e., the ambient pressure. Thus, if injury occurs due to the effects of implosion of the alveoli, one would expect changes in the ambient pressure to require corresponding changes in the blast overpressure to produce the same biological effect. In fact, except for the effects of the inertia of the moving chest wall and diaphragm and the possibility of spalling effects, the ratio of the blast overpressure to the ambient pressure would be expected to be constant for an LD_{50} value. For example, at an ambient pressure of 12 psia (the ambient pressure at the laboratory in these experiments), 50% lethality was obtained when mice were exposed to a mean reflected pressure of 31.6 psig. Therefore, the ratio of the overpressure to the ambient pressure was 2.6. Thus, during the blast exposure, the pressure in the alveoli would become equal to the pressure of the blast wave when the lungs have been compressed to a size equivalent to 1/2.6 of the original lung volume. If the blast injuries leading to lethality result from a compression of this magnitude, at an ambient pressure of 18 psia, the overpressure required for 50% lethality would be expected to be 2.6 x 18 = 46.8 psig. In the Series I experiments, at an ambient pressure of 18 psia, the LD_{50} overpressure was 44.5 psig with 95% confidence limits of 41.9-47.4

psig. Thus, the results were in agreement with the predicted value. However, at the other experimental ambient pressures, the results approached the predicted values but varied from them in a specific way. Table 10 shows, instead of remaining constant with changes in the ambient pressure, the LD_{50} pressure ratio decreased slightly but consistently with increases in the ambient pressure. The LD_{50} pressure ratios were 2.90, 2.60, 2.47, 2.30, and 2.19 for experimental ambient pressures of 7, 12, 18, 24, and 42 psia, respectively. The experimental values were 12% greater than predicted at an ambient pressure of 7 psia and 16% less than predicted at an initial pressure of 42 psia. Hence, while the data support the contention that implosion of the lungs constitutes a major mechanism of primary blast injury, the consistent decrease in LD_{50} pressure ratios with increasing ambient pressure implies that other factors may also have been involved in producing the lethality in these experiments.

Factors which might have contributed to the lethality can be considered in two categories: (1) biophysical processes, in addition to implosion of the lungs, which might have produced blast injuries; and (2) conditions affecting the chances of survival of the blast-injured animals during the 1-hour post-shot hold period.

The major consideration in the latter category is whether elevated ambient pressure could have decreased - and reduced ambient pressure could have increased - the chances of survival of the blast-injured mice. Increased ambient pressure for prolonged periods of time may lead to oxygen poisoning. Controls held for one hour at the highest experimental ambient pressure were not affected by the pressure. Lung blast injury inhibits pulmonary function and leads

to anoxemia (Clemedson, 1949). Therefore, chances of oxygen toxicity resulting from increased oxygen tension due to elevated ambient pressure would be less likely to occur in the blast-injured animals than in the controls. Furthermore, at reduced ambient pressure, where the oxygen tension was below normal, survival of the animals was greater than predicted. Therefore, variation of the results from the predicted values cannot be explained on the basis of the development of oxygen toxicity, and there is no reason to believe that the observed variation could have been caused by effects of the experimental ambient pressure during the hold period.

With reference to the first category, the results of this investigation do not support the contention that biophysical mechanisms operating independent of the ambient pressure had produced lethal injuries in these animals. This is evident from the data presented in Table 1. For example, there was no lethality among 12 mice exposed to blast overpressures of 62-69 psig while under an ambient pressure of 42 psia, whereas overpressures of this magnitude produced 100% lethality at all of the lower ambient pressures. It should be emphasized that the reflected overpressures presented here are not absolute pressures but gauge pressures and therefore pressures above the experimental ambient level. Spalling effects and the shearing of tissues as a result of inertial effects have been suggested as possible mechanisms of blast injury (Schardin, 1950). Since these effects are markedly influenced by the magnitude of the blast overpressure and are probably affected very little by the ambient air pressure, they evidently were not causes of injuries leading to lethality in these experiments.

Another factor which must be considered is the extent to which inertia of the moving chest wall and diaphragm contribute to the implosion of the lungs of an organism exposed to air blast. Because of inertia, the lungs are compressed into a smaller volume than that required for equalization of the pressure between the air in the lungs and the overpressure of the blast wave. Therefore, the pressure in the lungs rises above that of the blast wave. Increases in the blast overpressure result in increased acceleration of the chest wall during implosion. The intra-thoracic pressure would therefore rise faster, there would be less time for compensating air flow through the respiratory passageways and the damaging effects of the implosion might thus be greater than it would be at lower blast pressures. Since the magnitudes of the blast overpressures were greater at the higher ambient pressures in these experiments, increased effects of inertia relative to the overall process of implosion of the lungs may be the reason why the lethality was greater than predicted at the higher and less than predicted at the lower initial pressures.

Be this as it may, it is currently quite clear that the ambient pressure is indeed a physical parameter of major importance in specifying blast effects.

5.4 Practical Implications of the Study

In view of the results, consideration should be given to the practical problem of developing some system of scaling air blast tolerance to changes in elevation. Since the LD_{50} pressure ratios varied but little with changes in the initial pressure in these experiments, a tentative but simple method of scaling air blast

tolerance to changes in elevation or changes in ambient pressure would be to consider the LD_{50} pressure ratio as being a constant. For example, by extrapolation of an interspecies correlation involving six different mammals exposed to sharp-rising overpressures of 400 msec duration, an estimated LD_{50} of 50 psig has been obtained for man (Richmond and White, 1962). Since the data were compiled at an ambient pressure of 12.0 psia, the LD_{50} pressure ratio would be 4.2. Consequently, to obtain an estimate of the LD_{50} for other ambient pressures, one simply multiplies the ambient pressure in question by this pressure ratio. Thus, at sea level where the ambient pressure is 14.7 psia, the estimated LD_{50} would be 62 psig; while at 26,400 ft, where the ambient pressure is 5.2 psia, the LD_{50} would be 22 psig. Such a scaling procedure must remain tentative however, until data can be obtained from the exposure of other species to air blast at various ambient pressures.

5.5 Suggestions for Future Research

One of the purposes of this study was to develop shock tube and related techniques for exposing animals to air blast at altered ambient pressures. The following are suggestions for future studies:

1. Animals exposed to air blast at altered ambient pressures should be retained at the experimental ambient pressure for a period of time following blast exposure and tolerance values should be based on lethality within this hold period. A transparent window should be installed in the shock tube so the animals can be observed during the hold period.

2. The peak intra-thoracic pressure is an indication of the extent to which the lungs of an organism are subjected to implosion during air blast exposure. If there is a distinct relationship between the per cent mortality and the degree of implosion of the lungs of animals, the ratio of the peak intra-thoracic pressure to the ambient pressure should be constant for a given per cent lethality. This contention should be investigated by making intra-thoracic pressure measurements in animals exposed to LD_{50} over-pressures at different ambient pressures.
3. The effects of ambient pressure on the tolerance of animals to blast waves of short duration should be investigated. Exposure of small animals to detonations of small, high-explosive charges in a large detonation chamber in which the ambient pressure could be controlled might be used for this purpose.
4. The effects of ambient pressure on the tolerance of animals to step-rising pressures might be investigated by exposing animals in the sealed expansion chamber of a shock tube having an adjustable reflecting plate.
5. Pressurization of animals following blast injury might be expected to benefit the animals in two ways.
    1. The pressure <u>per se</u> would reduce the effects of air emboli.
    2. Increased oxygen tension accompanying the pressurization might be beneficial by alleviating anoxemia resulting from lung blast injury.

Comparative studies of the therapeutic effects of early pressurization of blast-injured animals with room air *versus* pressurization with a gaseous mixture calculated to maintain normal rather than elevated oxygen tension might be a useful procedure for illucidating the relative effects of these two factors.

CHAPTER 6

CONCLUSIONS

The following conclusions may be drawn from the results of this investigation:

1. The tolerance of mice to air blast increased linearly with increases in the ambient pressure. The relationship between ambient pressure and the LD_{50}-1-hour overpressure may be represented by the equation:

   $$\log P_f = 0.590 + 0.842 \log P_i \text{ or}$$

   more simply by

   $$P_f = 6.84 + 2.029 P_i$$

   where P_f is the LD_{50} reflected pressure and P_i is the ambient pressure.

2. Lethality from exposure to air blast was increased by early post-shot decompression and was decreased by compression.

3. Post-shot compression or decompression, carried out one hour after blast-exposure, had little. if any. effect on lethality.

4. Decompression, following soon after blast-exposure, resulted in a decrease in the survival time of the fatally injured mice.

5. The types of lesions sustained by mice exposed to air blast at different ambient pressures were not different from those generally reported in the literature on blast biology; only the overpressures required to produce injury and death differed with changes in ambient pressure.
6. The ratio of the LD_{50} overpressure to the ambient pressure decreased slightly with increases in the initial pressure. The values were 2.90, 2.60, 2.47, 2.30, and 2.19 for ambient pressures of 7, 12, 18, 24, and 42 psia, respectively.
7. The results support the contention that a major biophysical mechanism of injury resulting in lethality from exposure to sharp-rising pressures of "long" duration involves implosion of the lungs.

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TABLE 1

REFLECTED PRESSURE AND ASSOCIATED MORTALITY FOR MICE EXPOSED TO AIR BLAST AT DIFFERENT EXPERIMENTAL AMBIENT PRESSURES (SERIES I)

Group	Experimental Ambient Pressure, psia	Number of Animals	Mean Overpressure in Reflected Shock, psig	Per cent Mortality	
				1 hr	24 hr
I	7.0	3	12.3	0	0
II		15	20.4	40.0	40.0
III		15	22.3	80.0	80.0
IV		27	23.7	85.2	89.0
I	12.0	15	28.4	13.3	26.7
II		15	30.7	53.3	73.3
III		15	36.0	86.7	93.3
I	18.0	15	39.6	33.3	40.0
II		18	43.3	44.4	61.1
III		15	50.4	66.7	86.7
I	24.0	15	50.5	13.3	53.3
II		33	56.0	60.6	72.7
III		12	61.7	75.0	100.0
I	42.0	9	62.1	0	0
II		3	69.1	0	0
III		15	83.1	20.0	33.3
IV		15	89.4	60.0	80.0
V		15	115.3	86.7	93.3

TABLE 2

RESULTS OF PROBIT ANALYSIS OF THE SERIES I DATA

Initial Pressure, psia	Number of Animals	LD_{50}-1-hr Reflected Pressure (ΔP), psig	Probit Equation Constants	
			intercept, a	slope, b
7	60	20.3 (19.0-21.5)*	-14.481	14.889**
12	45	31.0 (29.3-33.3)	-17.254	14.889
18	48	44.5 (41.9-47.4)	-19.543	14.889
24	60	55.3 (52.4-58.3)	-20.948	14.889
42	57	91.8 (86.1-98.3)	-24.225	14.889
Total	270			

*Numbers in parentheses are the 95% confidence limits.
**Standard deviation of the slope constant, b, is ±2.154.

TABLE 3

REFLECTED PRESSURE AND ASSOCIATED MORTALITY FOR MICE EXPOSED TO AIR BLAST AT DIFFERENT EXPERIMENTAL AMBIENT PRESSURES (SERIES II)

Group	Experimental Ambient Pressure, psia	Number of Animals	Mean Overpressure in Reflected Shock, psig	Per cent Mortality	
				1 hr	24 hr
I	7.0	18	21.0	5.6	16.7
II		15	22.8	66.7	66.7
III		12	23.4	75.0	75.0
I	18.0	15	26.8	0	0
II		6	29.9	0	16.7
III		21	35.1	23.8	42.8
IV		21	39.0	61.9	76.2
V		6	46.8	100	100
I	30.0	15	46.5	13.3	13.3
II		15	50.8	40.0	40.0
III		15	59.2	73.3	73.3
I	36.0	9	33.6	0	0
II		18	48.7	11.1	11.1
III		15	62.8	33.3	33.3
IV		15	69.7	86.7	86.6
I	42.0	15	61.9	0	0
II		15	63.3	40.0	40.0
III		15	68.8	26.7	33.3
IV		24	71.0	83.3	83.3

TABLE 4

RESULTS OF PROBIT ANALYSIS OF THE SERIES II DATA

Initial Pressure, psia	Number of Animals	LD_{50}-1-Hour Reflected Pressure (Δp), psia	Probit Equation Constants intercept, a	slope, b
7	45	22.7 (21.0-24.6)*	-18.805	17.554*
18	69	37.9 (35.2-41.2)	-22.717	17.554
30	45	53.6 (49.4-58.7)	-25.359	17.554
36	57	61.3 (55.7-67.2)	-26.379	17.554
42	69	68.4 (64.2-73.2)	-27.211	17.554
Total	285			

*Numbers in parentheses are the 95% confidence limits.
**Standard deviation of the slope constant, b, is ±2.946.

TABLE 5

TIME-MORTALITY DATA FOR MICE HELD AT P_i FOR 1 HOUR POST-BLAST

(Series I)

P_i (psia)	Total Number Animals	Total Dead		Number and Percent of Mortally Injured Animals Dying Within the Indicated Time											
				With Rigor Mortis (30 min)		Total 1 hr		65 min		75 min		105 min		120 min		24 hrs	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
7.0	60	42	70.0	37	88.1	41	97.6	41	97.6	41	97.6	41	97.6	41	97.6	42	100
18.0	48	30	62.5	16	53.3	23	76.7	24	80.0	25	83.3	27	90.0	27	90.0	30	100
24.0	60	44	73.3	29	65.9	31	70.5	31	70.5	31	70.5	31	70.5	31	70.5	44	100
42.0	57	32	56.1	22	68.8	27	84.4	31	96.9	32	100	32	100	32	100	32	100

TABLE 6

TIME-MORTALITY DATA FOR MICE DECOMPRESSED IMMEDIATELY POST-BLAST

(Series II)

P_i (psia)	Total Number Animals	Total Dead		Number and Percent of Mortally Injured Animals Dying Within the Indicated Time													
				1-5 min		6-10 min		11-20 min		41-50min		51-60min		120 min		24 hrs	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
7.0*	45	22	48.9	17	77.3	19	86.4	19	86.4	20	90.9	20	90.9	21	95.5	22	100
12.0	45	29	64.4	16	55.2	17	58.6	21	72.4	21	72.4	22	75.9	22	75.9	29	100
18.0	69	30	43.4	20	66.7	21	70.0	21	70.0	22	73.3	22	73.3	22	73.3	30	100
30.0	45	19	42.2	18	94.7	18	94.7	18	94.7	18	94.7	19	100	19	100	19	100
36.0	57	20	35.1	18	90.0	19	95.0	19	95.0	19	95.0	20	100	20	100	20	100
42.0	69	31	44.9	30	96.8	30	96.8	30	96.8	30	96.8	30	96.8	30	96.8	31	100

*Since P_i for this group was below ambient, these animals were compressed when returned to ambient, rather than being decompressed.

TABLE 7

QUOTIENTS OF LUNG INJURY IN MICE - SERIES I

P_i (psia)	Group	Pressure Ratio P_f/P_i	Mortality Percent	Survivors		Fatalities	
				Number of Animals	Quotient of Lung Injury W_1/W_o*	Number of Animals	Quotient of Lung Injury W_1/W_o*
7.0	I	1.76	0	3	1.18±0.09		
	II	2.91	40.0	9	1.35±0.06	6	1.99±0.16
	III	3.18	80.0	3	1.86±0.25	12	2.39±0.14
	IV	3.38	85.2	3	1.64±0.27	23	2.12±0.09
12.0	I	2.37	13.3	10	1.71±0.11	2	2.43±0.20
	II	2.56	46.7	2	2.03±0.16	7	2.32±0.10
	III	3.00	86.7	1	1.64	13	2.46±0.10
18.0	I	2.20	33.3	9	1.46±0.09	5	2.20±0.08
	II	2.42	44.4	7	1.72±0.08	8	2.19±0.07
	III	2.80	66.7	2	1.76±0.27	10	2.24±0.19
24.0	I	2.10	13.3	6	1.55±0.07	2	2.06±0.08
	II	2.33	60.6	8	1.74±0.07	20	2.23±0.12
	III	2.57	75.0			9	2.64±0.16
42.0	I	1.48	0	8	1.16±0.09		
	II	1.99	20.0	10	1.38±0.08	3	2.22±0.04
	III	2.13	60.0	3	1.46±0.03	9	2.02±0.06
	IV	2.74	86.7	1	1.70	13	2.01±0.06

*Mean ± Standard Error.

TABLE 8

QUOTIENT OF LUNG INJURY IN MICE - SERIES II

P_i (psia)	Group	Pressure Ratio P_e/P_i	Mortality Percent	Survivors		Fatalities	
				Number of Animals	Quotient of Lung Injury W_1/W_o	Number of Animals	Quotient of Lung Injury W_1/W_o
7.0	I	3.00	5.6	16	1.56±0.06	1	1.55±0.00
	II	3.26	66.7	5	1.65±0.09	10	2.08±0.06
	III	3.34	75.0	3	1.58±0.16	9	2.07±0.11
18.0	I	1.49	0	15	1.30±0.07		
	II	1.66	0	5	1.46±0.06		
	III	1.95	23.8	12	1.46±0.09	5	1.95±0.11
	IV	2.17	52.4	8	1.71±0.07	11	2.19±0.08
	V	2.60	100			6	2.44±0.14
30.0	I	1.54	13.3	13	1.36±0.06	2	2.44±0.34
	II	1.69	40.0	9	1.24±0.10	6	1.96±0.12
	III	1.97	73.3	4	1.45±0.11	11	2.22±0.14
36.0	I	0.93	0	8	1.09±0.03		
	II	1.35	11.1	16	1.12±0.07	2	2.33±0.38
	III	1.74	33.3	10	1.39±0.10	5	1.99±0.22
	IV	1.94	86.7	2	1.42±0.08	13	1.92±0.12
42.0	I	1.47	0	13	1.17±0.13		
	II	1.51	40.0	8	1.01±0.06	6	1.87±0.09
	III	1.64	26.7	8	1.35±0.09	4	2.04±0.16
	IV	1.69	93.3	4	1.22±0.10	20	2.14±0.07

*Mean ± Standard Error.

TABLE 9

OCCURRENCE OF HEMOTHORAX AND PNEUMOTHORAX IN MICE

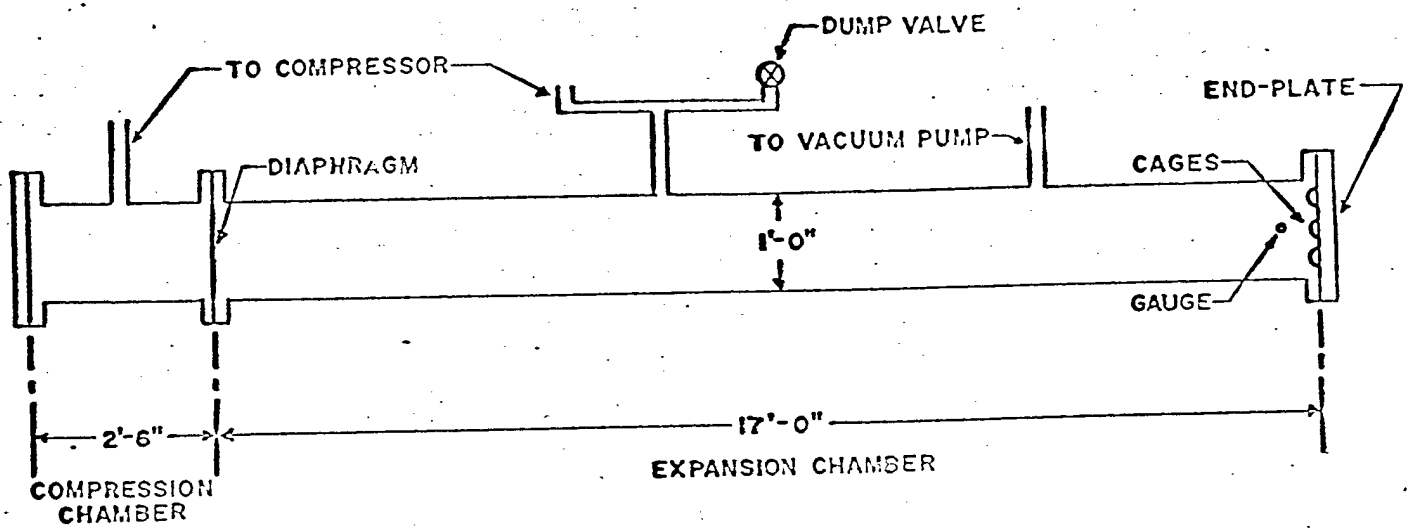
P_I (psia)	Total Number Animals	Number 1-hour Deaths	Series I Fatalities				Series II Fatalities			
			Hemothorax		Pneumothorax		Hemothorax		Pneumothorax	
			Number	Percent	Number	Percent	Number	Percent	Number	Percent
7	60	41	0	0	0	0				
7	45	20					0	0	0	0
12	45	23	1	4.3	1	4.3				
18	48	23	1	4.3	5	21.7				
18	69	24					2	8.3	3	12.5
24	60	31	6	19.4	11	35.5				
30	45	19					0	0	2	10.5
36	57	20					2	10.0	1	5.0
42	57	25	9	36.0	23	92.0				
42	69	30					5	16.7	10	33.3

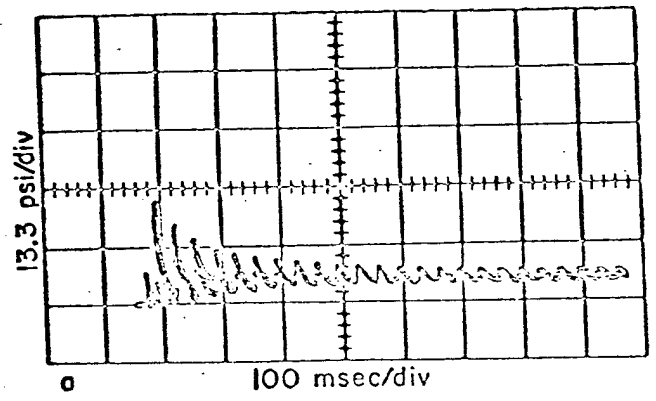
TABLE 10

COMPARISON OF LD_{50} OVERPRESSURES AND PRESSURE RATIOS FOR MICE EXPOSED TO AIR BLAST AT DIFFERENT EXPERIMENTAL AMBIENT PRESSURES (SERIES I)

Experimental Ambient Pressure, P_i (psia)	LD_{50}-1-Hour	
	Overpressure, ΔP (psig)	Pressure Ratio ($\Delta P/P_i$)
7	20.3	2.90
12	31.2	2.60
18	44.5	2.47
24	55.3	2.30
42	91.8	2.19
	Average	2.49

SHOCK TUBE LAYOUT





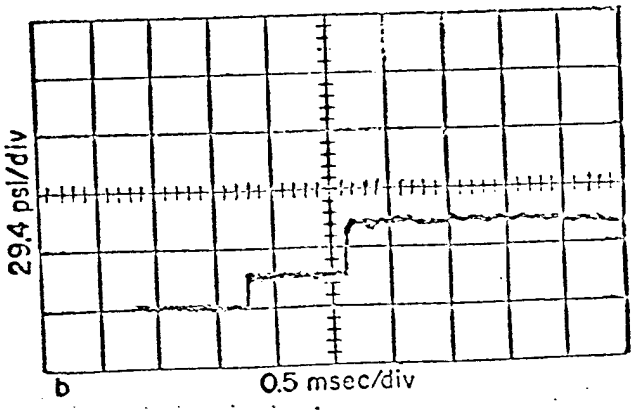


Figure 1

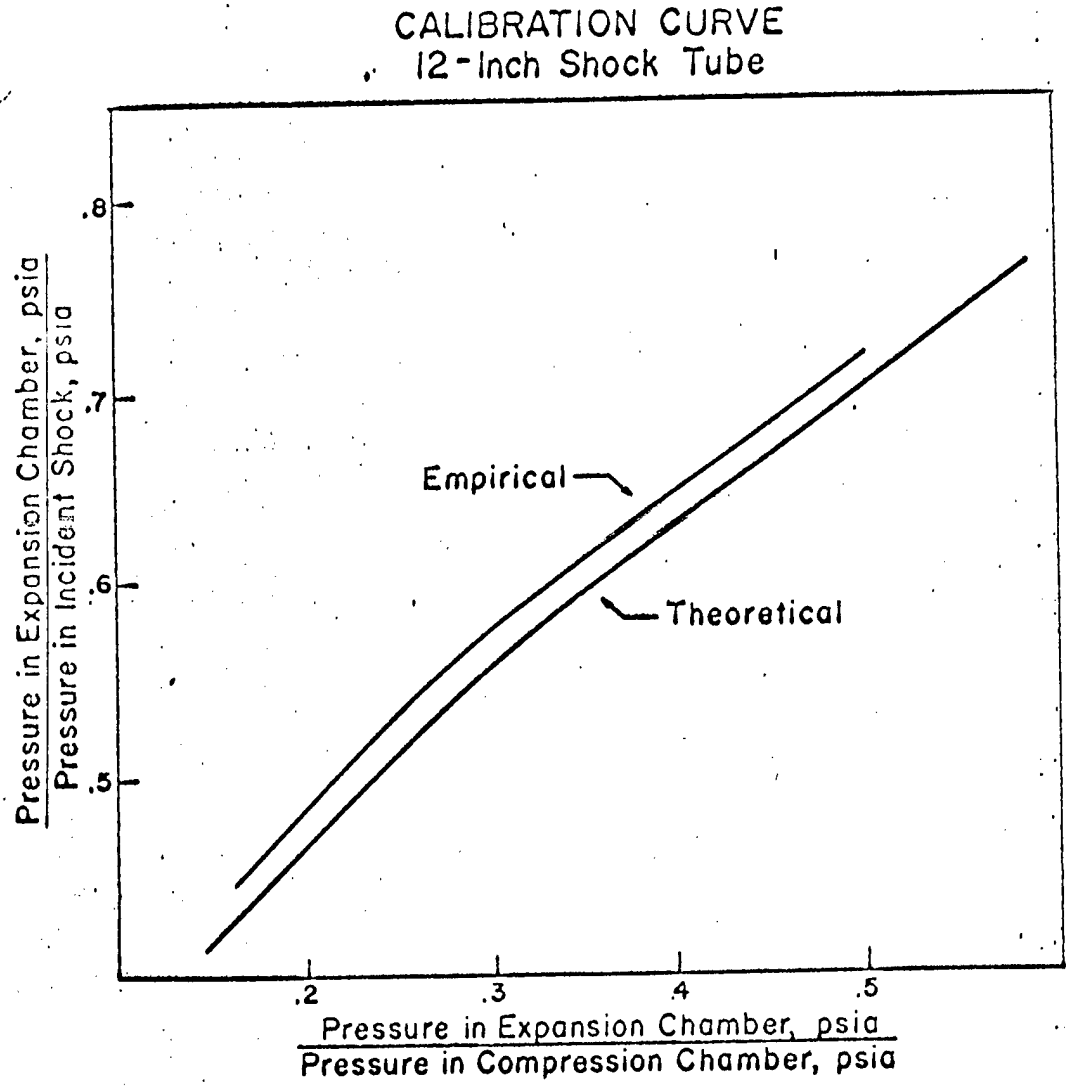


Figure 2. Comparison of the calibration curve for the 12-in. shock tube with the theoretical curve for shock strength as a function of the starting pressure ratio (Bleakney, Weimer, and Fletcher, 1949).

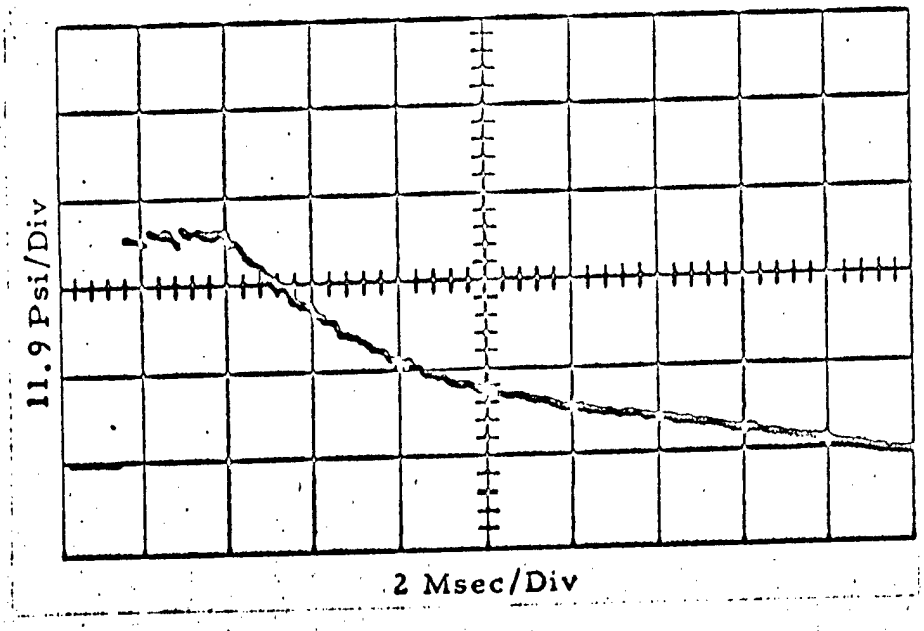


Figure. 3. Reflected shock wave recorded by a pressure transducer mounted on the end-plate of the shock tube.

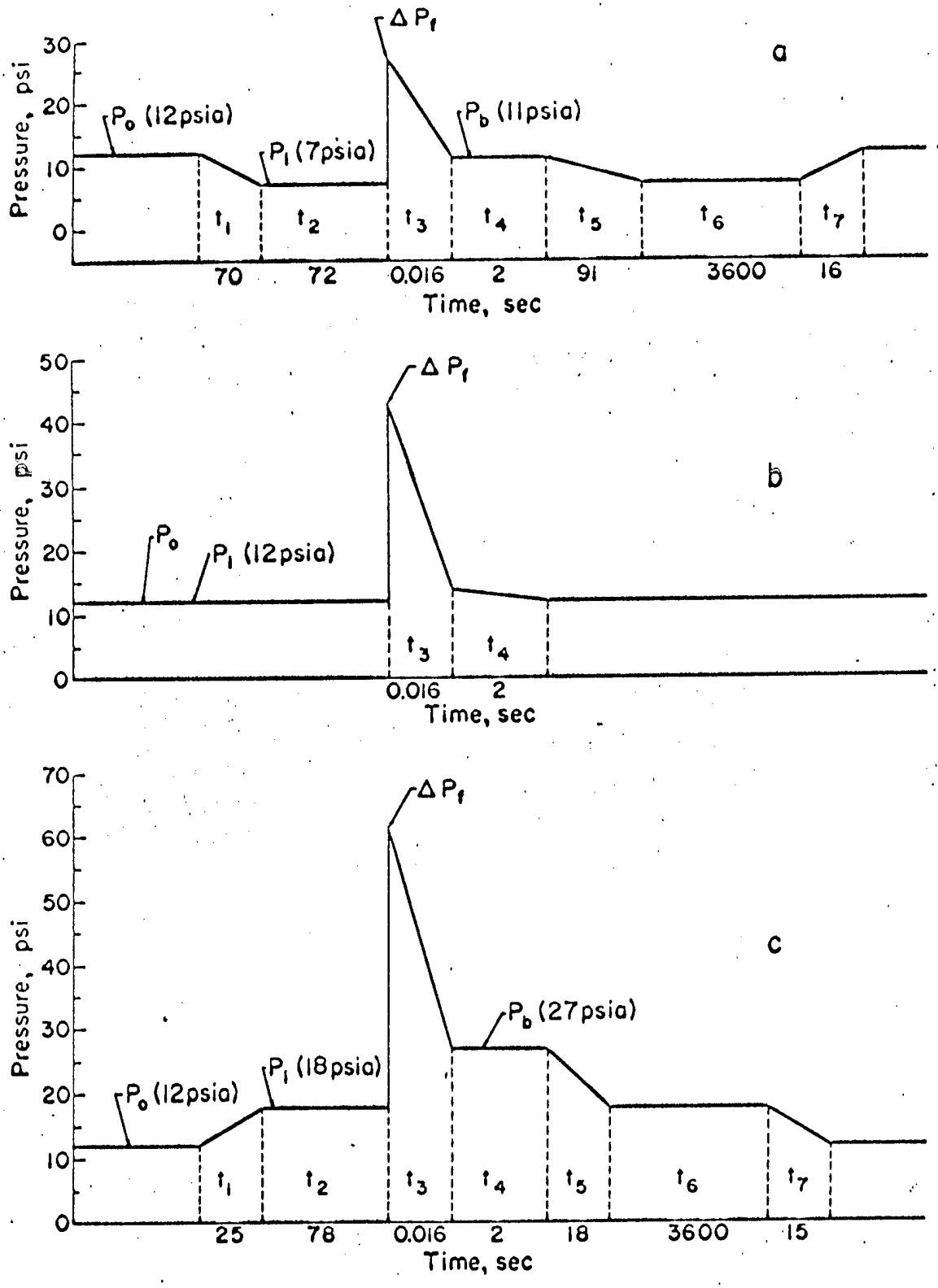
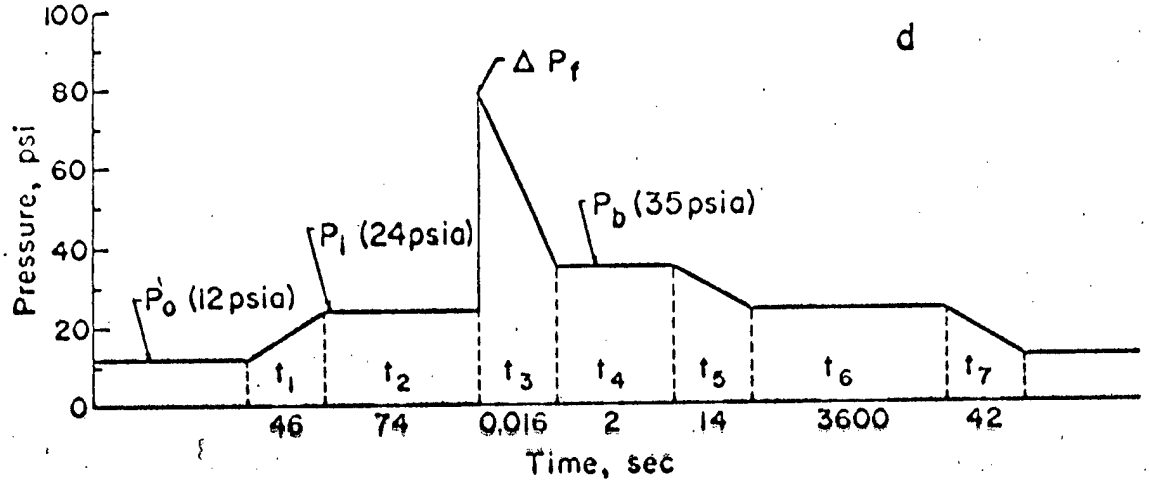


Figure 4 (a-c). Overall pressure-time profiles for Series I.



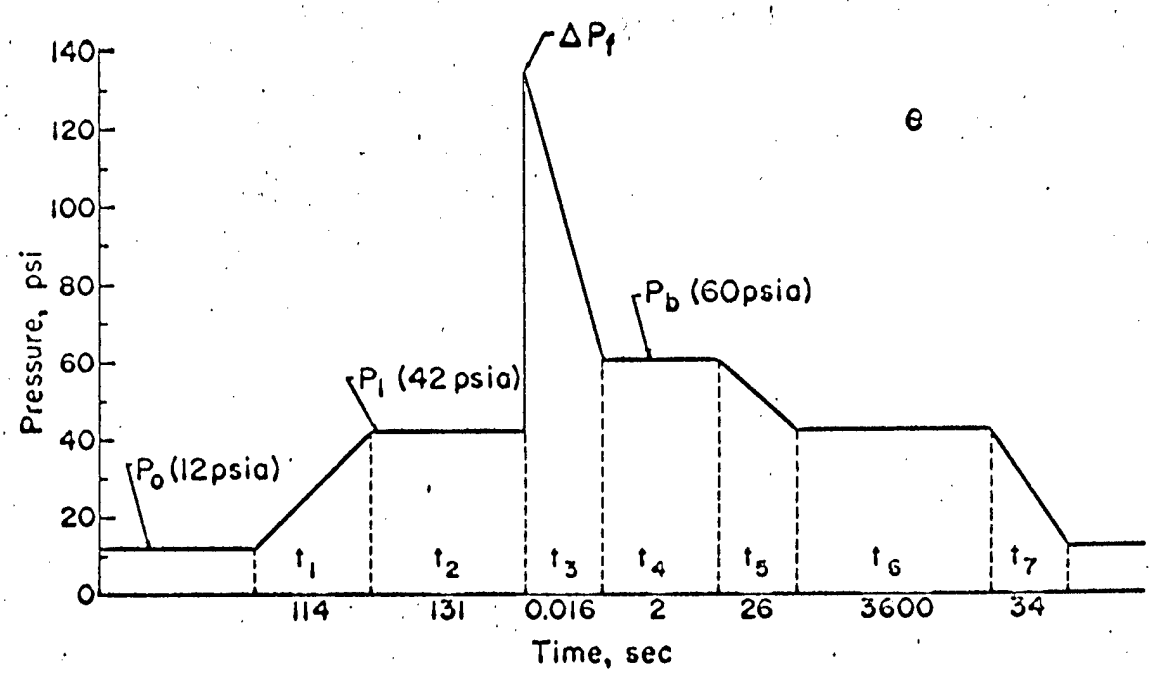


Figure 4 (d-e). Overall pressure-time profiles for Series I.

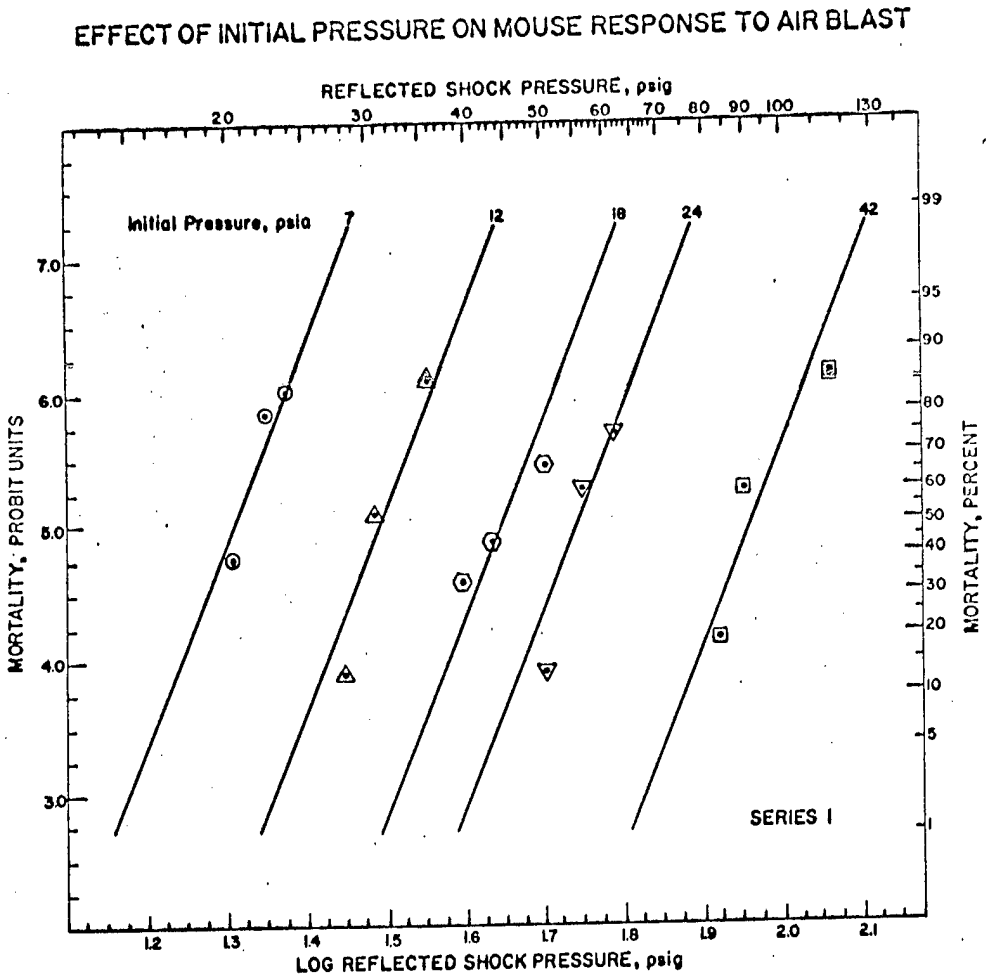


Figure 5. Probit regression lines relating the percent mortality in probit units to the log of the reflected shock pressures for mice subjected to air blast at different initial air pressures.

TOLERANCE OF MICE TO AIR BLAST AS RELATED TO THE INITIAL PRESSURE

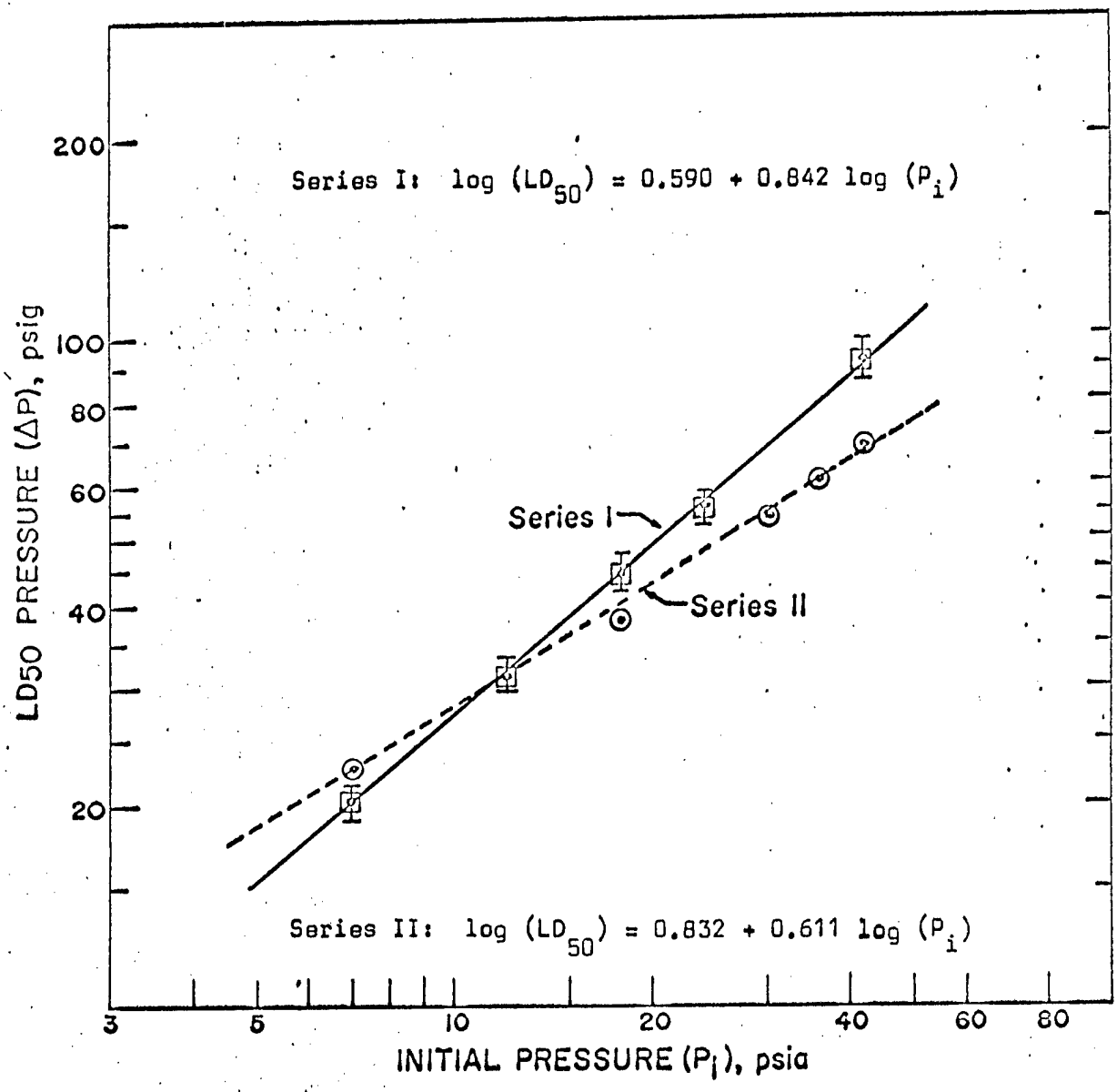


Figure 6

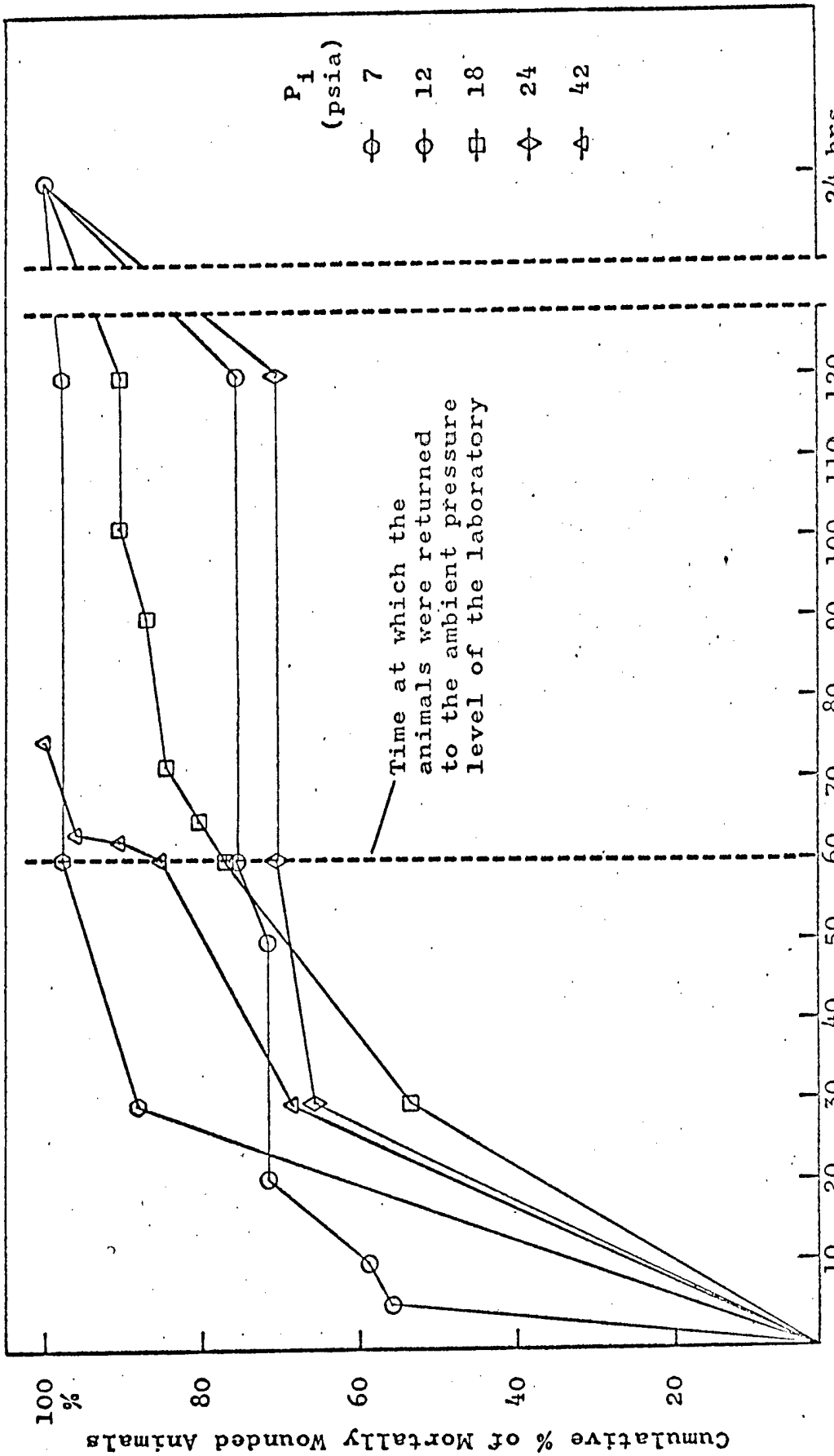
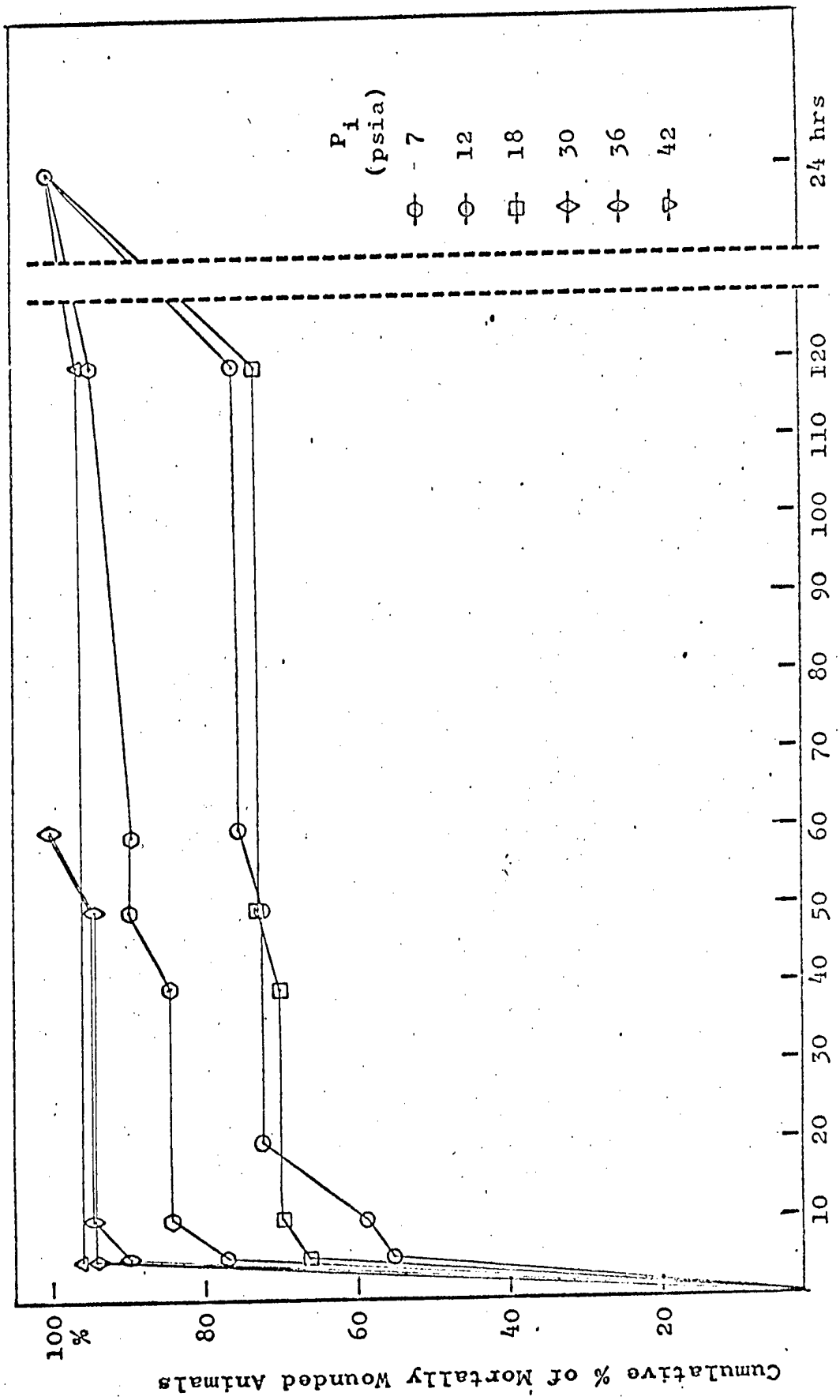

Mouse Mortality as Related to Time Following
Air Blast Exposure in Series I
Cumulative % of Mortally Wounded Animals
100 %
80
60
40
20
10
20
30
40
50
60
70
80
90
100
110
120
24 hrs
Time of Death, Minutes
Time at which the animals were returned to the ambient pressure level of the laboratory
P_i (psia)
7
12
18
24
42

Figure 7



Mouse Mortality as Related to Time Following Air Blast Exposure in Series II

Figure 8