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Primary Blast Injuries in the Open and in Foxholes Resulting from Nuclear Type Detonations

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The nature of primary blast injuries that affect the gas-containing organs in the body are described and illustrated in this report. A history of blast-injury research that includes descriptions of the proposed damage mechanisms, cause of death and clinical manifestations is also presented. Estimates of the incident blast levels required to produce lung and GI tract injuries in man are based on the results of tests with animals. There are three blast exposure scenarios: 1) for personnel prone, end-on to the blast in the open; 2) for personnel in open, two-man foxholes, with the blast wave rising in multiple steps; and 3) for personnel in the foxhole, with the blast wave rising in a single step.

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SUMMARY

The three main air-blast damage mechanisms are: 1) the direct or primary effect, caused by the blast wave impacting and compressing the body; 2) secondary effects, produced by flying debris striking the individual; and 3) tertiary effects, produced when a person is hurled by the blast winds into solid objects. The unique characteristic of direct effects is damage to internal organs only. This damage is usually combined with burns or other trauma, but this report deals only with direct effects of blast.

Blast injury was first described in the late seventeen hundreds in connection with victims of mine explosions. There were accounts of soldiers killed in battle without any external signs of injury (detonation deaths) during the Balkan War and World War I. The cause of death and the nature of the injuries were unknown, although there was much speculation, especially about blast affecting the nervous system.

It was not until research was conducted during WW II in the United Kingdom and Germany that the nature of blast injuries, the pathophysiology, cause of death, and the damage mechanisms in terms of the physical components of the blast wave were clearly explained. The German investigators discovered that the cause of death was air emboli in the coronary and cerebral arteries. They provided evidence to support the view that these air emboli enter the circulation from the disrupted alveoli in the lung. Another important discovery by the German researchers was the duration effect--as the duration of the blast waves increases, the peak overpressure required for mortality in dogs decreases from approximately 200 psi for durations of about 2 ms to near 80 psi for a duration of 12 ms.

Schardin, a German researcher, proposed three damage mechanisms: spallation, implosion and inertia. Spallation occurs when a shock front passes from a dense medium into a less dense medium and destroys the denser material at the interface; implosion occurs when gas bubbles in a fluid media are compressed by a shock wave into much smaller volumes with increased pressure which generate shock waves upon re-expansion. Inertia becomes a factor when a shock front strikes two objects of equal volume and shape but of different mass, and accelerates the less dense one more than the more dense one.

Study of blast victims of the wars on Israel and from terrorists' bombings has improved our understanding of direct blast injuries, which has led to progress in their detection, management and treatment. Previously, few in the medical field were familiar with blast injuries and their treatment.

With the advent of nuclear weapons there was a renewed interest in blast research. Laboratories in the United States, England, France, Germany, Sweden and China have conducted research to define the blast injuries and their treatment. Evaluating the effects of repeated muzzle blasts, developing protective clothing, and analyzing the risk from blast waves within enclosures were other areas of research common to these countries. Mathematical models were developed in most of these areas.

The nature of direct blast injuries is essentially hemorrhagic due to tissue disruption. The three organ systems most damaged are the lungs and air ways, the gastrointestinal tract (GI tract) and the auditory system. Damage originates in the air- or gas-containing portions of these systems; these may injure nearby solid organs, such as the heart, liver and spleen.

The short survival time of animal and human victims of blast is attributed to air emboli in the heart and brain. The latter accounts for a variety of central-nervous-system dysfunctions showing disorientation, hind-limb paralysis, convulsion, gasping respiration, etc.

Prognosis of patients who survive a few hours is good with respect to lung hemorrhage. Pulmonary hemorrhage improves in 24 hours and is resolved in a few weeks. Prognosis of victims who have received severe GI-tract injuries is poor. It is hard to diagnose and may result in peritonitis if the organs rupture or if they rupture secondarily later on. Hyperbaric treatment using an atmosphere of oxygen has been used to treat cases with air emboli.

Injury to the auditory system may result in hearing loss of two kinds. One is a conductive hearing loss resulting from ear drum rupture and/or damage to the ossicular chain. The other is neurosensory hearing loss associated with the blast energy entering the inner ear and destroying hair cells in the cochlea that convert vibrations to nerve signals. Mild forms of ear injury can produce a temporary hearing loss and are, for the most part, self-healing. Severe forms of injury result in a permanent hearing loss that in the case of conductive loss can only be corrected surgically.

The air blast dose for personnel in the open (prone and end-on) is the peak incident pressure as measured by a pressure transducer oriented side-on to the approaching wave. This is termed an "ideal" or "classical" blast wave form. It is characterized by a shock front with an immediate rise to peak pressure which then decays through the ambient level into a negative phase. The dynamic pressure is the difference between the pressure measured by a face-on transducer and a side-on transducer. The dynamic pressure is taken into account when determining the blast load for individuals oriented prone side-on to the blast or standing.

When the blast wave strikes a hard surface, it produces a reflected shock having a peak pressure from 2 to 8 times that of the incident one. A subject against or very close to the reflecting surface receives the reflected pressure as the blast dose.

Since the interest is in nuclear blast, it was assumed that all the blast overpressures were of long duration and only one value of incident blast overpressure was associated with a given injury level. The duration of the overpressure was considered in scaling the results of animal experiments to man.

The incident overpressure levels required to produce specified degrees of injury to the lungs and gastrointestinal tract in man were estimated from the results of tests using animals. The procedure of Bowen was used to scale the blast response of experimental animals to a 70-kg man at sea level and to a long-duration (200 ms) blast.

If an animal with a body mass, m, is exposed at an ambient pressure, P_0, to a classical blast wave with a positive phase duration, t_e, and a peak overpressure, P_e, and sustains a given injury level, then semi-empirical theory can be used to predict the peak overpressure, P_{s1}, that will inflict the same injury level in man exposed at sea level to a classical blast with a positive-phase duration, t_{s1}. The P_{sw} is a blast tolerance index derived from the dose-response curves from 12 species of mammals. The peak overpressure for man is estimated by:

$$P_{s1} = P_e \frac{(61.5)}{(P_{sw})} \frac{(14.7)}{(P_0)} \frac{1+6.76(t_{s1})^{-1.064}}{1+6.76[t_e(70/m)^{1/3}(P_0/14.7)^{1/2}]^{-1.064}} \qquad (1)$$

The five levels of lung injury selected were as follows: 1) Trivial--(threshold) petechial hemorrhages; 2) Slight -- ecchymotic areas or small light hemorrhage spots on the lung's surface, 3) Moderate -- small isolated areas of confluent hemorrhage involving < 30% of the lung's surface area; 4) Severe -- large areas of confluent hemorrhages extending deep into the parenchyma, involving 30 to 60% of the lung's surface area; 5) Very Severe -- entire lobes having confluent hemorrhages and covering > 60% of the lung's surface area.

Data on lung injuries from ten studies were placed into the above five injury categories, the corresponding overpressure values were scaled to man at sea level and averages were calculated for each of the levels. The mean peak overpressures associated with each of the injury levels are as follows: Trivial, 14.4 psi; Slight, 19.2 psi; Moderate, 29.0 psi; Severe, 34.3 psi and Very Severe, 42.0 psi. The highest pressure for no injury was 10.3 psi. Moderate lung hemorrhages increase the respiratory rate in dogs and sheep, and result in a detectable increase in lung weight due to the presence of blood and edema in that organ. Very severe pulmonary hemorrhage can be lethal.

The overpressure value represents the peak incident pressure in a long-duration wave and are applicable to man prone oriented end-on to the blast.

The same procedure was used to estimate the incident overpressures necessary to cause GI tract injuries in man. The five categories, with the corresponding overpressure values for man, are as follows: 1) Trivial, petechia or small spots of discoloration, 8.4 psi; 2) Slight, small areas of light, subserosal contusions, 14.0 psi; 3) Moderate, small areas of submucosal contusions with little or no disruption of the mucosal lining, 23.6 psi; 4) Severe, large areas of dark submucosal contusions with disruption of the mucosal lining and blood clots extending into the lumen, 29.4 psi; 5) Very Severe, disruption of the mucosal lining with hemorrhaging into lumen, or perforation of the entire wall and usually rupture of liver or spleen, 37.4 psi. The highest overpressure for no injury was 7.0 psi.

Incident overpressures that would cause lung hemorrhages in personnel located inside an open, two-man foxhole (2 x 6 x 4.5 ft) were based on five experiments on animals exposed to blast while inside a similar foxhole. The

incident overpressures required to produce the chosen degrees of lung hemorrhages were as follows: 1) none, 7.6 psi; 2) trivial, 11.0 psi; 3) slight, 14.6 psi; 4) moderate, 19.9 psi; 5) severe, 29.3 psi; and very severe, 34.4 psi.

It was not possible to estimate the overpressures for GI tract injury for man in foxholes directly because the injuries were not reported in sufficient detail in the experiment; instead, the incident overpressures that caused each of the lung injury levels in animals were reduced by 24.1% to obtain those for GI tract injuries. This was based on data (summarized in Table 9) that show overpressure values for GI tract injury for man in the open were, on the average, 24.1% lower than those required to produce the corresponding level of lung injuries. The higher tolerance of animals to reflected waves that rise in several steps (rather than in a single step) is clearly shown in the data from these experiments.

Another, more conservative set of estimates of the incident overpressures required to cause graded injury levels in foxholes assumed that the reflected overpressure in the foxholes rose to peak in a single step. The incident overpressure required for each injury level for man in the open was taken as the required peak reflected overpressure inside the foxhole for the same injury levels. The incident overpressures for the corresponding reflected pressures in the foxhole were taken from an empirically derived curve relating reflected pressure in a two-man foxhole to the incident pressure. The incident blast levels obtained by this method were on the order of 50% lower than those that assumed the wave in the foxhole rose in several steps. Values of incident overpressure required for lung injury, assuming a one-step rise, were as follows: none, 3.6 psi; trivial, 5.3 psi; slight, 7.5 psi; moderate, 12.0 psi; severe, 15.3 psi; and very severe, 18.5 psi.

Corresponding incident overpressures required for GI tract injuries were as follows: none, 2.3 psi; trivial, 2.9 psi; slight, 5.2 psi; moderate, 9.5 psi; severe, 12.2 psi; and very severe, 16.2 psi.

PREFACE

Technico Southwest Incorporated (TSI) is conducting a study of the effects of combined injuries on performance for the Defense Nuclear Agency under contract DNA 001-88-C-0207. In order to accomplish the goals of this study, a working group consisting of outstanding physicians and scientists who specialize in the effects of radiation, thermal and blast injuries has been selected. This group has met over a period of two years and has heard presentations by scientists who are in the forefront of the research in these areas.

The area that presents the most complex problems is that of the effects of blast injuries on performance. This is because there are primary or direct effects of blast on the lungs and gastrointestinal tracts; secondary effects from missiles accelerated by the blast; and tertiary or tumbling and deceleration effects when persons are thrown by the wind produced by a nuclear detonation. Each of these effects results from a different mechanism and occurs at different overpressures.

Donald Richmond and Edward Damon worked for the Lovelace Medical Research Foundation in Albuquerque, NM which has done more research into the effects of blast on mammalian systems than any other group of scientists. These studies form the basis for many of the blast risk criteria in the Personnel Risk and Casualty Criteria developed by the United States Army Nuclear and Chemical Agency. TSI is fortunate in having been able to obtain the services of these two outstanding scientists. In response to the needs of the combined injury working group, TSI has requested that Drs Richmond and Damon assemble and interpret data on the effects of blast for the range of overpressure values defined by that working group. This report is the result of their intensive efforts to fulfill the needs of the working group.

CONVERSION TABLE

Conversion factors for U.S. Customary to metric (SI) units of measurement.

MULTIPLY → BY → TO GET
TO GET ← BY ← DIVIDE

angstrom	1.000 000 X E -10	meters (m)
atmosphere (normal)	1.013 25 X E +2	kilo pascal (kPa)
bar	1.000 000 X E +2	kilo pascal (kPa)
barn	1.000 000 X E -28	meter^2 (m^2)
British thermal unit (thermochemical)	1.054 350 X E +3	joule (J)
calorie (thermochemical)	4.184 000	joule (J)
cal (thermochemical)/cm^2	4.184 000 X E -2	mega joule/m^2 (MJ/m^2)
curie	3.700 000 X E +1	giga becquerel (GBq)*
degree (angle)	1.745 329 X E -2	radian (rad)
degree Fahrenheit	T_K=(t°f+459.67)/1.8	degree kelvin (K)
electron volt	1.602 19 X E -19	joule (J)
erg	1.000 000 X E -7	joule (J)
erg/second	1.000 000 X E -7	watt (W)
foot	3.048 000 X E -1	meter (m)
foot-pound-force	1.355 818	joule (J)
gallon (U.S. liquid)	3.785 412 X E -3	meter^3 (m^3)
inch	2.540 000 X E -2	meter (m)
jerk	1.000 000 X E +9	joule (J)
joule/kilogram (J/kg) (radiation dose absorbed)	1.000 000	Gray (Gy)**
kilotons	4.183	terajoules
kip (1000 lbf)	4.448 222 X E +3	newton (N)
kip/inch^2 (ksi)	6.894 757 X E +3	kilo pascal (kPa)
ktap	1.000 000 X E +2	newton-second/m^2 (N-s/m^2)
micron	1.000 000 X E -6	meter (m)
mil	2.540 000 X E -5	meter (m)
mile (international)	1.609 344 X E +3	meter (m)
ounce	2.834 952 X E -2	kilogram (kg)
pound-force (lbf avoirdupois)	4.448 222	newton (N)
pound-force inch	1.129 848 X E -1	newton-meter (N·m)
pound-force/inch	1.751 268 X E +2	newton/meter (N/m)
pound-force/foot^2	4.788 026 X E -2	kilo pascal (kPa)
pound-force/inch^2 (psi)	6.894 757	kilo pascal (kPa)
pound-mass (lbm avoirdupois)	4.535 924 X E -1	kilogram (kg)
pound-mass-foot^2 (moment of inertia)	4.214 011 X E -2	kilogram-meter^2 (kg·m^2)
pound-mass/foot^3	1.601 846 X E +1	kilogram/meter^3 (kg/m^3)
rad (radiation dose absorbed)	1.000 000 X E -2	Gray (Gy)**
roentgen	2.579 760 X E -4	coulomb/kilogram (C/kg)
shake	1.000 000 X E -8	second (s)
slug	1.459 390 X E +1	kilogram (kg)
torr (mm Hg, 0°C)	1.333 22 X E -1	kilo pascal (kPa)

* The becquerel (Bq) is the SI unit of radioactivity; 1 Bq = 1 event/s.
**The Gray (Gy) is the SI unit of absorbed radiation.

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SECTION I
INTRODUCTION

According to Zuckerman there are three main damage mechanisms from air blast (Ref. 30). 'Primary effects" are those caused by the blast wave striking and compressing the body. This results in a blast injury syndrome that includes damage to the air- or gas-containing organs; namely, the ear, lungs and the upper respiratory tract, and the gastrointestinal tract. The injury at high blast levels may also include damage to solid organs such as heart, liver and spleen and ribs. In the past many medical and paramedical personnel did not recognize nor know how to treat primary blast effects. Work with blast victims in the Middle East wars and terrorist bombings has informed physicians in recognition and treatment of primary blast injuries.

"Secondary" blast effects result when the body is injured by flying debris, building collapse, tree blowdown and crater ejecta. Window glass fragments produce a particularly common secondary effect and is usually the most far-reaching. Injuries range from abrasions and contusions to skeletal fractures and massive crushing injuries. A high incidence of secondary effects may be expected when blasts occur in built-up areas.

"Tertiary" effects result when the body is thrown by the blast wave (or blast winds) against the ground, equipment items, structures, tree, etc. These injuries are very similar to those from free falls and secondary effects. Medical personnel are familiar with the management and treatment of these forms of mechanical trauma, which are common in modern society.

Primary effects are commonly referred to as "pure" or "direct" blast injury effects, while secondary and tertiary are called "indirect" effects.

"Solid blast" is a term given to the mechanism in which the air shock or ground shock accelerates a harder structure or portion of it against the body. Deck heave or deck slap induced by underwater explosions is an example of this damage mechanism.

1.1 HISTORICAL BACKGROUND.

According to Clemedson (Ref. 5), original interest in blast injury centered on mine explosions. Jars, in 1788 was the first to describe a phenomenon that now would be called "blast injury". Jars' ideas of conditions were essentially correct and in agreement with present-day understanding of blast injury.

The Swiss, in 1914, were the first to systematically study blast injury in experimental animals. Their interest arose from examination of three soldiers killed during the Balkan War by a bursting grenade (shell) without external injuries, and accounts of soldiers and sailors injured by shells passing close by them. The bow-shock produced by projectiles travelling supersonically could have accounted for these blast injuries. In the Swiss experiments, rabbits were exposed to 110-g dynamite blasts inside a sand pit having a stone cover. The distance from the charge and the number of blasts were variables. The gross blast injuries described were those known today, but the Swiss could not account for the sudden deaths (Ref. 24).

No blast deaths were reported in the World War I military medical literature. It was known that blast would blow a man to pieces or throw him away and cause ear injury, but there were no accounts of the internal injuries caused by primary blast. During the 1918 to 1919 period, blast injury studies were conducted at Sandy Hook Proving Grounds, New Jersey (Ref. 11). Dogs, cats, rabbits, and frogs were exposed to the muzzle blasts from 10-inch naval rifles and 12-inch mortars. The blasts from the 10-inch rifles, on the order of 280-psi peak pressure, repeatedly produced shock in dogs, while the higher blast pressures of 388 psi from the 12-inch mortar did not. They defined shock as a halving of the normal blood pressure. It was suggested that the critical factor was the duration of the positive pressure phase, longer for the rifle than for the mortar. Researchers undertook rather comprehensive physiological measurements, using both gross and microscopic observations, but did not find any evidence of lesions in the brain or nervous tissue. Until that time, there was much speculation in the literature that the blast primarily affected the nervous system. Much of this emphasis may have been due to the large number of "shell shock" casualties of WW I who evidenced a variety of psychophysiologic symptoms after prolonged exposure to heavy artillery barrages.

There were accounts of detonation deaths (soldiers found dead with few, if any, external injuries) during the Spanish Civil War (1936 until 1939), but it was not until WW II, with an increase in aerial bombing attacks on civilian population centers in Germany and England, that detonation deaths again attracted attention. In the United Kingdom (UK) interspecies studies were conducted that related the blast overpressure levels required for 50% mortality (P_{50}) to the body weight (Ref. 9). Mice, rabbits, guinea pigs, goats and monkeys were exposed to blasts from 1-, 8- and 66-lb charges (Fig. 1). An extrapolation of this P_{50} versus body weight curve predicted a P_{50} of 370 psi for a 60-kg man and one of 470 psi for an 80-kg man. These P_{50} values for man would only apply to short-duration overpressure because they are based on the results from small explosive charges. To evaluate the importance of the blast wave duration, mice were exposed to blasts from 1-, 8-, and 66-lb charges but the same P_{50} was obtained in each case. Much later it was learned that all three durations were relatively long for an animal as small as the mouse and that explosive charges of less than 1.0 lb would be required to demonstrate a higher P_{50}. The nature of the blast injuries and the pathophysiological effects were carefully described by the UK group. Their observations have withstood the test of time. The other highlights of their work were their demonstration that the blast wave must impact the thorax directly to produce lung hemorrhage and that sponge rubber may shield the body from some direct blast effects.

The results of very extensive studies undertaken in Germany did not become available until after the end of WWII. Curiously, German civil law prohibited autopsies on most bombing victims, which delayed a pathologic description of primary blast injury. However, military medical studies of blast waves were undertaken throughout the war.

Benzinger, Desaga, Rossle and Schardin summarized the results of their very extensive research conducted during WW II (Ref. 7, 8, 23 and 25). This was published in 1950 and reprinted in 1971 by the U.S. Air Force in a book entitled "German Aviation Medicine, World War II", Vol. II. Their appreciation of blast injury is best exemplified by a quotation from Benzinger: "The blast wave is a shot without a bullet, a slash without a sword. It is present

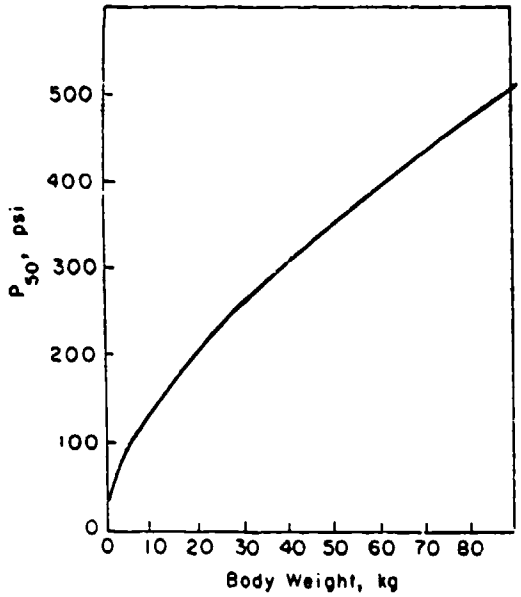

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Figure 1. Relation between P_{50} (overpressure for 50% mortality) and body weight.

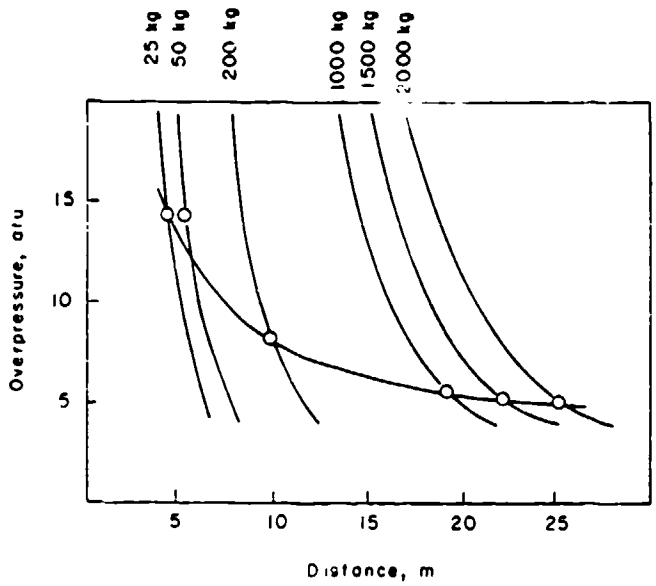

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Figure 2. Fatal blast overpressures for dogs as a function of distance and charge weight (overpressure-duration effect).

everywhere within its range. Blast would be as dreaded a weapon as chemical if its effects were not limited to small areas. However, it would be premature to believe that this situation will always remain the same."

The pathoanatomical and pathophysiological changes due to air and underwater blast were thoroughly described by the German investigators. They pointed out that the nature of the internal injuries produced in the two media were the same. The Germans were the first to discover that arterial air embolism was the cause of immediate death from blast injury. They reasoned that air entered the pulmonary venous circulation from the disrupted alveoli and was then distributed to the coronary vessels, the brain, and vascular beds in other organs of the body. Injecting very small volumes of air into the carotid artery produced the central nervous systems signs that had previously been observed in blast-injured dogs (and man). Electrocardiographic changes observed in blasted animals were reproduced by injecting about 1 cc of air into the pulmonary veins of dogs. These findings corroborate autopsy findings of internal air emboli as the most probable mechanism of early death from primary blast injury.

The duration effect was an additional significant finding of the German studies (Fig. 2). They found that the fatal static or side-on overpressure for dogs decreased by a factor of three when the duration of the positive phase was increased from about 1.8 to 12 ms. As illustrated in Fig. 2, the fatal peak overpressure decreases for larger charge weights. The larger charge weights result in longer overpressure durations at the required peak pressure.

These researchers also demonstrated that the blast wave must strike the thorax and not enter through the upper respiratory tract to inflict lung injury. In contrast to the British findings, Desaga found that no protection from air blast was afforded by placing foam rubber materials about the thorax. In fact, he showed that lung hemorrhage was intensified by this material covering. This conclusion agrees with more recent findings.

Schardin took an engineering approach to the medical observations and proposed three damage mechanisms for blast injury. The first was <u>spallation</u>. As a shock front passes from a more dense medium to a less dense medium, it reflects back into the denser material as a tension wave that throws off (spalls) materials at the interface. Schardin demonstrated this by detonating

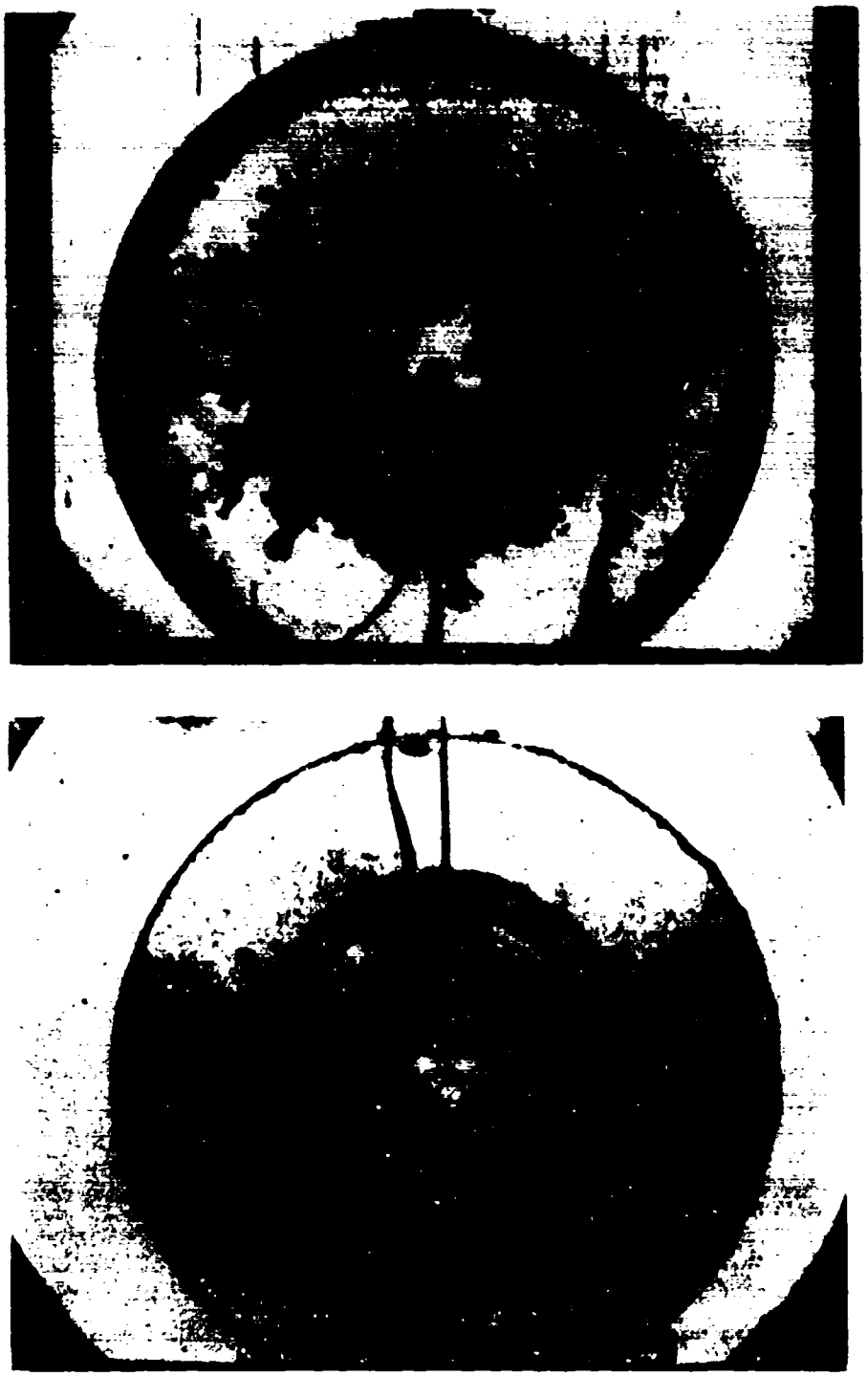


Photo from Ref. 25

Figure 3. Spalling effect, pictures of a small lead azide pill detonated in center of glass plate.

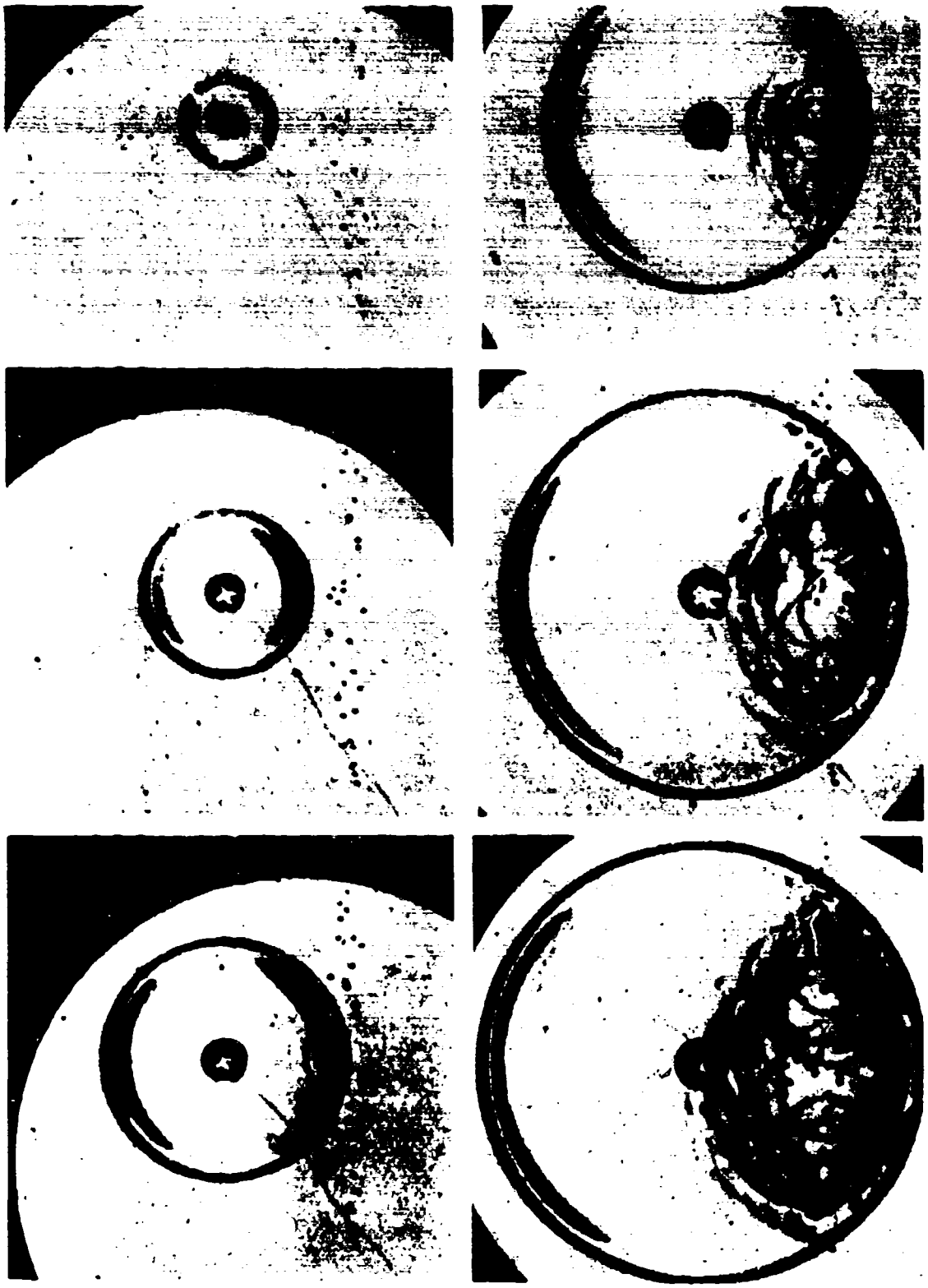


Photo from Ref. 25

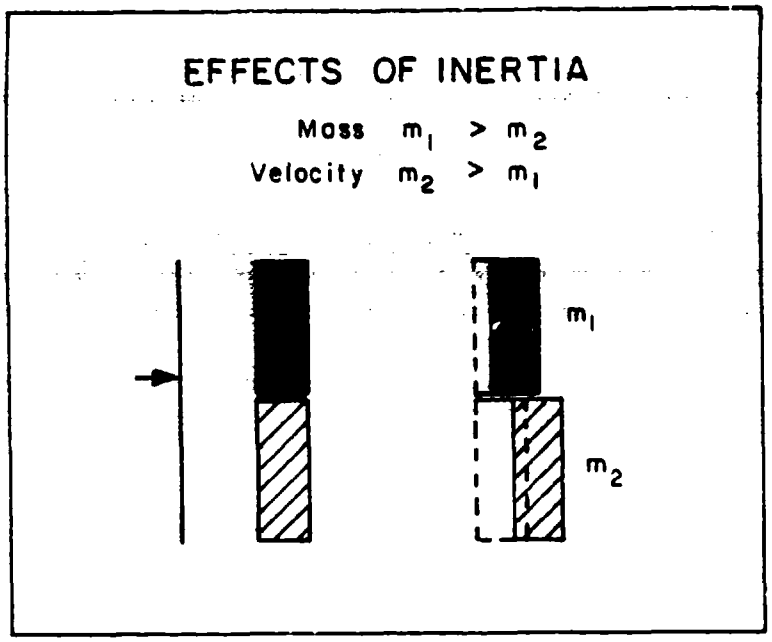
Figure 4. Implosion of air bubbles by an underwater shock wave.

a small lead azide charge in the center of a glass disc. Before the breakage cracks reach the outer edges of the glass disc, the shock front has already reflected from the periphery and spalled material from it (Fig. 3). This is also seen as the breaking up of the water surface when a shock wave from an underwater explosion, such as a depth charge, reaches the surface, i.e., the water-air boundary.

The secondary damage mechanism described by Schardin was an implosion phenomenon illustrated when a shock wave propagates through a stream of air bubbles passing through water and the bubbles are compressed by the shock wave (implosion effect). As the strongly compressed bubbles expand, they become the centers of new pressure waves (Fig. 4). Schardin suggested that a similar phenomenon might occur in the lung tissues of animals struck by a strong shockwave or in the gas-containing portions of the gastrointestinal tract.

The third damage mechanism for blast injury proposed by Schardin was inertial effects. He pointed out that a leaf exposed to an intense air blast had its woody veins remaining, but the delicate portions between the veins were blown away. The veins have more inertia than the light tissue between them and the two materials could be expected to accelerate at different rates when struck by the blast, thereby creating great stress at the boundaries. The delicate alveolar tissue between bronchi and blood vessels would be an analogous condition. In support of this, pathologic studies showed hemorrhage around the bronchovascular structures in the lung. Figure 5 illustrates inertial effects. When two objects of equal size and shape but of different mass are struck by a blast wave, the one having less mass (m_2) accelerates to a higher velocity and travels farther then the one (m_1) of greater mass.

In keeping with a mechanistic interpretation, Schardin also suggested that, if the duration of the blast wave is less than the natural period of the system (the frequency at which it resonates), then the impulse (pressure-time integral) becomes the determining damage component of the blast wave. If, on the other hand, the duration of the blast wave is greater than the natural period, it is the peak pressure that will account for the damage observed.



Data from Ref. 2

Figure 5. Effects of inertia, when two objects of equal size and shape but of differing mass are struck by a blast wave.

During the 1942-1945 period, the U.S. Navy studied air blast and immersion blast effects using rabbits, guinea pigs, monkeys, and goats (Ref. 7). They concluded that blast injury only occurred when animals were within the flash zone and that air blast was not an important casualty producer. They also stated that this may not be true for people exposed to blast inside confined spaces. They considered underwater blast to be an important casualty producer. At that time, there were many accounts of death and injury among sailors in the water during explosions of depth charges and torpedoes.

1.2 POST-WAR BLAST RESEARCH.

Recognition of the enormous range of blast effects from nuclear weapons caused a heightened interest in blast research worldwide. Two questions that arose early were: 1) Did the long-duration flow or blast winds associated with the nuclear blast wave present a displacement hazard to equipment and personnel? 2) Did the pressure-duration curve of Desaga (Fig. 2) continue significantly downward, thereby increasing the lethal zone from these large yield, very long-duration exposures?

After WW II, the blast research of the German scientists, under the direction of Schardin, was continued at the French-German Research Institute at Saint-Louis (ISL), France (Ref. 10). In the United Kingdom, blast research was conducted at the Chemical Defense Research Establishment at Porton, Salisbury, Wiltshire (Ref. 6). Since 1964, with the detonation of their first nuclear weapon, there has been uninterrupted blast research by ZG Wang at the Research Institute of Surgery, Chongquing, People's Republic China (Ref. 29). Clemedson's group pioneered research in blast injury at the Swedish Defense Research Establishment. Their work has played a major part in the understanding of the mechanisms and consequences of primary blast injury (Ref. 3, 4). In 1953, the Atomic Energy Commission contracted the Lovelace Foundation, Albuquerque, New Mexico, to study the biological effects of nuclear blast under the direction of Clayton S. White.

One of the major contributions of the Lovelace researchers is the extention of the mortality curves of Desaga (Ref. 2) to overpressure durations of >1000 ms. The incident overpressures associated with fifty percent mortality for man in relation to the duration of the overpressure is given in Figure 6. These curves are based on the dose-response curves from 13 animal species subjected to blast waves of various duration generated by high explosives in the open and blast produced inside shock tubes. In the curve applicable to man end-on to the blast, the incident shock or the side-on pressure constitutes their air blast dose. In the curve for personnel broadside to the approaching blast wave, the blast dose is the sum of the dynamic pressure and the incident pressure. The third curve is for individuals against or very close to a reflecting surface where the reflected pressure is the effective dose.

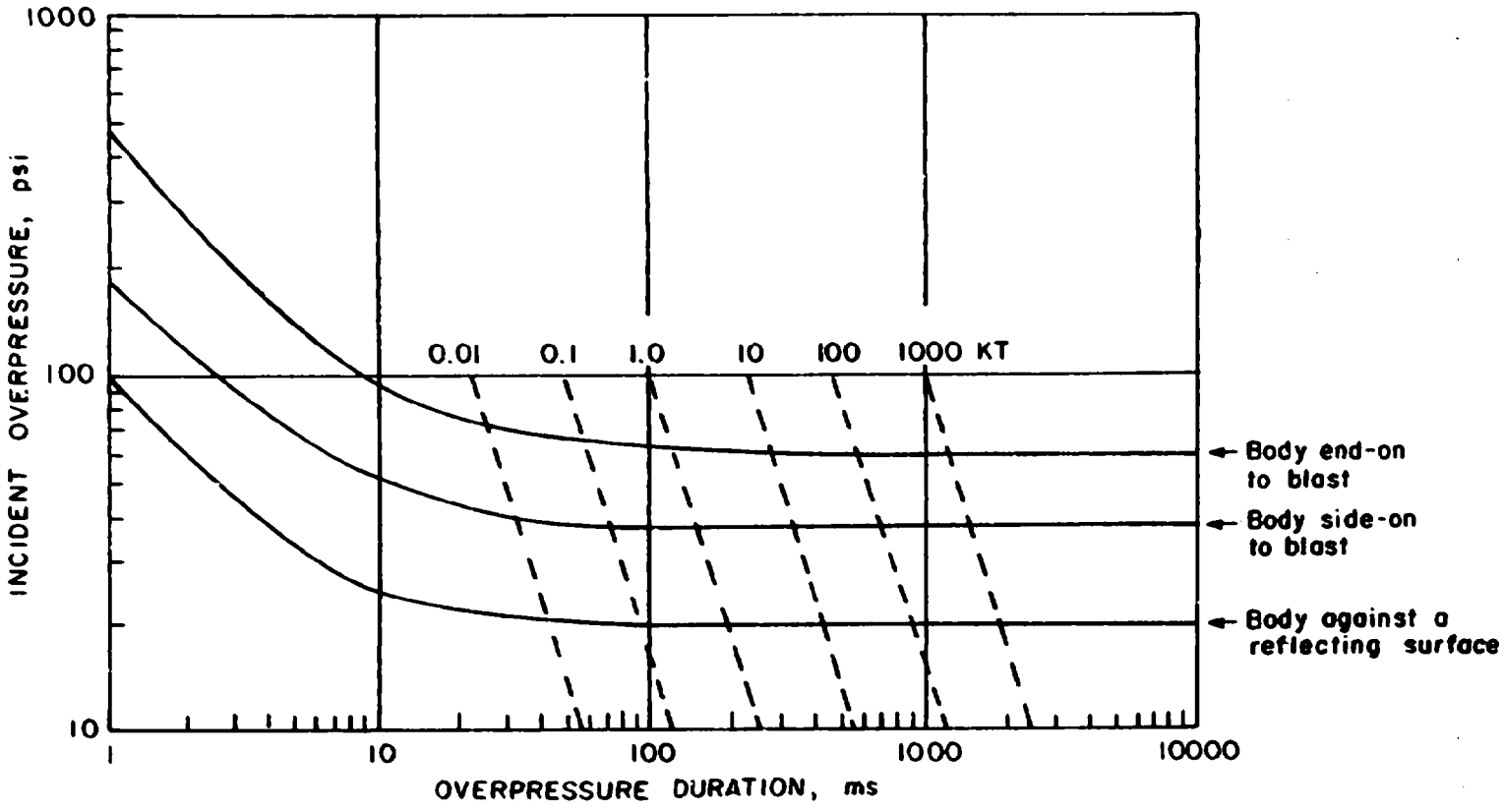


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Figure 6. P_{50} incident overpressure for man as a function of positive overpressure and body orientation.

The blast problems addressed in each of these countries were quite similar. Some of the areas of research were:

* describing the nature of blast injury for its prognosis and treatment.

* developing mathematical models to describe the response of the body under blast loading.

* evaluating the effects of exposure to repeated blasts in connection with the risk from muzzle and breech blasts.

* defining the damage parameters of complex blast waves that are a result of firing from enclosures, blast behind armor, terrorist bombing, etc.

* developing protective garments for personnel.

* evaluating the effects of enhanced blast munitions, such as fuel-air-explosive.

The nature of primary blast injuries and associated external symptoms and prognosis are described in the following section.

SECTION 2
NATURE OF PRIMARY BLAST INJURIES

Primary blast injury is almost always accompanied by some mechanical wounds and burns, especially when the source is small yield conventional ordnance, although there have been instances where soldiers died in battle without any external signs except bloody froth about their nose and mouth. Uncomplicated blast injury can be expected from 1) fuel-air explosive munitions and tactical nuclear weapons 2) in small field fortifications, ship compartments, and armored vehicles hit by various penetrators, and 3) explosions in a water environment.

2.1 EXTERNAL SIGNS OF INJURY.

The external clinical signs of blast injury are respiratory distress in the form of rapid shallow breathing or slow labored breathing with difficulty in exhalation. There may be blood or bloody froth exuding from the nose and mouth, tightness and pain in the chest, and cyanosis. Hemorrhaging from the auditory canal may be seen. Gastrointestinal injury may be evidenced by involuntary guarding, tenderness to touch, and a rigid abdomen as well as bleeding from the anus or a bloody stool. The victim may also show evidence of having arterial air embolism; evidence of cerebral dysfunction, such as confusion and disorientation; colonic movement; and limb paralysis. Some of the behavioral effects commonly reported are:

* All will be dazed and confused immediately following blast exposure.
* The neurological state will vary from normal to comatose.
* Neurological sequelae may be loss of equilibrium, convulsions and focal neurological disturbances.
* Blast exposure usually results in an inability to concentrate.

2.2 DIAGNOSIS.

The first symptom looked for in diagnosing blast injury should be evidence of air emboli since emboli are an immediate threat to life. Air emboli may be seen in the retinal vessels with an ophthalmoscope or evidenced as wedge-shaped blanched areas on the underside of the tongue. Coronary air emboli can be evidenced by electrocardiogram changes that are similar to ischemic ones.

A chest x-ray is an important diagnostic tool in determining the extent of pulmonary contusions. The presence of a pneumothorax, hemothorax, subpleural air blebs, and pneumomediastinum can be seen in the x-ray. Chest sounds are equally important and may vary from a few localized rales to widespread rales and rhonchi. A blood gas analysis to give the partial pressures of oxygen and carbon dioxide will aid in evaluating the physiological extent of the lung damage. The respiratory rate and blood pressure are other indicators of pulmonary damage. Hypotension is a symptom of extensive lung hemorrhage as is bradycardia (increased heart rate).

Gastrointestinal tract injuries may be more extensive than indicated by the clinical signs. Abdominal x-rays may show gastric dilation and dilated loops of the intestine, but small fissures or perforations usually will not be seen in the films. The absence of bowel sounds may indicate visceral rupture. Aspiration of abdominal fluids and blood cell counts are other tests to assess visceral perforation. Inspection with a sigmoidoscope may be useful for identifying colonic damage.

Eardrum rupture may be seen with an otoscope. The severity of eardrum perforation, unilateral or bilateral, may be a general indication of the intensity of the blast and extent of other injury.

2.3 CHARACTERISTICS OF PRIMARY BLAST INJURIES.

Primary blast injuries involve hollow or gas-containing organs. It is generally agreed that damage to solid organs occurs because they are in direct contact with the imploding hollow ones. Hollow organs occur in three systems in the body: the respiratory system, the gastrointestinal system, and the auditory system.

2.3.1 Intrathoracic Injuries.

Those organs in the chest damaged by blast are the lungs and airways, the heart, tissues of the chest wall itself, including the ribs and intercostal muscles.

2.3.1.1 Lung. Damage to the lungs is hemorrhagic in nature. Among the blast injuries, pulmonary hemorrhage is the most impressive. The lungs are

considered to be the target organ since its disruption allows air emboli to enter the circulation and cause rapid death. The severity of lung hemorrhages ranges from a few petechiae (pin-head sized hemorrhages) and ecchymotic areas (concentration of petechiae) on the surface of the lungs, to confluent hemorrhages ranging from small isolated areas to those involving entire lobes. Confluent hemorrhages may extend deep into the parenchyma and surround the bronchial tree. A darker area, almost black in color, is usually present and appears to represent the impression made in the lung by the ribs. Actually, good experimental evidence shows that these so-called rib markings are produced by the intercostal tissues impacting the lung and not the ribs.

Investigators have used the lung weights of animals as an index of the amount of blood and edema in the damaged lung. The lung weight of animals exposed to blasts in the lethal range may be two to three times its normal value. Examples of lungs having slight, moderate, severe and very severe degrees of hemorrhage are shown in Figures 7, 8, 9, and 10 (these and other photos are from the authors' research at the Lovelace Medical Research Foundation).

Intense blasts commonly cause subpleural air blebs and may even rupture the lung itself. Lung lobes may be punctured by the ends of fractured ribs. Both of these events account for air and blood in the thoracic cavity (pneumo and hemothorax). Contusions along the intercostal muscles are common blast lesions in the chest wall. In severe cases, the tissues may tear, and the ribs, fracture. The usual site of rib fracture is at the midaxillary region and at the vertebral junction.

2.3.1.2 Upper Airways. The mucosal lining of the sinus, larynx, pharynx and trachea may become bruised as a result of rather low blast levels. Severe forms of this injury involve hemorrhaging beneath the mucosa that results in a hematoma and an elevation of the mucosa that reduces the cross sectional area of the lumen, Figure 11.

Figure 7. Dorsal and ventral views of lungs from surviving sheep with slight lung hemorrhage. Lung = 0.84% of the body weight.

Figure 8. Dorsal and ventral views of lungs from surviving sheep with moderate lung hemorrhage. Lung = 1.28% of the body weight.

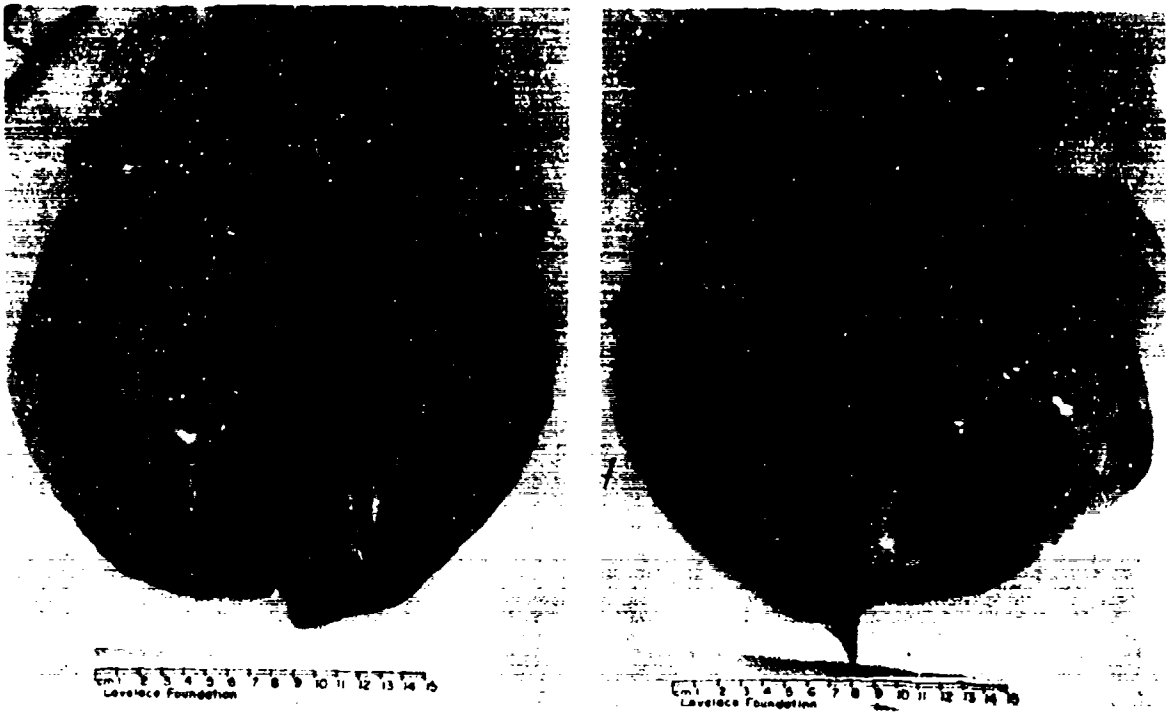
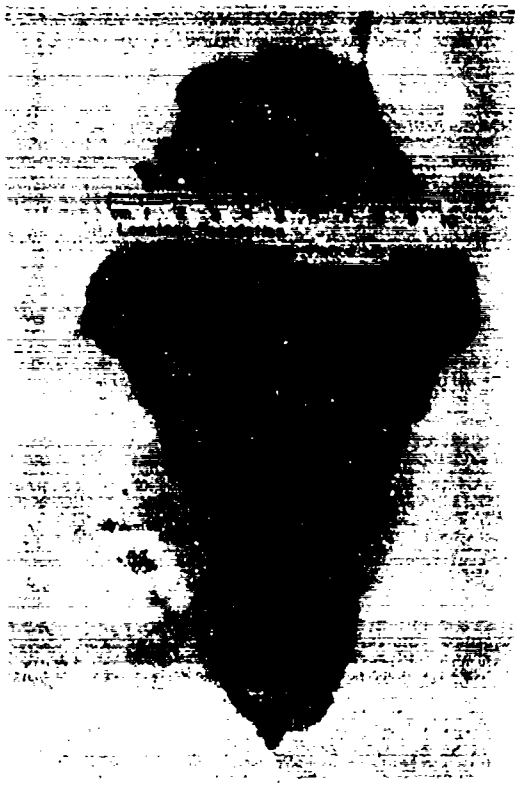


Figure 9. Dorsal and ventral views of lungs from a surviving sheep with severe lung hemorrhage. Lung = 1.94% of body weight.

Figure 10. Dorsal and ventral views of lungs from a sheep (two minute death) with very severe lung hemorrhage. Lung = 2.55% of body weight.



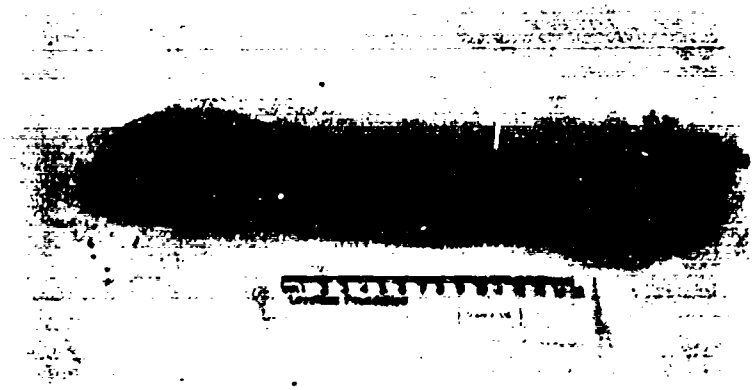


Figure 11. Upper photo: sinus hemorrhages of sheep with a 12-minute death. Lower photo: extensive contusions in the trachea and larynx of surviving sheep.

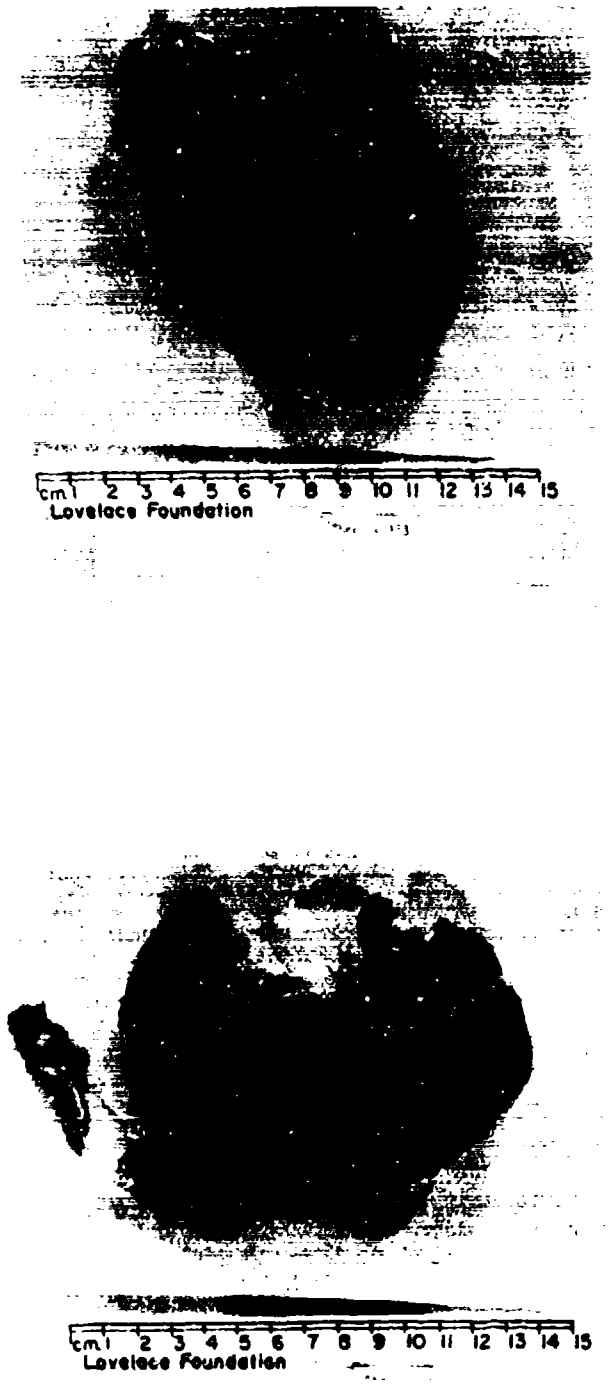


Figure 12. Heart of a sheep survivor. Upper photo shows contusions in the epicardial musculature. Lower photo shows the same heart opened to show contusions of the endocardial lining of the left ventricle.

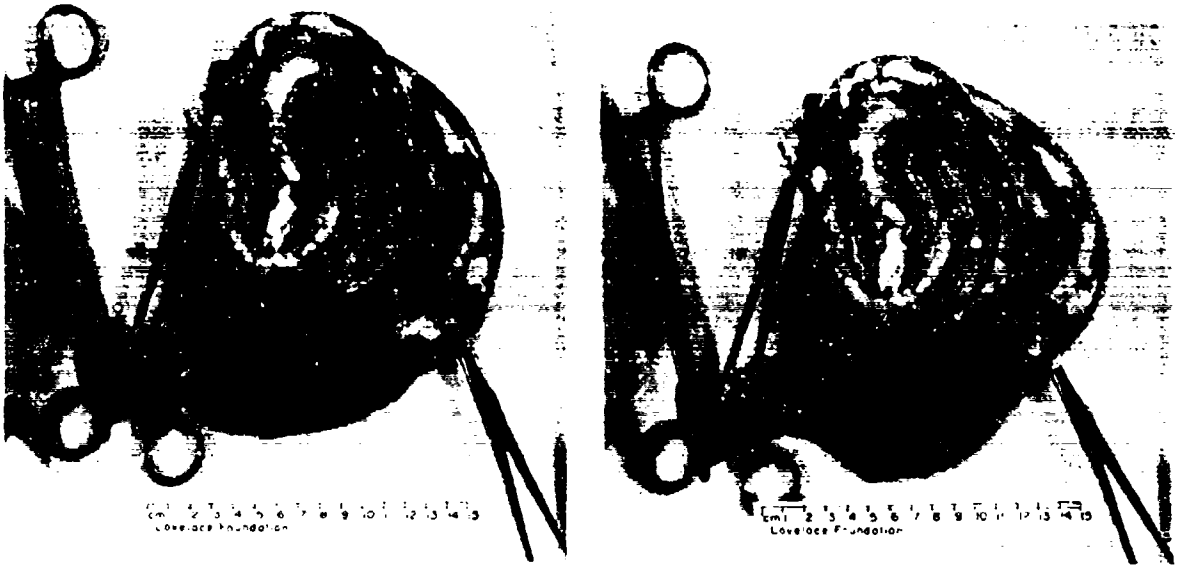


Figure 13. Distal portion of large colon and spiralis of surviving sheep. Left view is of the submucosal contusion as seen through the outer intact wall. Right view is with the large colon opened to show ulceration and hemorrhages into the lumen.

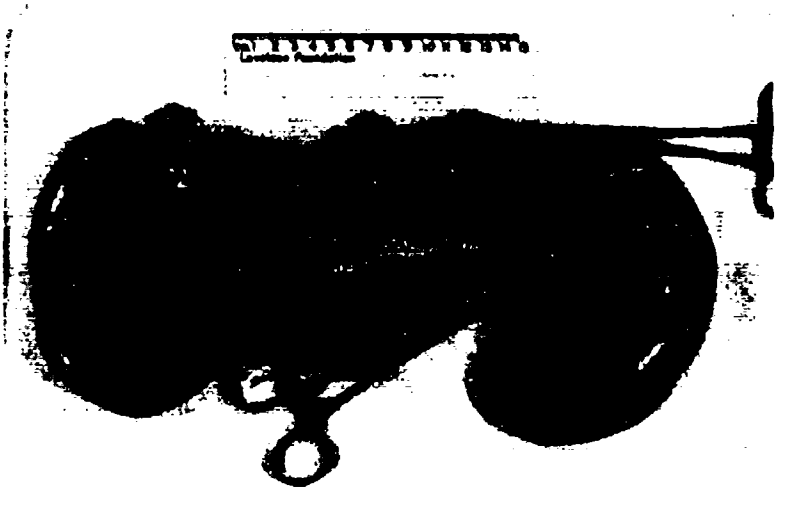


Figure 14. Caecum and large colon of sheep with a five-minute death showing a perforation surrounded by a small area of dark contusion.

2.3.1.3 Heart. Blast lesions to the heart are contusions in its outer surface (epi-cardial muscle) and inner surface (endocardial muscle) and are shown in Figure 12. These contusions are usually confined to the ventricles (in particular, the left one). Since the heart does not contain air, the damage probably is a result of its contact with the lung. An enlarged right ventricle is a relatively common finding at postmortem examinations of humans and animals subjected to blast. Cardiac contusions occur at relatively low blast levels. As already mentioned one of the important blast lesions is coronary air embolism.

2.3.2 Intra-Abdominal Injuries.

Gastrointestinal Tract. Damage is usually confined to the stomach and large intestine at locations where there is gas, but may spread to other portions of the GI tract and to solid organs such as the liver, spleen and kidneys in close contact with the gas-containing regions.

In general, the organs of the digestive tract are made up of an outer layer (serosa) and an inner layer (mucosa) with usually two thin muscle layers between them. Injury of minimal severity consists of light contusions to the serosal tissue only. Moderate injury consists of submucosal contusions involving the muscular lining with hemorrhaging into the lumen (Fig. 12). Perforation of the entire organ wall would be the most dangerous form of injury, because the contents of the GI tract spill into the abdominal cavity. It is common to find small ulcerations of the mucosal lining with blood clots attached. There may be large areas of submucosal contusions without perforations of the wall, or perforations of the wall surrounded by just a small area of submucosal contusion (Figs. 14 and 15).

Contusions of the wall of the digestive tract may rupture after becoming necrotic several days after blast exposure, and causing peritonitis from the introduction of the contents of the GI tract into the abdominal cavity. Such a result has been seen in underwater blast victims, but has not been studied with animals. Damage to the solid organs, including the liver (Figure 16), spleen and kidneys, ranges from light subcapsular contusions to subcapsular hematomas to rupture. Rupture or maceration of these organs results in a hemoperitoneum (blood in the abdominal cavity).

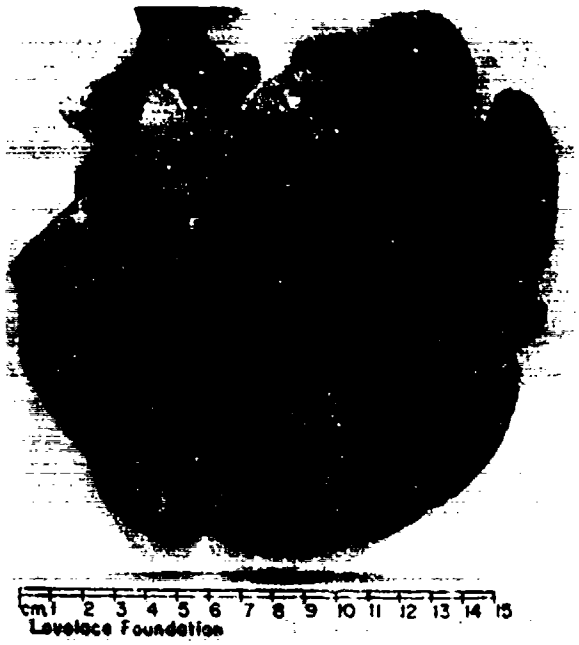


Figure 15. Dorsal view of lungs and GI tract from dog sacrificed 24 hrs following blast exposure. Very severe lung hemorrhage, lung weight 2.48% of the body weight (upper photo). Gastrointestinal tract (lower photo) of the same animal showing perforations of colon, ulcerations lining the rectum, and submucosal contusions of stomach and small intestine.

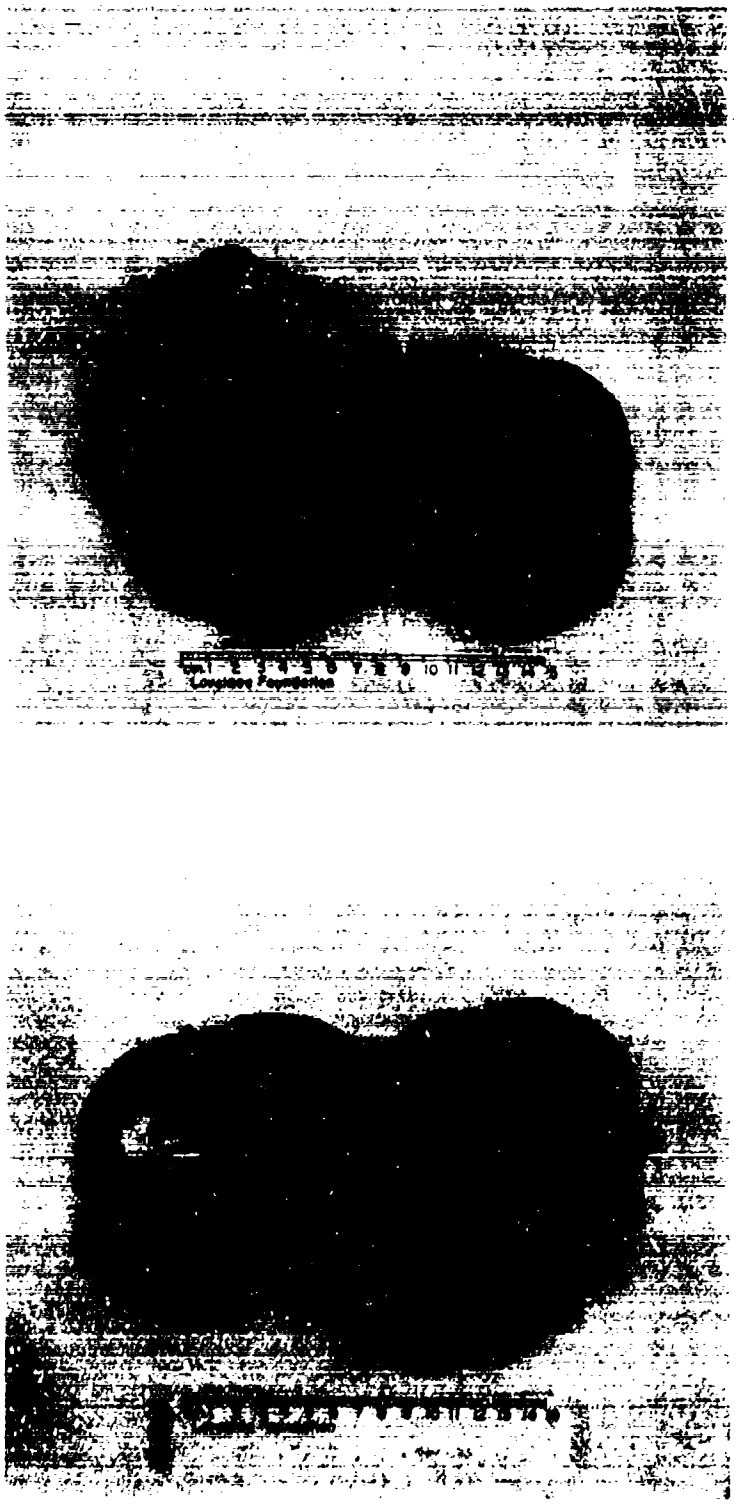


Figure 16. Liver from sheep, a seven-minute death. Upper photo is of the parietal surface showing subcapsular hematomas. Lower photo of visceral surface shows lacerations.

2.3.3 Auditory System.

The auditory system consists of three regions: the outer ear (made up of the pinnae and auditory canal); the middle ear (containing the tympanic membrane (ear drum) and ossicles that transmit sound vibrations from the ear drum to the inner ear); and the inner ear, a conch-shaped cavity (cochlea) containing embedded hair cells and a fluid (endolymph). These hair cells convert the sound vibrations in the fluid into nerve impulses that are conducted to the brain via the auditory nerve. The organ for balance, the semicircular canals, are connected to the cochlea and share the same endolymph.

2.3.3.1 Ear Drum Rupture. The ear drum may begin to fail at blasts as low as 3 psi. This would consist of small tears or perforation of a trivial nature and would be self-healing. The most severe form of this lesion would be the eardrum completely destroyed with hemorrhage and fracture or dislodgement of the ossicles. Severe ear drum rupture could be expected to begin at around 8 psi. Hearing loss associated with middle ear injury has been termed conductive hearing loss. The amount of hearing loss would range from 0 to 15 dB to 50 to 70 dB for the most severe cases.

2.3.3.2 Inner Ear Injury. Blast levels lower than those required to rupture the eardrum may damage hair cells in the cochlea. Loss of hair cells usually causes a temporary threshold shift in hearing that might also be permanent. There is an immediate onset of tinnitus (ringing in the ears) interfering with hearing, and its intensity and duration is related to the extent of damage to the hair cell population. Hearing loss by this method is termed neurosensory. In the acoustical field, the blast level is expressed as impulse noise in dB units. For instance 1 psi is 171 dB, 3 psi is 180 dB and 14.7 psi is 194 dB. MIL-STD-1447 gives the damage risk criteria for personnel exposed to impulse noise. The criteria specify the number and intensity of impulse noises allowable per day. Hearing protection is required above 140 dB (0.03 psi).

2.3.4 Orbital Blow-out Fractures.

This lesion has been found in dogs, sheep and monkeys that were exposed to very intense blasts (>100 psi). Evidently the overpressure is transmitted

hydraulically by the eyeball to the bony floor of the eye orbit, resulting in a depressed fracture of these bones. This lesion has been described in people struck in the eye with blunt objects such as baseballs, wine bottle corks, fist, etc. The eyeball may be displaced downward in some of these cases, resulting in double vision.

2.3.5 Brain.

Most recent workers agree that the brain itself is not damaged by air blast. Before World War II there was speculation that the brain and spinal cord was a site of damage to explain the central nervous (CN) system dysfunction, shell shock, etc. As already mentioned, the German workers during WW II discovered that air emboli in the brain were the reason for the CN effects.

2.3.6 Arterial Air Embolism.

As the small air sacs (alveoli) in the lung area are disrupted, air enters the vascular system and travel throughout the body. These air bubbles observed in the coronary arteries of the heart and in the arteries in the brain (Fig. 17) are the cause of most deaths from blast. Deaths of most animals within one hour from blast are due to air emboli in their heart or brain. Air emboli are not found in surviving animals.

2.4 PROGNOSIS.

Typically, the survival time of animals and humans dying from primary blast is short. The majority die within one hour of blast from air emboli. More delayed deaths, within a few hours after blast are probably caused by suffocation from blood and fluids obstructing the airways and from intra-abdominal hemorrhaging. Even though the lung may be almost completely hemorrhaged, the small remaining normal areas are believed to be sufficient to sustain life, thus lung hemorrhage per se is not the cause of death.

Pulmonary hemorrhage resolves in a remarkably short time. In both humans and animals there is a marked improvement in 24 hours. There is a decided clearing of the hemorrhage in one week, and there are only slight remnants of the contusions at three weeks.

The prognosis of intra-abdominal injuries is poor. The mortality rate is high within the first week following injury with or without treatment.

Figure 17. Air emboli in cerebral blood vessels of sheep with a two-minute death.

SECTION 3
BLAST INJURY IN RELATION TO BLAST DOSE

Blast overpressures required to produce specified injury levels in man are presented in this section. Specifically, the incident overpressures that would inflict trivial, slight, moderate, severe and very severe injuries to the lung and gastrointestinal (GI) tract are estimated, based on best available data. The highest overpressure for which no overt injury occurs also has been estimated. These values are applicable to man when prone, oriented end-on to long-duration blasts at sea level, and to man exposed to blast while inside a foxhole.

3.1 PHYSICAL CHARACTERISTICS OF BLAST WAVES.

A typical pressure-time pattern measured by a transducer oriented side-on to the blast flow in free air is illustrated in Figure 18. It is known as an "ideal" or "classical" blast wave produced when the detonation and measuring transducer are well above the surface or when the transducer is on the ground and the detonation is a surface burst. These blast waves have a positive phase and a negative phase. In the positive phase, the air is compressed almost instantly to a peak value and then decays exponentially to the ambient level (base line). The pressure continues to drop below the ambient at the start of the negative phase. During the positive phase, the flow or blast winds are away from the explosive source, but reverse their direction during the negative phase.

Blast wave parameters of interest in connection with blast injury are the peak pressure in the incident or reflected waves, the dynamic pressure, and the duration of the positive phase. The peak pressure is read from base line to the maximum value, and the positive duration, from the shock front to the point (Figure 18) where the pressure first crosses the base line. The dynamic pressure (q) is a measure of the blast flow; it is the difference between the pressure measured face-on and that measured side-on. The q may be expressed as one-half the air density times the air particle velocity squared. Reflected pressure results when the incident shock strikes a hard surface perpendicularly (or nearly so) to its travel. The reflected pressures may be from 2 to 8

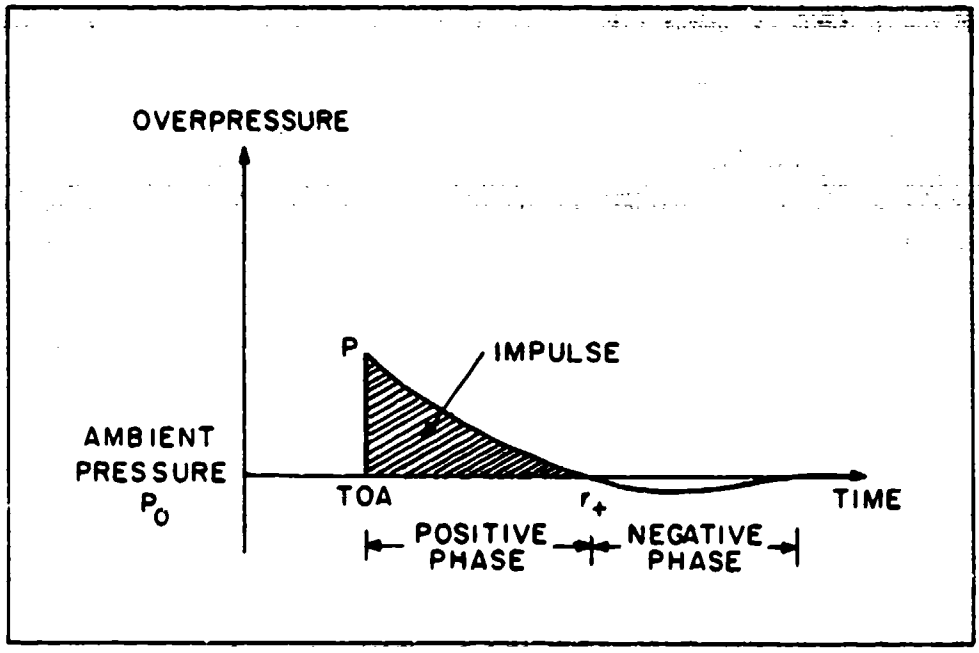


Figure 18. Pressure-time pattern of an ideal blast wave as measured in the open by a transducer-oriented side-on to the approaching wave.

times the magnitude of the incident shock.

For a given detonation, the peak pressures decrease with distance from the charge, and the positive pressure durations increase. The duration of the overpressure is related to the explosive yield. For small, high explosive charges, overpressure durations are on the order of tens of milliseconds; for nuclear explosions the duration is hundreds of milliseconds to seconds depending on yield. In order to double the distance at which a given peak overpressure is obtained, the yield from a nuclear explosion must be increased eightfold.

Values of reflected pressures, dynamic pressures and wind velocities as a function of the incident pressures are listed in table 1. It can be seen that the dynamic and reflected pressures increase in magnitude out of proportion to the incident pressure. A doubling of the incident pressure from 10 to 20 psi increases the dynamic pressure by a factor of four (2 to 8 psi). In a similar manner, the reflected pressure increases from 25 to 60 psi over that range of incident pressures.

Table 1. Approximate relationship between incident, reflected, and dynamic pressures and wind velocities calculated for blasts at sea level conditions.

Associated overpressures			Wind Velocity
Incident psi	Reflected psi	Dynamic psi	mph
1	2	0.02	40
2	4	0.1	70
5	11	0.6	160
10	25	2	290
20	60	8	470
30	90	16	670
50	200	40	940
100	500	125	1500

Note: A wind of 120 mph exerts a dynamic pressure of approximately 0.25 psi.

It is usually stated that the negative phase of the blast wave does not contribute to blast injury or physical damage. Sometimes doors and hatches of structures in some above-ground nuclear tests, although constructed to resist high overpressures, were torn away by the underpressure because underpressure was not taken into account in their design. Debris generated and lofted by the positive phase and swept closer to the explosive source by the negative phase winds was sometimes incorrectly interpreted to mean the damage was caused by the suction phase. There is insufficient evidence to define the role, if any, played by the negative phase in blast injury.

In experiments which animals are exposed to *pressure waves reflected against a large surface*, the effective pressure is reported as the air blast dose. These peak reflected pressures, when scaled to man, represent the effective pressure or peak incident overpressures that cause the specific injury levels, such as those in Table 6. In other experiments animals are tested *side-on* to the blast in the *free stream*. In this case, the incident pressure, as measured by a transducer side-on, is reported as the blast dose even though the target also received the dynamic pressure load. To apply side-on values to the end-on man situation, it is necessary to add the dynamic pressure to the side-on incident overpressure value. The sum of the two then

becomes the peak incident pressure. For example trivial lung injury occurs in subjects end-on to the blast receiving an incident shock of 14.4 psi as the effective pressure--the dynamic pressure does not add to the dose. Individuals broad-side to the blast, loaded with an incident shock of 11.5 psi and a dynamic pressure of 2.85 psi, would also experience an effective overpressure of 14.4 psi and trivial lung injury. An incident shock of 6.2 psi will result in a reflected pressure of 14.4 psi, which is the effective pressure for a subject against a reflector.

3.2 SCALING FOR ANIMAL DATA TO MAN

Blast overpressures that produced the selected injury levels in experimental animals were taken from the literature, then scaled to sea level, to 70-kg man and to blast waves of 200-milliseconds (ms) duration. The latter is considered to be a long duration beyond which the blast dose required for a given primary blast injury would not change. Scaling is necessary because much of the data from Lovelace Foundation, Albuquerque, N.M., was obtained at 12 psia (ambient pressure), the body weight of the animals was less than 70 kg, and duration of most of the blasts were less than 200 ms. Except for the ISL results (Ref. 27), data having overpressure durations of less than 10 ms are not included in the compilation, nor are rodent data. The method of scaling these parameters to sea level is taken from Reference 2. Assume an animal with a body mass, m, is exposed at an ambient pressure, P_0, to a classical blast wave with a positive phase duration, t_e, and a peak overpressure, P_e. According to the semi-empirical theory, the same injury level experienced by the animal is experienced by man exposed at sea level to a classical blast wave with a positive-phase duration, t_{s1}, and a peak overpressure, P_{s1}, thus:

$$P_{s1} = P_e \frac{(61.5)}{(P_{sw})} \frac{(14.7)}{(P_0)} \frac{1+6.76(t_{s1})^{-1.064}}{1+6.76[t_e(70/m)^{1/3}(P_0/14.7)^{1/2}]^{-1.064}} \qquad (1)$$

where m is expressed in kg, the duration, in ms, and the pressure, in psi. P_{sw} is a square-wave pressure that results in 50% animal mortality and is an index of blast tolerance that is independent of body mass. The P_{sw} is determined for each of 12 species of mammals from their dose-response curves (Ref. 2). The P_{sw} for a 70-kg man is 61.5 psi--the geometric mean of values for large

animals that range from 50 psi for steer to 72 psi for burros. Other P_{sw}s of interest are: 68.5 psi for sheep, 56.6 psi for dogs, and 62.5 psi for swine.

3.3 AIR BLAST INJURIES IN THE OPEN

3.3.1 Lung Injury.

Specific injury levels chosen for the lungs are defined in Table 2. A lung damage grading scheme in use at Walter Reed Army Institute of Research (WRAIR) is taken into account or incorporated into our scheme. The WRAIR grading system considers the percent surface area of the lung that is hemorrhaged. Trivial lung hemorrhages in the form of petechia (pin-head size hemorrhages) are usually graded as the threshold injury. This type of injury has no physiological consequences and, in fact, may occur in control animals. Moderate degrees of lung hemorrhage in dogs increase respiratory rate at 20 to 40 minutes after blast. Severe lung hemorrhage among animals causes an immediate increase in respiratory rate as well as rapid and shallow breathing. An increase in lung weight can be detected in cases of moderate lung hemorrhage. Severe lung hemorrhage can prove to be lethal when combined with exertion.

Table 2. Injury levels selected for the lungs.

Injury Severity	Lung Hemorrhage
None	none
Trivial	petechial
Slight	ecchymotic or small light spots
Moderate	small isolated confluent <30% areas
Severe	large confluent 30-60% areas extending deep into the parenchyma
Very Severe	entire lobes confluent >60% of area

Table 3. Long-duration incident blast overpressure required to produce selected lung injury levels in man prone end-on to the blast at sea level.

Incident Overpressure, psi Severity of Lung Injury						Experimental Conditions[b]	Reference
none	trivial	slight	moderate	severe	very severe		
12.4	18.2	26.9	34.6	42.1	48.4	Dog exposed to reflected waves in air-driven shock tube (N=60)	15
10.4	19.2	23.1	--	--	32.7	Sheep exposed to reflected waves in air-driven shock tube (N=30)	18
10.0	11.0	13.5	27.0	30.0	40.0	Sheep exposed to reflected waves in explosive-driven shock tube (N=62)	20
--	15.5	25.2	--	--	--	[c]Goats tested side-in to incident waves from 500-Ton Explosion (N=20)	13
8.1	--	15.8	--	31.2	48.5	Sheep vertical and facing incident blasts from 64-lb charges (N=8)	Lovelace Unpub 1975
--	--	17.6	24.7	36.7	45.0	Sheep prone on concrete pad given reflected waves from 64-lb charges detonated over head (N=18)	Lovelace Unpub 1966
10.1	--	--	--	--	--	Sheep side-on to 20 blasts; incident waves from small charges (N=4)	27
12.0	--	--	--	--	--	Swine side-on to 20 blasts; incident waves from small charges (N=4)	27
--	11.2	--	38.9	--	--	Sheep side-on to incident waves from 64-lb charges (N=16)	21
9.0	--	12.4	20.2	29.0	--	[c]Swine side-on to incident waves from H.E. charges 1-5-lb (N=16)	28
--	11.6	--	28.5	36.7	37.6	Sheep exposed to reflected waves in explosive driven shock tube (N=24)	12
Avg 10.3	14.4	19.2	29.0	34.3	42.0		

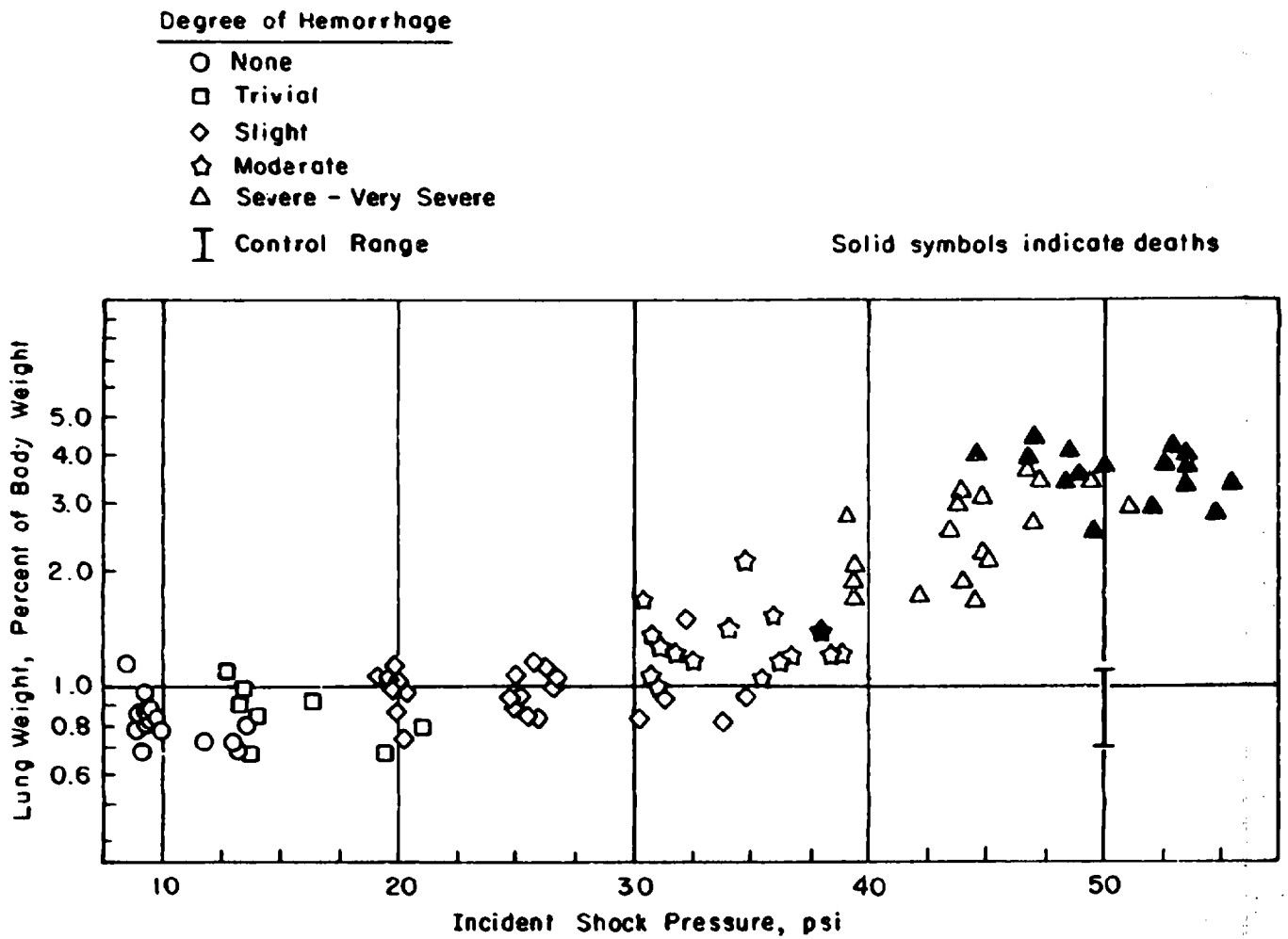
a -- means no data
b: experiments conducted at ambient pressure of 12.0 psia, except c.
c: experiments conducted at ambient pressure of 14.7 psia.

Table 3 summarizes the estimated incident blast overpressures required for the selected injury levels in man, and includes the experimental conditions involved during the animal test and the references (12, 13, 15, 18, 20, 21, 27, 28). The values are scaled from the animal data and are applicable to man at sea level. According to the averages, the highest overpressure for no lung injury in man is just over 10.3 psi. Moderate degrees of pulmonary hemorrhages should occur at about 29 psi. An incident pressure of 14.4 psi is expected to cause trivial lung hemorrhage in man in the end-on orientation. A comparable value of 12 psi is reported (Ref. 2) for the threshold of lung injury in man in this orientation; the authors obtained that value by taking one fifth of the 50% lethal dose (P_{50}). The 14.4 psi for trivial injury reported here is more in agreement with the one of 15 psi (Ref. 16) arrived at by taking one fourth of the P_{50}.

It should be noted that there is variation in the severity of lung hemorrhages within groups of animals receiving the same blast intensity. For illustration, Figure 19 shows the distribution of lung weights and degree of lung hemorrhage in dogs as a function of blast overpressure. The figure is taken from Reference 15 with the pressure values scaled to man at sea level. Experimenters did not always define terms like "threshold", "slight", "moderate" and "severe", so it was necessary to make subjective judgments in order to assign categories. An effort was made to keep subjective decisions to a minimum, and no attempt was made to determine pressure values or injury levels between groups.

3.3.2 Gastrointestinal Tract Injury.

Table 4 lists the injury levels selected for the GI tract. The same descriptors as those for the lung were used. Unfortunately, GI-tract injury received little attention and emphasis in the reports reviewed. Like the lung, the GI-tract injuries reported in the experimental animal studies were graded, and the corresponding blast values, scaled to man end-on at sea level (Table 5). In general, the overpressure levels for a specified injury in the GI tract are lower than for the lung. The highest pressure associated with no injury is 7.0 psi, which compares with 10.3 psi for the lung. Trivial injury occurs at 8.4 psi, compared to 14.4 psi for the lungs. Moderate injury is predicted at 23.6 psi, compared with 29.0 psi for the lung.



Data from Ref 15

Figure 19. Degree of lung hemorrhage and lung weight in relation to the incident overpressure. The lung weights are from dogs, and the incident overpressure values are those applicable to man end-on to a long-duration blast at sea level.

Table 4. Injury levels selected for the gastrointestinal tract.

Injury Severity	GI tract injury
None	none
Trivial	petechia or small area of discoloration
Slight	small areas of light subserosal contusions
Moderate	small area of submucosal contusions with little or no disruption of mucosal layer
Severe	large areas of submucosal contusions with disruption of mucosal membrane and blood clots extending into lumen
Very Severe	Disruption of mucosal layer with hemorrhage into lumen or perforation into abdominal cavity and rupture of solid organs

3.4 AIR BLAST INJURIES INSIDE FOXHOLES.

The shape of a blast wave is altered upon entering a structure. It consists of an initial diffraction phase associated with the entering shock front reflecting from the inner walls, floor and ceiling. These reflections or reverberations are superimposed on the fill phase, a relatively slow-rising pressure. Biologic response to these complex wave patterns is different from the response to the ideal or classical blast wave. A greater overpressure is required to produce the same effect. The magnitude of the diffraction and fill phases depends to a large extent on the structure's volume-to-entrance area (V/A) ratio. Open foxholes having V/A ratios on the order of 4 to 5 feet have the peak pressure in the diffraction phase and a relatively insignificant fill phase (Fig.20). The peak reflected pressure inside the foxhole may be several times that of the incident wave on the surface. However, specific injuries resulting from pressures that rise in several steps (such as inside the foxhole) are much less severe than those from ideal waves or reflected pressures rising in a single step having the same peak pressures. Structures

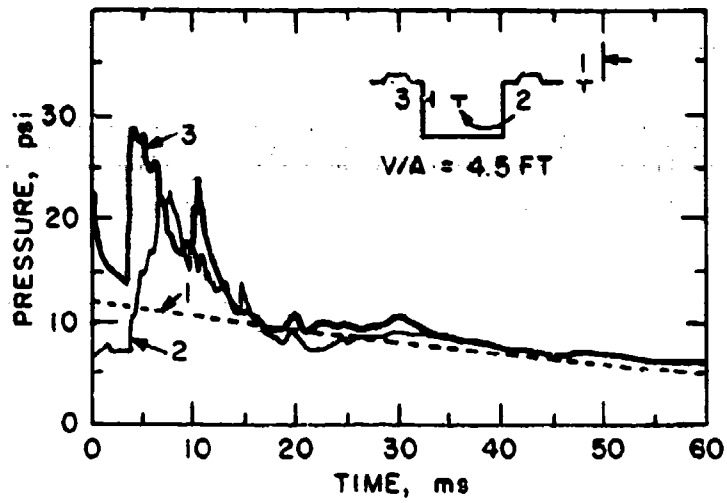
having large V/A ratios of several hundred feet, such as civilian structures and shelters, have relatively low pressure in the reflections within the diffraction phase and maximum pressure in the fill phase (Fig. 21). Displacement of personnel in line with openings into these structures constitutes the worst hazard in these enclosures.

Studies with animals exposed to blast in model and full scale foxholes show that foxholes side-on to the blast afford more protection than those end on to the blast. Leaning against the downstream wall or lying prone in the foxhole are the worst locations, whereas crouching near the upstream wall is the best location. There is little displacement inside the foxhole provided the occupant's head is below the lip. A foxhole shields its occupants against the dynamic pressure component of the blast wave that would be experienced on the surface.

3.4.1 Lung Injury

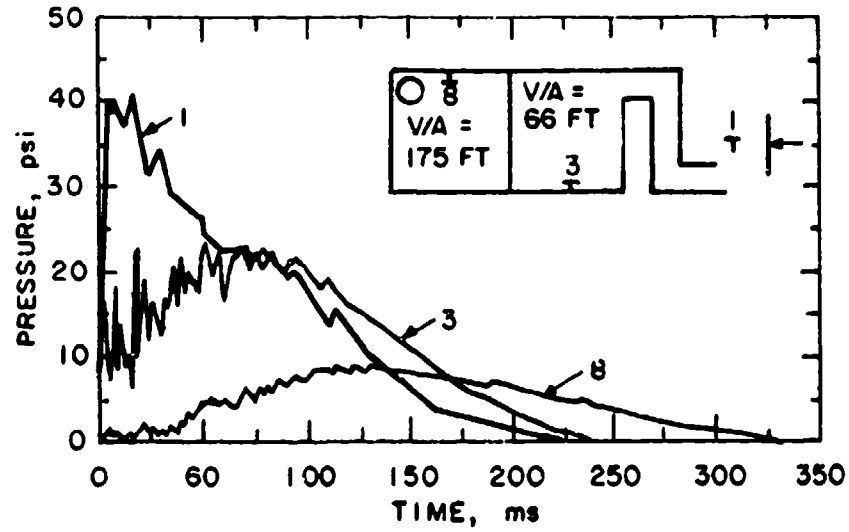
Incident blast pressures required to produce graded levels of lung hemorrhage for men in foxholes are estimated specifically for man crouching in the US Army standard open two-man foxhole, oriented side-on to long-duration blasts at sea level. Estimates are based on results from experiments where large animals were exposed to blast while in the standard foxhole (or its equivalent) oriented side-on to the blast (Ref. 14, 17, 22, 26). The degrees of lung hemorrhages selected are the same as those chosen for man in the open; trivial, slight, moderate, severe and very severe. Incident overpressures associated with these lung injury levels found in animals are scaled to man at sea level and for long-duration blast waves. The one exception is in connection with the one-ton shot, where the durations of 12 ms to 18 ms apparently are too short for the scaling method to apply. Consequently, the incident pressures obtained on the one-ton shot are divided by the ratio of the pressure value for 50% combat ineffectiveness (at 14 ms) with the one for 200-ms duration shown in Figure 5 of Reference 22.

As seen in Table 6, incident waves of 7.6 psi would not be expected to produce lung hemorrhages for man inside the two-man foxhole. Mean value of 14.6 psi would cause slight lung hemorrhages, and 19.9 psi would produce moderate lung hemorrhages. Severe lung injury would occur at incident pressures of 30 psi, and very severe, at 35 psi, which is near the one percent mortality value.



Data from Ref. 17

Figure 20. Pressure-time patterns measured on the side-wall (record 2), downstream wall (record 3), and incident (record 1) to a two-man foxhole oriented end-on to the blast from a 500 ton TNT charge.



Data from Operation Plumbob

Figure 21. Pressure-time patterns measured inside an underground shelter having two 12 X 12 X 8 ft rooms. One room filled through a 3 X 6 ft door (record 3) and the other, through a 3 ft diameter escape hatch (record 8). The incident wave (record 1) was from a 10-kt nuclear detonation.

Table 5. Long-duration incident blast overpressure required to produce selected gastrointestinal tract injury levels in man, prone, end-on to the blast at sea level.

	Incident Overpressure, psi Severity of GI-Tract Injury						Experimental Conditions[b]	Reference
	none	trivial	slight	moderate	severe	very severe		
	a)--	10.4	--	23.1	--	--	Sheep exposed to reflected waves in air-driven shock tube (N=30)	18
	7.0	8.0	12.0	18.0	30.0	--	Sheep and swine exposed to reflected waves in explosive-driven shock tube (N=62)	20
	7.0	--	12.5	--	21.7	29.5	Sheep vertical and facing incident blasts from 64-lb charges (N=8)	Lovelace Unpub 1975
	--	--	17.6	24.7	36.7	45.0	Sheep prone on concrete pad given reflected waves from 64-lb charges detonated overhead (N=18)	Lovelace Unpub 1966
	--	6.8	--	--	22.0	--	Sheep side-on to incident waves from 64-lb charges (N=122)	21
	--	--	--	28.5	36.7	37.6	Sheep exposed to reflected waves in explosive-driven shock tube (N=24)	12
Avg	7.0	8.4	14.0	23.6	29.4	37.4		

a --:no data b: all data obtained at a P_o=12 psia and corrected to sea level

Table 6. Incident blast overpressures required to produce lung injury in man exposed to blast crouching inside a foxhole.

	Incident Overpressure, psi Severity of Lung Injury						Experimental Conditions	Reference
	none	trivial	slight	moderate	severe	very severe		
	5.4	11.0	14.2	17.6	32.9	--	Dogs standing in standard 2-man foxhole side-on to 40kt detonation (N=16)	26
	--	--	15.0	20.0	25.0	30.0	Sheep verticle in standard 2-man foxhole side-on to blast from 1-TON HE (N=14)	Lovelace Unpub
	--	--	--	20.9	--	33.3	Goat standing in open portion of half covered 2-man foxhole side-on to 100 TON HE (N=5)	14
	9.9	--	--	--	--	--	Sheep verticle in standard 2-man foxhole side-on to blast from 500 TON HE (N=10)	17
	--	--	--	21.0	30.0	40.0	Estimates for man in standard 2-man foxhole side-on to long duration blast	22
Avg	7.6	11.0	14.6	19.9	29.3	34.4		

*US Army standard two-man foxhole oriented side-on to the blast foxhole measures 2ft x 6ft x 4.5 ft deep

Some estimates of the incident pressures required for lung injury in man in foxholes date back to 1971 (Ref. 18). They are based on model experiments wherein rats are exposed to blast in one seventh scale foxholes and from step-loading experiments using a number of animal species. It is reported that no lung injury occurs at 9.8 psi, there is a low probability of slight lung injury at 12.3 psi and a high probability of slight lung injury at about 15.9 psi. These results are in agreement with the results reported in the present study.

3.4.2 Gastrointestinal Tract Injury.

There is insufficient information in the reports on animal testing to estimate the extent of GI tract injury for man in foxholes. One set of estimates can be made by lowering the incident overpressures required to cause lung injury in man in foxholes (Table 7) because, on the average, the overpressures required to cause GI tract injury by 24.1% for man in the open (Table 5) are 24.1% lower than those for lung injury (Table 3). This set of estimates considers the fact that the animals tested in foxholes are exposed to reflected pressures that rise in several steps.

Another set of more conservative estimates of overpressures for lung and GI tract injuries within a foxhole assumes that the reflected pressures rise in a single step (Table 8). The peak reflected pressures for each injury's severity are of the same magnitude as the incident pressures for man end-on in the open (Table 3). The corresponding incident pressures applicable to the foxhole are taken from Figure C-47 (U), page C-72 of the US Army Personnel Risk and Casualty Criteria (PRCC), a curve relating the peak reflected pressure measured on the downstream wall of the foxhole, side-on to the incident wave. The log of the reflected pressure inside the foxhole is equal to 0.53144 plus 0.8616 times the log of the incident pressure.

The incident overpressures obtained by this method (Table 8) are considerably lower than those in Table 7 because of the increased biologic tolerance to step-rising reflected waves.

Table 7. The incident blast ovepressures required to produce lung and GI tract injuries in man located inside an open foxhole. (multiple step rising reflected wave)

Severity of Injury	Incident Overpressure, psi Lung Injury	GI Tract Injury
None	7.6	5.8
Trivial	11.0	9.4
Slight	14.6	12.5
Moderate	19.9	17.1
Severe	29.3	25.2
Very Severe	34.4	29.5

Applicable to man crouching inside a two-man foxhole oriented side-on to the blast. The foxhole measures 2 X 6 X 4.5 ft deep. The incident overpressures for GI Tract injury are 24.1% lower than those for lung injury.

Table 8. Estimated long-duration blast levels required to produce lung and gastrointestinal tract injuries in man located inside an open foxhole. [a](single step-rising reflected wave)

Injury Level	Lung Injury Overpressure, psi		GI-Tract Injury Overpressure, psi	
	Incident Outside[b]	Reflected Inside[c]	Incident Outside[b]	Reflected Inside[c]
None	3.6	10.3	2.3	7.0
Trivial	5.3	14.4	2.9	8.4
Slight	7.5	19.2	5.2	14.0
Moderate	12.0	29.0	9.5	23.6
Severe	15.3	34.3	12.2	29.4
Very Severe	18.5	42.0	16.2	37.4

[a]Open two-man foxhole 2 X 6 X 4.5 ft deep, side-on to blast.
[b]The incident pressures associated with the reflected pressures are from the PRCC Figure C-47 (U) page C-72.

[c]Reflected pressures are equal to the incident pressures required to produce the corresponding injury levels for man prone end-on to the blast in the open. Reflected pressure is measured on the downstream wall of the foxhole.

SECTION 4
CONCLUSIONS

The direct blast damage mechanism affects the three organ systems that contain air or gas. Injuries are hemorrhagic in nature, affecting only internal organs. There are few if any external signs of injury.

Respiratory system -- Typical lesions are pulmonary hemorrhage and contusions lining the air ways including the trachea, larynx and sinuses. Coronary and cerebral air embolism arising from the disrupted alveoli in the lung are the cause of early deaths.

Gastrointestinal system -- Injuries range from light contusions of the serosal membrane, submucosal contusions, with or without disruption of the mucosal membrane and hemorrhage into the lumen, to perforations into the abdominal cavity.

Solid organs (heart, liver, spleen and kidneys) in contact with the gas-containing portions of these systems may become damaged.

Auditory system -- ear drum rupture may consist of small perforations or complete destruction of the tympanic membrane, with displacement or fracture of the ossicular chain. Relatively low blasts may damage the hair cells of the inner ear resulting in a temporary or permanent hearing loss.

Blast criteria are stated in terms of the peak incident overpressure, for personnel in the open, foxholes and field fortifications. Other blast wave parameters considered in establishing the criteria are the dynamic pressure (blast wind), overpressure duration, and reflected pressure.

The blast response of over 300 large animals (sheep, swine and dogs) in terms of the peak overpressures and corresponding injuries were scaled to man at sea level. The peak incident overpressures expected to produce selected lung and gastrointestinal injuries in man exposed to long-duration blast waves while prone end-on and while crouching inside a two-man open foxhole have been calculated and are summarized in the following tables 9, 10 and 11.

Table 9. Summary of long-duration blast levels (free air) required to produce selected lung and gastrointestinal tract injuries in man prone, end-on at sea level.

Severity of Injury	Incident Overpressure, psi Lung	GI tract
None	10.3	7.0
Trivial	14.4	8.4
Slight	19.2	14.0
Moderate	29.0	23.6
Severe	34.3	29.4
Very Severe	42.0	37.4

The incident overpressures measured by a transducer oriented side-on to the blast represents the effective pressure applicable to man end-on to the blast. For personnel standing or prone, long axis perpendicular to the blast, the dynamic pressure has to be added to the side-on pressure to obtain the effective pressure. If individuals are against or near a reflecting surface, the peak reflected pressure becomes the effective pressure, but the required dose is still expressed in terms of the corresponding incident pressure.

Table 10. Summary of the incident long-duration blasts required to produce lung and GI tract injuries among personnel inside foxhole. (reflected pressure inside foxhole rises in multiple steps)

Severity of Injury	Incident Overpressure, psi Lung Injury	GI Tract Injury
None	7.6	5.8
Trivial	11.0	9.4
Slight	14.6	12.5
Moderate	19.9	17.1
Severe	29.3	25.2
Very Severe	34.4	29.5

Applicable to man crouching inside a two-man foxhole oriented side-on to the blast. The foxhole measures 2 X 6 X 4.5 ft deep. The incident overpressures for GI Tract injury are 24.1% lower than those for lung injury.

Table 11. Summary of the long duration incident overpressures required to produce lung injury and GI tract injury among personnel inside foxholes. (reflected pressure inside the foxhole rises in a single step)

Severity of Injury	Incident Overpressure, psi	
	Lung Injury	GI Tract Injury
None	3.6	2.3
Trivial	5.3	2.9
Slight	7.5	5.2
Moderate	12.0	9.5
Severe	15.3	12.2
Very Severe	18.5	16.2

Applicable to man crouching inside a two-man foxhole oriented side-on to the blast. The foxhole measures 2 X 6 X 4.5 ft deep.

SECTION 5
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