

AN ASSESSMENT OF THE CURRENT STATE-OF-THE-ART
OF INCAPACITATION BY AIR BLAST

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ABSTRACT

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Target vulnerability methodology requires a criticality measure for all internal components which contribute to a system or to a system's weapon effectiveness, including that of the human target. Such measures have been developed for personnel targets for kinetic energy penetrators; however, there is presently no generally accepted quantitative measure of incapacitation to infantry or crew personnel from the prime blast threat. (Vulnerability analysts presently use lethality data derived from Lovelace Foundation research to infer an incapacitation level for blast, but these criteria are not very realistic in that they tend to underestimate casualty production from blast threats.) Thus, a generalized criteria for estimating incapacitation to military personnel from air blast overpressures is urgently needed to provide vulnerability analysts a realistic measure of blast effectiveness as well as to establish a common base for comparing incapacitation to personnel from blast and from kinetic energy threat mechanisms.

To address this need, an assessment of the current state-of-the-art of incapacitation/injury by air blast has been made through survey of both early and modern research of blast effects against personnel. Most of the studies and findings appropriate for consideration in developing a blast casualty criteria were completed in the modern or post-1950 era, which coincided with publication of the German and British World War II blast research in the open literature and initiation of nuclear testing with various animal species. The models which were generated ranged from those associated with temporary threshold shifts in hearing to those for 99 percent mortality. Bounding these two extremes were a number of studies characterizing injury or physiological damage, to which incapacitation may be related or inferred by establishing limits beyond which an individual cannot effectively perform his designated mission. The research results most germane to this consideration were Hirsch's Eardrum Rupture Criteria, Richmond's Partial Impulse Criteria for LD₅₀ Blast Waves, and Lovelace's Threshold Lung Damage Criteria. These have been evaluated, their strengths and weaknesses identified, and recommendations for their utility in vulnerability assessment are provided.

The literature search conducted by Mr. Ronald R. Rudolph, the coauthor of this paper, uncovered, reviewed, and analyzed sixty two reports from seven countries which dealt with primary blast induced injuries. Not surprisingly, one third of the documents related to the extensive blast research performed by the Lovelace Foundation, mostly during the 1960's. Another thirty three related to other US sponsored research and there were seven Swedish documents and one each from the United Kingdom, France, USSR, and Yugoslavia. Many other excellent US and foreign reports on blast injuries were scanned during the initial review but these were eliminated from further consideration because the damage mechanisms were not primary blast. The intent of our effort was to collect data oriented towards or applicable to personnel incapacitation from primary blast effects, thus secondary and tertiary effects were not considered.

We found little support in the literature for keying on the eyes, brain, central nervous system, or the skeleton as measures of effectiveness for estimating incapacitation or for relating military casualty production to sublethal dosages. In fact, our 1980 study supports the general findings and conclusions of both early and post 1950 researchers that the ear and lung systems were the most vulnerable body systems with regard to the pure blast damage mechanism. Of the two, the hearing system is universally recognized as the most vulnerable component, but not the most critical, to pure blast. Eyes are vulnerable but only if the blast causes flying debris (secondary effects). Serious lung hemorrhaging due to primary effects, quite often leading to death, apparently occurs at blast levels too low to cause damage to other body components such as the heart, the components of the abdomen, the central nervous system, or the skeleton. (The Lovelace Biomedical and Environmental Research Institute have recently found (1) that the threshold values from laryngeal lesions, 41 kPa (6 psi), and gastrointestinal tract injury, 55 kPa (8 psi), were below that for lung hemorrhage, 76 kPa (11 psi). In the opinion of medical experts, these are considered slight injuries which would not be expected to impair human performance.) Heart damage has apparently been observed in some experimentally produced exposures of animals; however, this type of damage has generally been assessed to be a consequence of lung hemorrhaging. Skeletal damage does not occur unless the blast winds are great enough to cause body translation (tertiary effects). The skull apparently provides sufficient protection to the brain if the body of the

exposed victim cannot be translated. No evidence was uncovered that suggested that significant damage to the central nervous system could result from blast exposures lower than that required to produce lung hemorrhaging.

Prior to our survey of the current state-of-the-art of knowledge in primary blast effects, there was no generally accepted quantified measure for inferring incapacitation of military personnel from blast induced weapons or devices. In lieu of a generalized incapacitation criteria for personnel, vulnerability analysts have usually resorted to inferring incapacitation from blast lethality criteria developed by the Lovelace Foundation for Medical Education and Research (2). These criteria functionally relate percent lethality to two characteristics of air blast; maximum overpressure and duration of the positive phase of the incident overpressure. Impulse is another characteristic to which damage is frequently correlated. While incapacitation does not imply lethality, lethal criteria were assumed to provide an upper bound for incapacitation criteria. In this context, a lower bound on incapacitation criteria could be defined from criteria associated with temporary threshold shifts in hearing. Criteria at the lower extreme are called damage risk criteria, hearing conservation criteria and design standards. They are established principally to protect the hearing organs of personnel from the damaging effects of overpressure, in general, and impulse pressure (gun fire), in particular. Examined from this point of view lethality criteria belong to the incapacitation criteria class; the design standards, damage-risk and hearing conservation criteria do not. Any criteria between these two bounded types should also belong to the incapacitation type provided that the criteria establish limits beyond which an individual cannot effectively perform his designated mission.

Criteria falling within these general bounds are:

- o Hirsch's Eardrum Rupture Criteria (3)
- o Richmond's Partial Impulse Criteria for LD₅₀ Blast Waves Augmented with the One-Quarter Rule for Estimating Threshold Lung Damage (4)
- o The Lovelace Threshold Lung Damage Criteria (2)

I would like to review each of the preceding criteria in some detail, and present the lethality, threshold lung damage, eardrum rupture, and hearing damage risk criteria in a format suitable for rational examination and then suggest some utility for their application to an interim

blast incapacitation model.

By 1962, the Lovelace team had enough data collected to be able to make a tentative estimate of man's tolerance to sharp-rising overpressures from blast (5). This estimate was based on data collected on nearly three thousand animals that either had been exposed in shock tubes to sharp rising-overpressure with long durations or in test arenas to sharp rising-overpressures with short durations. In all instances the tolerance of the animal was assessed in terms of lethality. Probit analysis was used to determine the overpressure, LD_{50} , required for the occurrence of 50 percent lethality for each of several overpressure pulse durations. The results are presented in Figure 1. Note that 50 percent lethality curves are presented for six mammalian species, two large and the remainder small. In investigating man's tolerance to blast, the Lovelace team found various species of mammals belong to either one of two groups, depending on the average gaseous volume of lungs per body mass, or the average lung density. These groups can be roughly thought of as small and large mammal groups. The goat and dog, as well as man, belong in the large species, or high tolerance group, the remaining animals in the low tolerance group. Also note the change in shape or break upward in the curves. The area where the curve breaks upward is called the critical duration and is unique to species, as can be inferred from the data presented on the graph. I will have further comments on this species characteristic later in the paper.

Regression analysis was then used to express $\log(LD_{50})$ as a linear function of $\log(BW)$, where BW is the body weight, for each of several overpressure durations: 3, 5, 10, 30, 60, and 400 msec. The results for one of these durations, 400 msec, are displayed in Figure 2. The coefficients in the displayed linear regression equation were determined on the basis of BW being expressed in grams and LD_{50} in pounds per

square inch. The formulas were then used to calculate the LD_{50} value for a 70-kg (154 pound) body weight (assumed to be the average weight of a man) for each of the above mentioned overpressure durations. The result is the middle curve shown in Figure 3. The same process was used to develop the LD_1 and LD_{90} lethality curves also displayed in the figure. For several reasons mentioned by the authors, the curves displayed in Figure 3 were only to be used as a guide. They suggested that a band running from 20 percent below to 10 percent above each curve might bracket the actual tolerance value.

Between 1962 and 1968, the Lovelace team continued to make refinements in its analytical techniques, based upon examination of the considerable amount of experimental data which by then had become available. For example, the mammal species data base was increased to thirteen with inclusion of results for the hamster, cat, burro, steer, monkey, sheep, and swine. The result was that in 1968 this team was able to express percent survival in terms of (1) maximum reflected overpressure, (2) duration of the wave, (3) body mass of the animal, and (4) an individual species tolerance index (2). At the same time, and probably most importantly, scaling of available

empirical information made it possible to apply the results to certain exposure situations in the free stream, i.e., without the reflecting surface. Figure 4 presents the revised Lovelace LD₁, LD₅₀, and LD₉₉ lethality curves for a 70-Kg man. The curves are plotted as a function of peak or maximum incident overpressure versus the duration of the positive phase and are applicable to free-stream situations where the long axis of the body is perpendicular to the direction of propagation of the shocked blast wave. Two other criteria were also developed but these will not be presented here in view of space limitations for the paper. They deal with the free-stream situation where the long axis of the body is parallel to the direction of propagation and the condition where the thorax is near a surface against which a shocked blast wave reflects at normal incidence.

was observed that petechial hemorrhages first appeared at the 83-110 kPa (12-16 psi) level and small isolated hemorrhages were produced at the 138-207 kPa (20-30 psi) area. It was not until the pressures reached the lethal range that more serious confluent hemorrhages occurred and lung weight increased significantly over control weights. The authors concluded that the threshold for petechial lung hemorrhage in dogs amounts to approximately one quarter of the LD₅₀ dose and more serious injury occurs at about the three quarter dose. Experiments with sheep exposed to reflected pressures of short duration showed threshold lung damage occurring at 207-241 kPa (30-35 psi). The threshold in this case was only slightly less than one fourth of the LD₅₀ dose of 1144 kPa (166 psi) for sheep. The Lovelace team concluded that, "It seems safe to generalize on the matter and use one fourth of the LD₅₀ dose as the beginning of lung damage and three fourths of LD₅₀ (about the threshold of lethality) as the beginning of severe lung damage." Thus, the establishment of the one-quarter LD₅₀ lethality dose for onset of threshold lung damage.

Figure 5 displays the threshold lung damage curve and the LD₁ lethality curve for the orientation of the long axis of the body perpendicular to the blast winds. (The remaining three curves shown in this figure will be discussed and explained below.)

It is the lower or 1 percent lethality curve that the vulnerability community uses as a measure of incapacitation. The logic for this choice, other than the fact that nothing more appropriate was available at the time, was that the 99 percent who survived would most certainly be completely incapacitated. It is also obvious that the use of the 1 percent lethality curve as a threshold for incapacitation underestimates the true number of casualties from blast because most certainly there would be some casualties who would be completely incapacitated for lesser levels of pressure-duration than defined for this curve.

The next descending measure of injury for which criteria exist is that for the thorax. Threshold lung damage criteria were developed by the Lovelace Foundation based primarily upon post mortem examination of the lungs of two animal species used in the lethality experiments (6). The first, for dogs showed that the incidence and degree of lung hemorrhage increased lung weight when the maximum overpressure was increased. It

The threshold criteria are referred to as "cookie cutter" criteria in that the probability of lung damage is zero if the overpressure is below the curve and unity if above. Note that the region of the LD₁ lethality curve wherein the curve breaks upward, which I earlier defined as the critical duration, lies between 25 and 30 msec, for man. Von Gierke (7) and others had observed that the magnitude of the thorax

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resonance frequency duration, the time at which the tissue is a maximum strain, is of the same magnitude and had concluded that it is the thorax resonance that determines the critical blast duration, or the bend in the blast sensitivity curves. Because of this, the critical duration has also been called the critical resonance frequency duration of the system.

The scaling of impulse for 50 percent mortality was accomplished by Richmond (6) but the consideration of impulse as a damage mechanism was first documented by the German scientist Schardin during World War II who showed by experimentation that mammalian response to air blast is more nearly dependent on overpressure impulse ($\int P dt$) if the durations are short and on overpressure alone if the durations are long. Clemenson, Von Gierke, and the Lovelace team all had observed that it is natural to relate long and short to response time or natural period of the mammalian thorax since the lungs are the principle target organs. In developing his Partial Impulse Criteria, Richmond relied on information pertaining to the determination of the medium lethal pressure required for 50 percent mortality of dogs and goats for fast rising shock waves. The test data, displayed earlier in Figure 1, and reproduced in Figure 6, were obtained from 204 dogs having body weights ranging from 11.4 to 25.4 kg, with a mean of 16.5 kg, and 115 goats with body weights ranging from 16.1 to 29.5 kg, and with a mean of 22.2 kg. Durations ranged from a maximum of 400 msec to a minimum of 1.5 msec. Probit analysis was used to obtain the LD_{50} graphs shown in this figure. The dashed lines shown are iso-impulse lines for reflected pressure, computed from measured peak pressures and duration. By repeated trials, a scaled time, t_o , was found which resulted in a near constant, scaled partial impulse, found by integrating the reflected pressure over the partial duration interval (t, t_o).

As shown in Figure 7, the value of the scaled partial impulse for these species was 207 kPa-msec/kg^{1/3} (30 psi-msec/kg^{1/3}).

Note in this figure that atmospheric pressure can be adjusted since the atmospheric pressure at the Albuquerque, New Mexico facility where the tests were conducted is approximately 83 kPa (12 psi) rather than the usually assumed 101 kPa (14.7 psi). Because it is used to compute critical partial impulse, the time t_o is referred to as the critical partial impulse time or characteristic response time.

From these data, Lovelace reported that for 16.5-kg dogs, an impulse of 526 kPa-msec (76.4 psi-msec) delivered over 1.53 ms corresponds to the medium lethal dose. For 22.2-kg goats, the values were 580 kPa-msec (84.2 psi-msec) applied over the initial 1.69 msec of the pulse. Comparable figures for a 70-kg mammal were 855 kPa-msec (124 psi-msec) delivered during the first 2.47 msec portion of the curve. Thus, the first estimate of a 70-kg man's characteristics response time was 2.47 msec at an ambient pressure of 83 kPa (12 psi).

The technique just described and displayed on this figure is known as Richmond's Partial Impulse Criteria for LD_{50} Blast Waves (6) which he augments with his One-Quarter Rule for estimating threshold lung damage. The computation of characteristic response time was derived from scaling equations established in developing a Lovelace Lung Model (8).

Note in Line 2 of Figure 7 that the empirically derived value of the constant, K_o , was 0.6 for the Albuquerque test facility where ambient pressure is 83 kPa (12.0 psi). In 1968, Bowen (2) determined, by a trial and error, a characteristic response time of 2.23 ms for a 70-kg man in a 101 kPa (14.7 psi) environment. Based upon this finding, Richmond's Partial Impulse Criteria reveals that if a 944 kPa-msec (137 psi-msec) effective pulse is delivered in a

characteristic response time of 2.23 msec against a 70-kg man in a 101 kPa (14.7 psi) ambient pressure, the man has a 50 percent chance of mortality. Under the one quarter empirically derived rule, the criterion for threshold lung damage is then a 234 kPa-msec (34 psi-ms) impulse delivered in 2.23 ms.

The Swedish scientists, Ciemedson and Jonsson, have also recently completed investigations directed towards estimating the risk of personnel to blast (8). Based upon experiments with rabbits and mathematical modeling and scaling, they determined the risks to gun crewmen serving recoilless rifles from within bunkers. In regards to the threshold lung injury to man, the authors felt that the analysis of the effects of complex pressure patterns developed by the rapid fire of a weapon in an inclosed room should be treated in terms of criteria for classical waves of long duration, because the waves in the bunker were too complicated to model. They applied Richmond's Partial Impulse Criteria to their experimental data to determine the critical impulse applicable to 50 percent lung damage, differing only in their threshold assumption for risk, i.e., one fifth rather than one quarter. They concluded that the transmittal of an impulse of 822 kPa-msec (128 psi-msec) at 101 kPa (14.7 psi) ambient during a critical duration time of 2.47 msec or less gives man a 50 percent chance of survival.

Let me now discuss some aspects of the damage risk criteria which I earlier arbitrarily defined as a lower bound for incapacitation criteria. The US Army Human Engineering Laboratory was in the forefront of identifying the need for the development of a hearing damage risk criteria as basic to the entire impulse noise problem (9). They pointed out three ways to attenuate impulse noise for Army weapons; (1) reduce the pressure at its source, (2) separate the operator from the impulse noise source by either distance or a barrier, or (3) develop ear protective devices. A suitable damage-risk criterion was therefore needed to measure the effectiveness of the options. The first major effort in establishing a suitable damage-risk criterion was undertaken by a working group established in 1965 in response to a request by the US Surgeon General to specify damage-risk criteria to sound. This working group, referred to in the literature as CHABA-46, an acronym for the Committee on Hearing, Bioacoustics, and Biomechanics of the National Research Council, analyzed the then available research data and concluded that a set of rules could be prescribed with respect to damage-risk criteria and contours for steady sound, but further research data had to be acquired

with respect to the physical parameters of impulse noise before criteria and contours could be specified for this hazard to hearing (10).

The problem of establishing damage risk criteria for impulse noise specifically was first addressed in 1967 in a joint effort involving researchers from the United Kingdom and the United States (11). The results of this study included establishment of definitions for the principal parameters of a single impulse noise, defined as follows and described in Figure 8.

Peak Pressure Level - the highest pressure level achieved, expressed in DB (reference 0.0002 dynes/cm²) or in psi (pressure difference AB in Figure 8a).

Rise Time - the time taken for the single pressure fluctuation that forms the initial or principle positive peak to increase from ambient to the peak time level, usually less than 1 msec (time difference AB in Figure 8a).

Pressure A-duration - time required for the initial or principle pressure wave to rise to its positive peak and return momentarily to ambient (time difference AC in Figure 8a).

Pressure Envelope B-duration - total time that the envelope of the positive and negative pressure fluctuation is within 20 db of the peak pressure level (time difference AD, and EF when a reflection is present in Figure 8b).

Based upon these definitions, the combined US-UK team developed ear damage-risk criteria at the 75th percentile for pulses arriving at the ear at grazing incidence, and for repetitive rates in the order of 6-30 impulses per minute with the total number

of impulses limited to 100 per exposures. These criteria were updated in 1968 by a CHABA Working Group 57, wherein the pulses were assumed to reach the ear at normal incidence for 95th percentile protection. (12) The CHABA-57 criteria adjusted for a single impulse are shown in Figure 9, with the B-duration curve plotted in Figure 5 as representation of the lower bound for incapacitation considerations as eluded to earlier in the discussion.

Health standards posed to military personnel in the vicinity of weapons are dictated by regulations called military standards (MIL STD's). A MIL STD is neither a hearing damage risk criterion or a hearing conservation criterion. It is a design standard, evolved from consideration of hearing aural detection, state-of-the-art noise reduction and federal and state legislation, and is intended to cover typical operational conditions. This standard is applicable to the design of all new military systems, sub-systems, equipment and facilities which limit acoustic noises to personnel areas. The MIL STD shown in Figure 10, which is a derivative of the damage-risk

criterion proposed by CHABA-57, modifies the basic impulse damage-risk criterion for B-duration taking into account variations in the number of exposures (1000, 100, and 5) and the attenuation of impulses by ear plugs and/or muffs. These are the reasons for the multiple limits (x, y, and z) and the basic rationale for the spacing. In a recent analysis of this subject, Rudsky (13) questioned the credibility of the MIL STD in that the design constraints upon which the standards are based have not been supported by adequate biological data. He felt that satisfying the MIL STD requires a tradeoff in some facets of system performance but the stringent requirements placed on today's weapon developments allow less and less flexibility to alter the various parameters. The z curve for 5 exposures per day with ear plugs or muffs are plotted in Figure 5 along with the LD, lethality, the threshold lung and the CHABA-57 B-duration curves.

I would like now to consider one more damage mechanism, i.e., that for eardrum rupture. One of the most prominent researchers in this area was Hirsch who surveyed data from the pre-1950 periods and found that Zalewski, Mackle, Pearlman, Shilling, and Corey had made estimates of threshold ear damage ranging from 27 kPa (3.9 psi) to 54 kPa (7.9 psi) with an average of 34 kPa (5 psi), (3). Further examination of accident data on ear drum rupture collected by other experimenters prompted Hirsch to offer the cumulative frequency distribution for eardrum damage plotted in Figure 11. Also plotted in Figure 11 are shock tube data collected on dogs by the Lovelace Institute (14). Interpolation of the data from these two sources results in an estimate of 103 kPa (15 psi) for a 50 percent eardrum rupture, and 34 kPa (5 psi) for threshold eardrum rupture.

A 50 percent eardrum rupture curve has

been plotted in Figure 5. This curve is original to my paper and needs explanation because we found no experimental data in the literature from which one could readily relate eardrum rupture to the duration of the shock wave.

Researchers for the most part have equated eardrum rupture to peak overpressures, but not duration. Lovelace, in their study on the Relationship Between Eardrum Failure and Blast-Induced Pressure Variations (15), did comment on the effects of some of the components in the blast wave to eardrum rupture but offered no criterion or methods for relating pressure to duration. Moreover, the results of their goat and dogs experiments indicated that while the eardrum was more sensitive to fast-rising than slow-rising blast waves, the data were insufficient to prove the point or state what might be expected for blast waves with both fast and slow components having different magnitude and time constants.

The 50 percent eardrum rupture curve shown in this figure was generated by drawing a curve parallel to the threshold lung curve through the 103 kPa (15 psi) value at a positive duration of 2 msec. Although the 2 msec time is assumed to represent a fast rising short duration blast environment, such as that in the vicinity of gun or howitzer crew stations or that near or medium distance from small chemical detonations or bomb bursts, its choice and the selection of the shape of the curve were both subjective and somewhat arbitrary on my part. For that matter, the curve could have been a straight line through the 103 kPa (15 psi) value, parallel to the abscissa, although it seems evident that a threshold curve for eardrum damage should vary significantly with overpressure, at least initially, and insignificantly with duration, as with both the lethality and threshold lung curves. Whatever the shape of the curve, it is my judgement that a 50 percent threshold eardrum damage curve represents a threshold for incapacitation. I would also be remiss if I did not also point out that the selection of eardrum damage is not universally accepted as a measure of severity of a blast injury. For example, Lovelace (15), did not consider failure of the eardrum (or lack of it) as a reliable clinical sign for judging the severity of a blast injury because of the wide tolerance limits of the tympanic membrane. This stemmed from their findings with animals that the drum often remains intact when exposure pressures produce serious lung injury, but may also rupture at pressures well below hazardous ones. Josephson, the US Navy's wound ballistic expert, also felt that neither ear injury nor eye injury alone

would necessarily incapacitate a military person, but the combination of eye, ear, and lung injury would incapacitate a combat soldier (16). He further assumes that incapacitation starts immediately after exposure and lasts for some indefinite period of time, but that this time is long enough to make soldiers ineffective as a combatant in the engagement in which the injury was received.

Loss of hearing, however is a form of incapacitation in that it can render a soldier combat ineffective as regards to his capability to perform certain tasks. In this context, I therefore offer the 50 percent eardrum damage curve as a threshold for incapacitation, recognizing that although eardrum rupture may be accompanied by pain and loss of hearing, there is little evidence in the literature to support that this form of injury results in an incapacitated casualty. It should be noted that the threshold eardrum damage curve is applicable to unprotected ears. Higher limits would apply to infantry soldiers wearing helmets or crew personnel using headgear equipped with earphones or other communication devices.

I also suggest that the LD₁ lethality curve is in itself too severe a measure of incapacitation for military personnel and feel that its application to vulnerability studies of the individual infantry soldier, and crew personnel in various air and ground vehicles, underestimates casualty production as well as the effectiveness of the blast producing weapons being evaluated. I further recommend that the threshold lung damage curve be substituted as a more conservative measure and that it be used as an upper bound for incapacitation, that is, that it be considered to represent the 99 percent incapacitation level. My recommendation of the more conservative threshold lung injury as a measure of maximum incapacitation is again subjective. There is, unfortunately, nothing in the literature to either support or contradict this assumption because previous researchers did not evaluate the degradation in performance of either civilians or soldiers performing tasks, given a blast induced injury, i.e., incapacitation has to date not been quantified. Several wound ballisticians with whom I've discussed the preceding have indicated that the threshold lung curve might be too conservative a measure of complete incapacitation. If in the future a more stringent measure of total incapacitation were found to prevail, I would then suggest that the LD₁ lethality curve be used to represent a threshold for complete incapacitation and that the threshold lung damage curve be used to indicate a 50% incapacitation threshold.

These, combined with the zero incapacitation associated with the threshold eardrum damage curve, would offer the vulnerability analyst a discreet numerical scheme for computing the vulnerability of personnel targets to the blast threats.

Finally, Figure 12 presents the LD₁ lethality curve, the 95-percent eardrum protective curve, and the newly defined threshold and 99 incapacitation curves overlaid with three sets of blast measures from three different blast sources. The objective of this very busy graph is to give perspective

to my recommended changes and to compare incapacitation estimates using the old and new blast criteria. Shown are blast measurements taken in the vicinity of a 105mm Howitzer (17), a grid displaying blast pressures for a range of bomb sizes (18), and blast measures taken inside an armored personnel carrier for a series of shaped charge high explosive antitank (HEAT) rounds with cone diameters ranging from 84 to 250mm, all of which have perforated the hull (19). The two data points for 20 and 50g TNT charges, identified with circle symbols, were also measured within the APC and are used as reference measures for comparing the HEAT data. Using the LD₁ criteria for lethality, incapacitation would have been assigned only to those personnel ranging from within 6.0m of the blast source for a 113 kg bomb to within 18.0m for a 907 kg bomb. Using the 99 percent incapacitation criteria the incapacitation zone is increased to about 11.0m for a 113 kg bomb and about 29.0m for a 907 kg bomb. For threshold incapacitation, all personnel within 46.0m of a 907 kg bomb are judged to be incapacitated to some degree and those within about an 18.0m radius of the 113 kg detonation are incapa-

citated. By the same analogy, no personnel in the APC were considered incapacitated based upon the LD₁ lethality measure. However, by implementing the 99 percent incapacitation curve, personnel in the APC penetrated by the larger diameter HEAT rounds would be considered completely ineffective or totally incapacitated by blast, and the medium to larger HEAT rounds would incapacitate other personnel to some lesser but as yet undefined level. The smaller HEAT rounds would cause no incapacitation of the APC crew/passengers, but ear plugs/muffs would be required in accordance with the Army's MIL STD. Crew personnel serving the 105mm Howitzer would not be incapacitated under any of the criteria, except that ear protection would be required within 3.0m of the muzzle.

The major conclusion from this somewhat simplistic analogy suggests, at least to me, that equating casualty production or onset of incapacitation to the LD₁ lethality curve is not realistic. I have offered a more conservative measure for defining complete incapacitation, which in the context of the blast weapon effects data shown on the graph, does seem more reasonable. Obviously, the effects of replacing the present criteria has to be compared and quantified in terms of changes in vulnerability calculations for infantry and crew personnel subjected to blast-induced weapon threats. It is also apparent that additional biological data and/or further extension and modeling of the existing data bases are necessary. The former will be accomplished as an extension of the work described within this paper. The latter I leave to those experts, scientists, and researchers whose excellent experiments and research made this paper possible and upon whom we, the vulnerability community, must rely for a more fundamental assessment of the effects of blast on our military forces in the modern battlefield.

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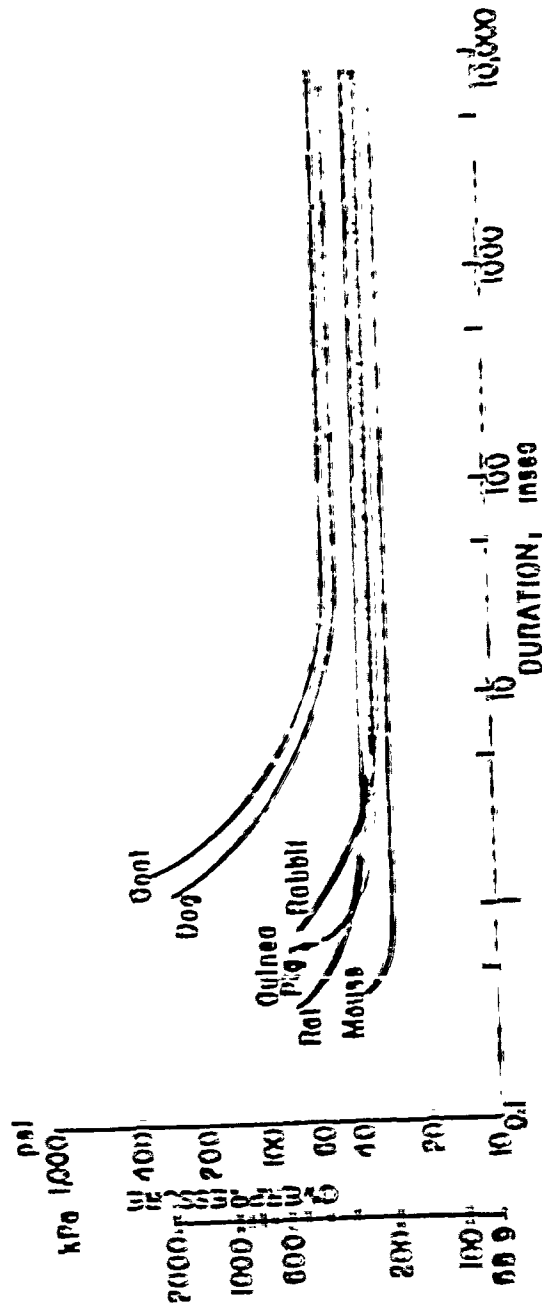


Figure 1. Fifty-percent constant lethality curves for six species relating pressure to duration .

REGRESSION EQUATION

$$\log(LD_{50}) = 1.3673 + 0.00930 \log(DW)$$

Where (LD₅₀) = Pressure required for 50% mortality, psi.

DW = Average body weight of the group, grams.

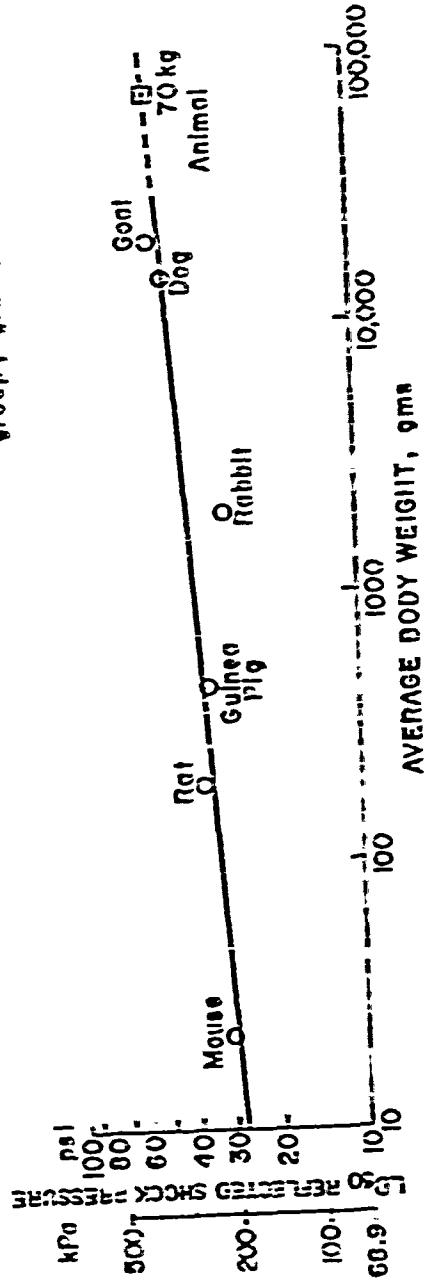


Figure 2. Relation between body weight and fast-rising overpressures of 400 milliseconds duration needed to produce 50 percent lethality.

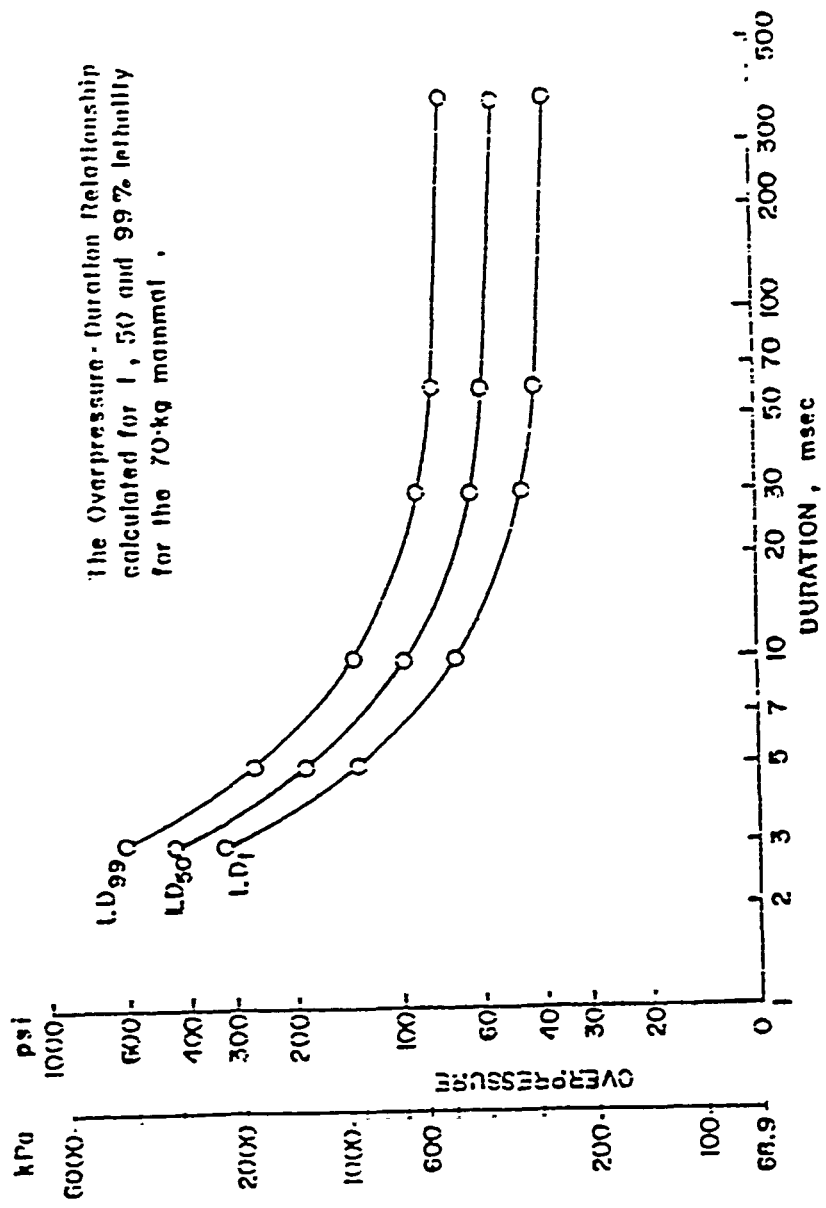


Figure 3. Constant lethality curves predicted for animals in the weight range of man from extrapolating interspecies-response data .

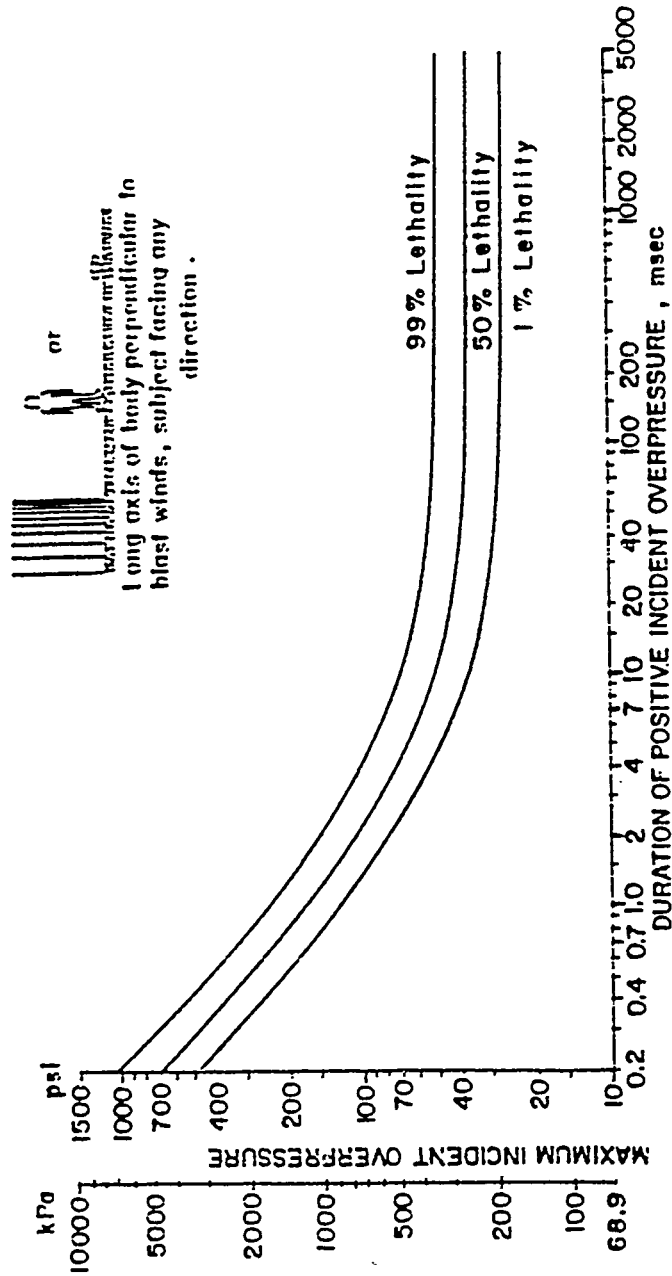


Figure 4. Lethality curves predicted for 70-kg man applicable to free-stream situations where the long axis of the body is perpendicular to the direction of propagation of the shocked blast wave .

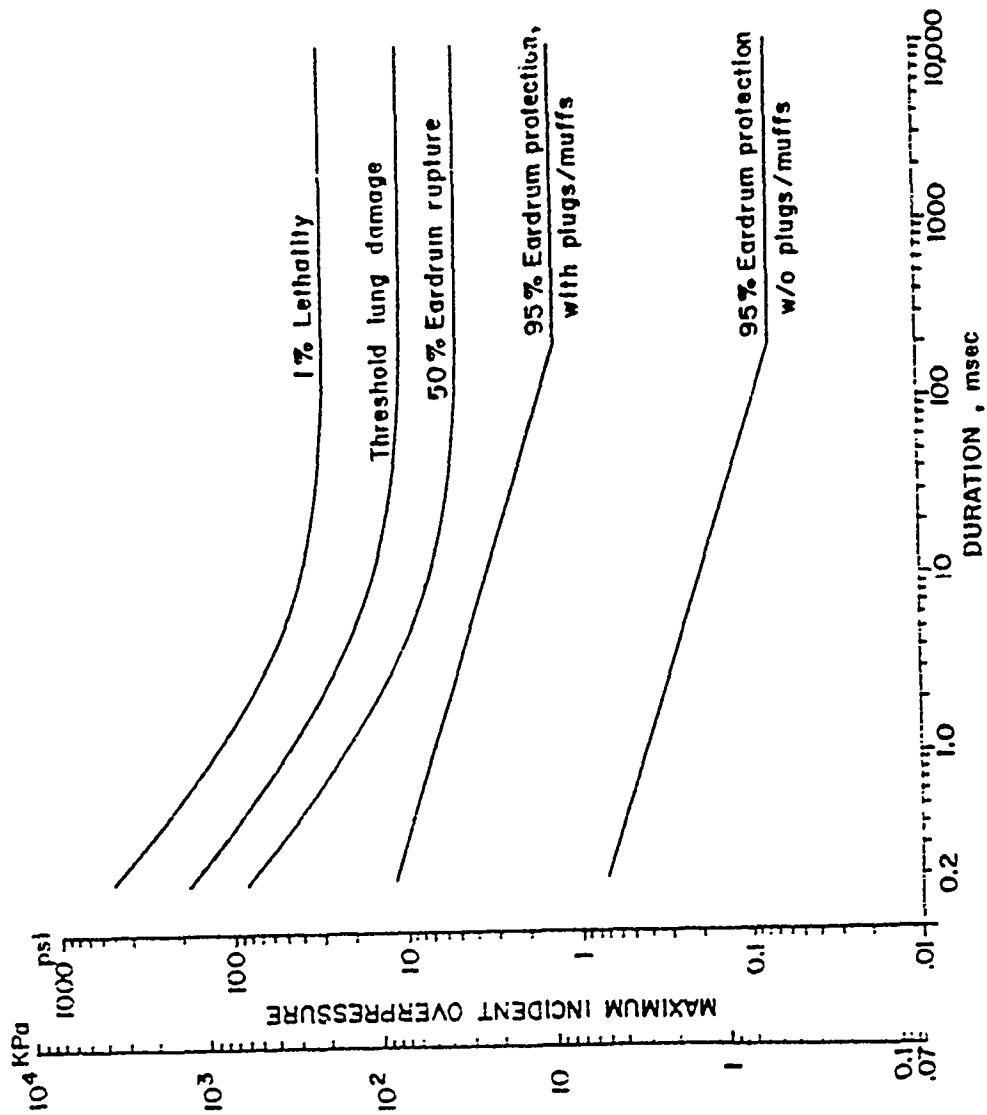


Figure 5 . Lethality and damage/ Injury curves predicted for a 70-kg man applicable to the free stream situation .

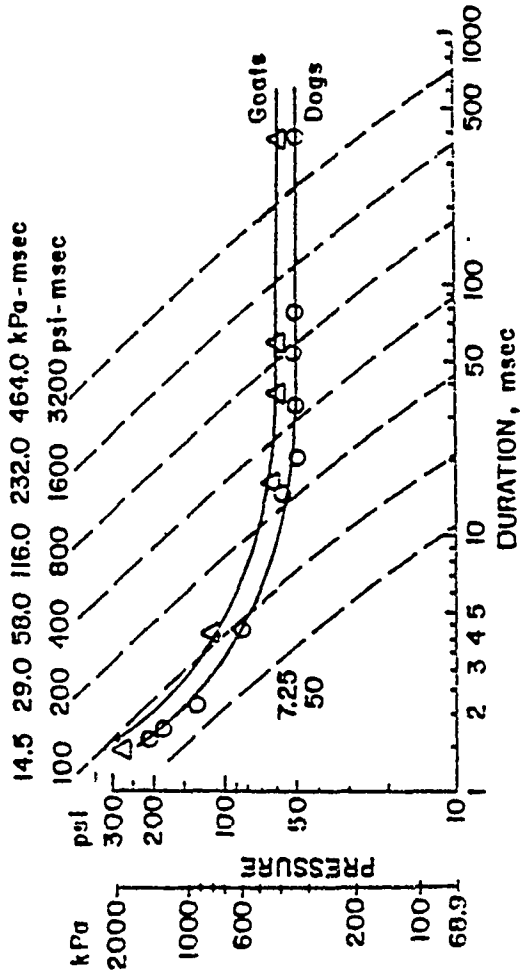


Figure 6. Pressure-duration relationship and 50-per cent lethality for dogs and goats .

I_0 is overpressure impulse in psi-msec from arrival of blast wave to time t_0 .
 t_0 is $0.6m^{1/3}(12/P_0)^{1/2}$ msec.
 m is body mass of the animal in kg. ○ 16.5 kg Dogs
 P_0 is ambient pressure in psi and □ 22.2 kg Goats
 t_d is duration of the blast wave in msec.

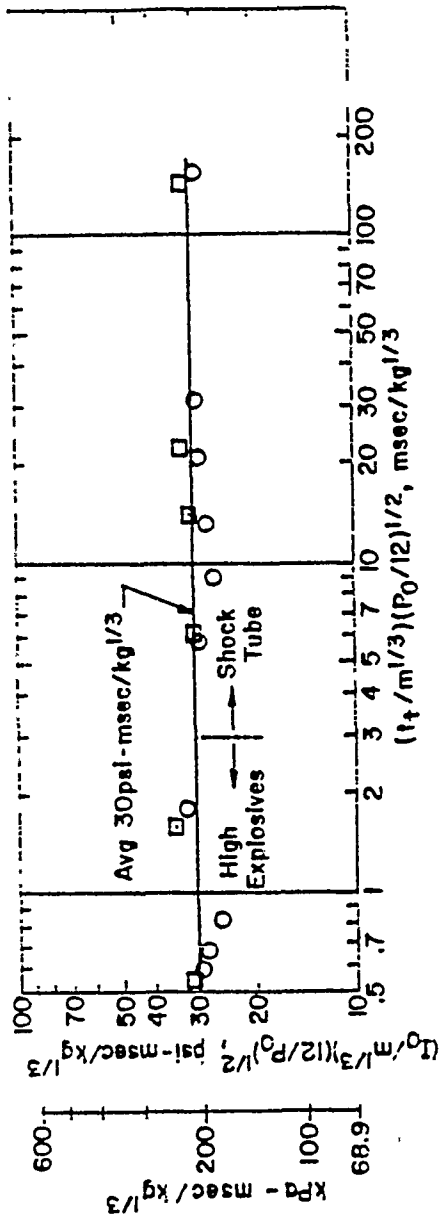
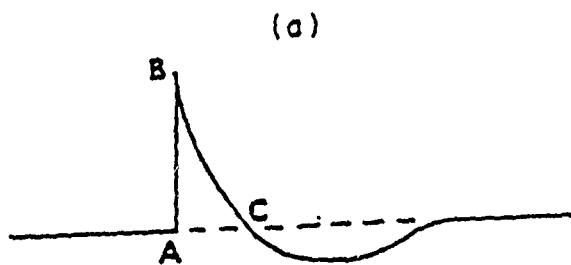
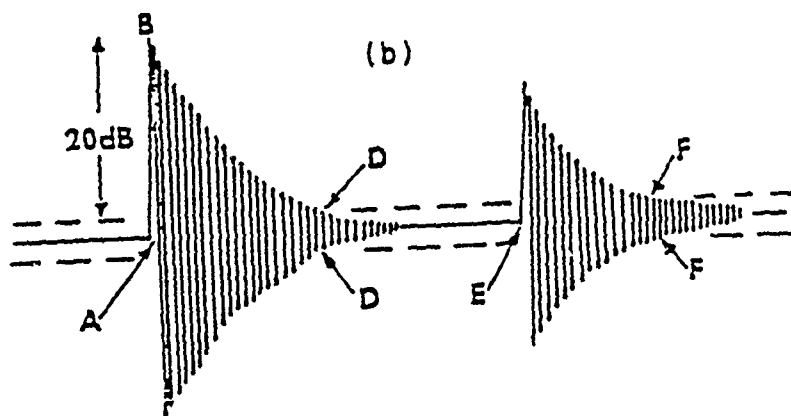


Figure 7. Partial impulse of reflected blast waves resulting in 50-per cent lethality .



Peak level : pressure difference AB
 Rise time : time difference AB
 A-Duration : time difference AC



B-Duration : time difference AD
 (+EF when a reflection is present).

Figure 8. Idealized oscilloscopic waveforms of impulse noises .

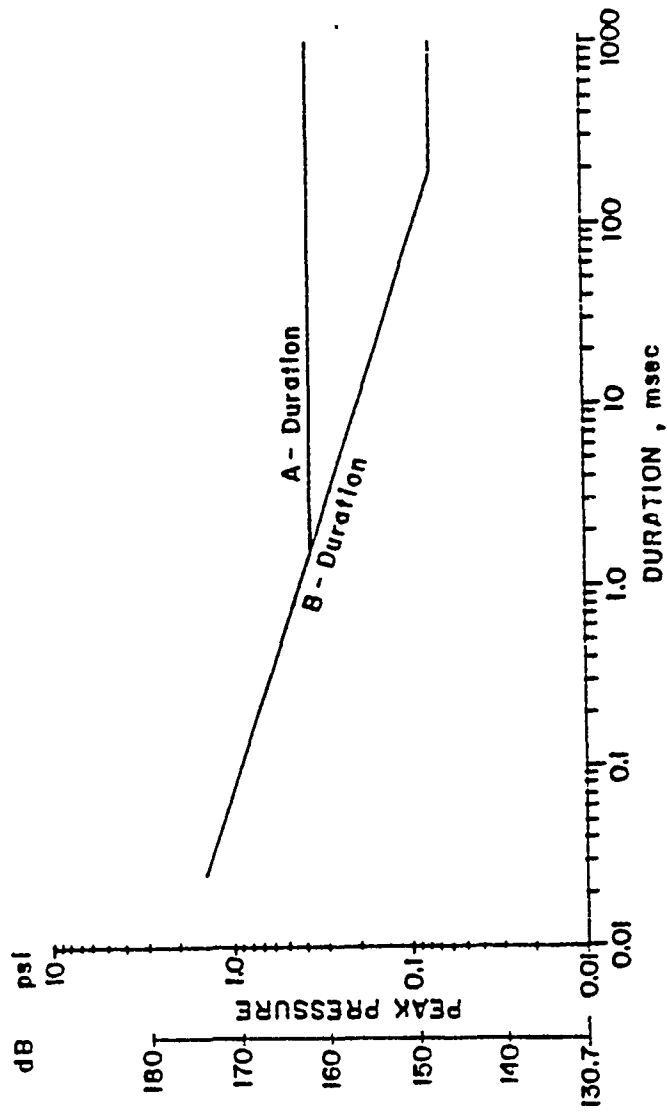


Figure 9. Basic impulse design risk criteria for a single pulse .
 (Chaba working group 57)

Max. Expected Number of Exposures in a Single Day	Impulse noise limit		
	No Protection	Either Plugs or Muffs	Both Plugs and Muffs
1000	W	X	Y
100	W	Y	Z
5	W	Z	**

** Higher than Curve Z are not permitted.

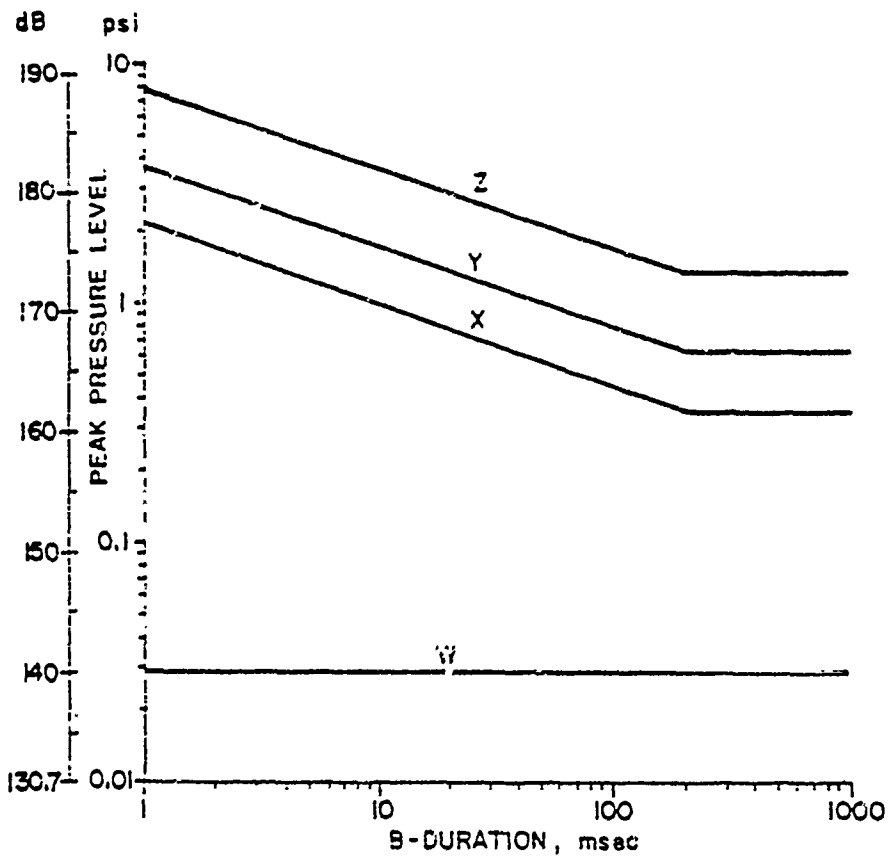


Figure 10. Military Standard (MIL STD) impulse noise limit selection criteria .

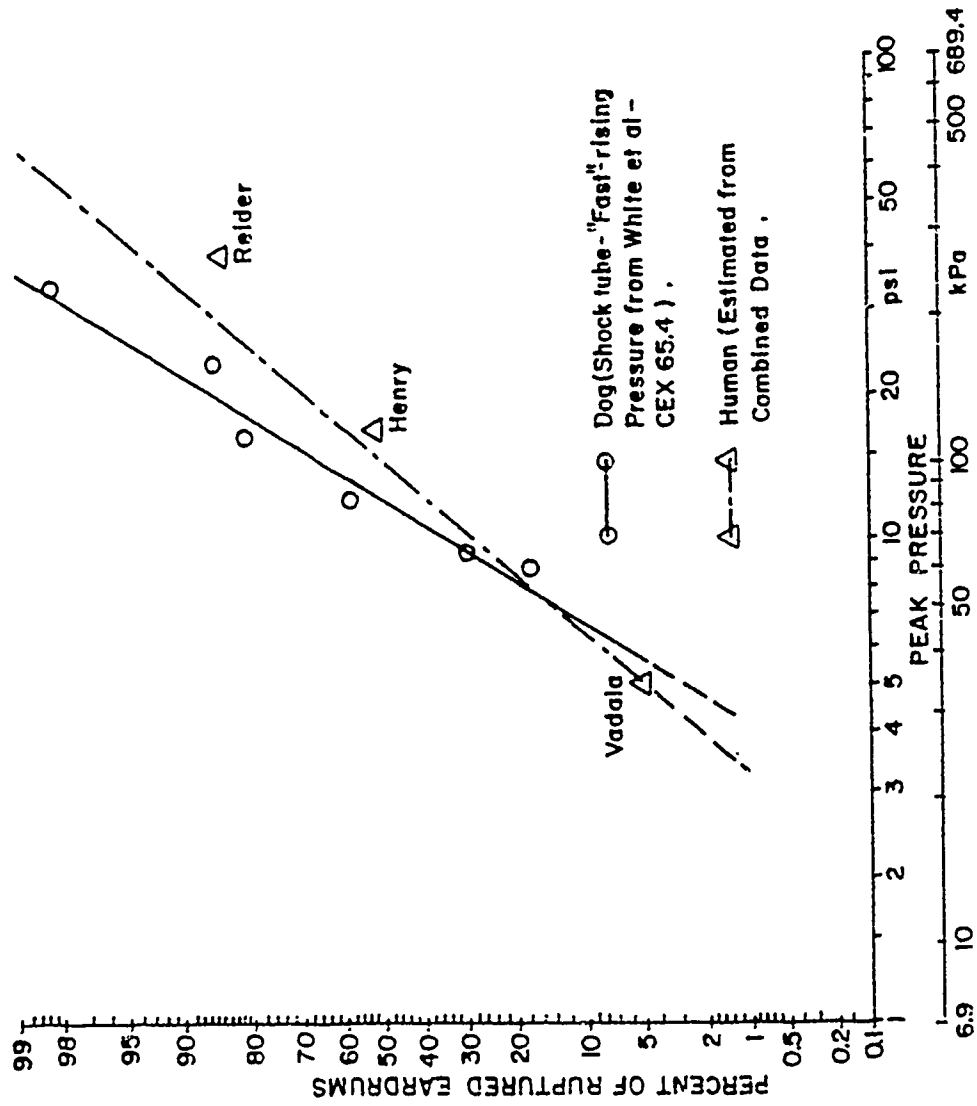


Figure II. Tolerance of eardrums to "fast"-rising overpressures .

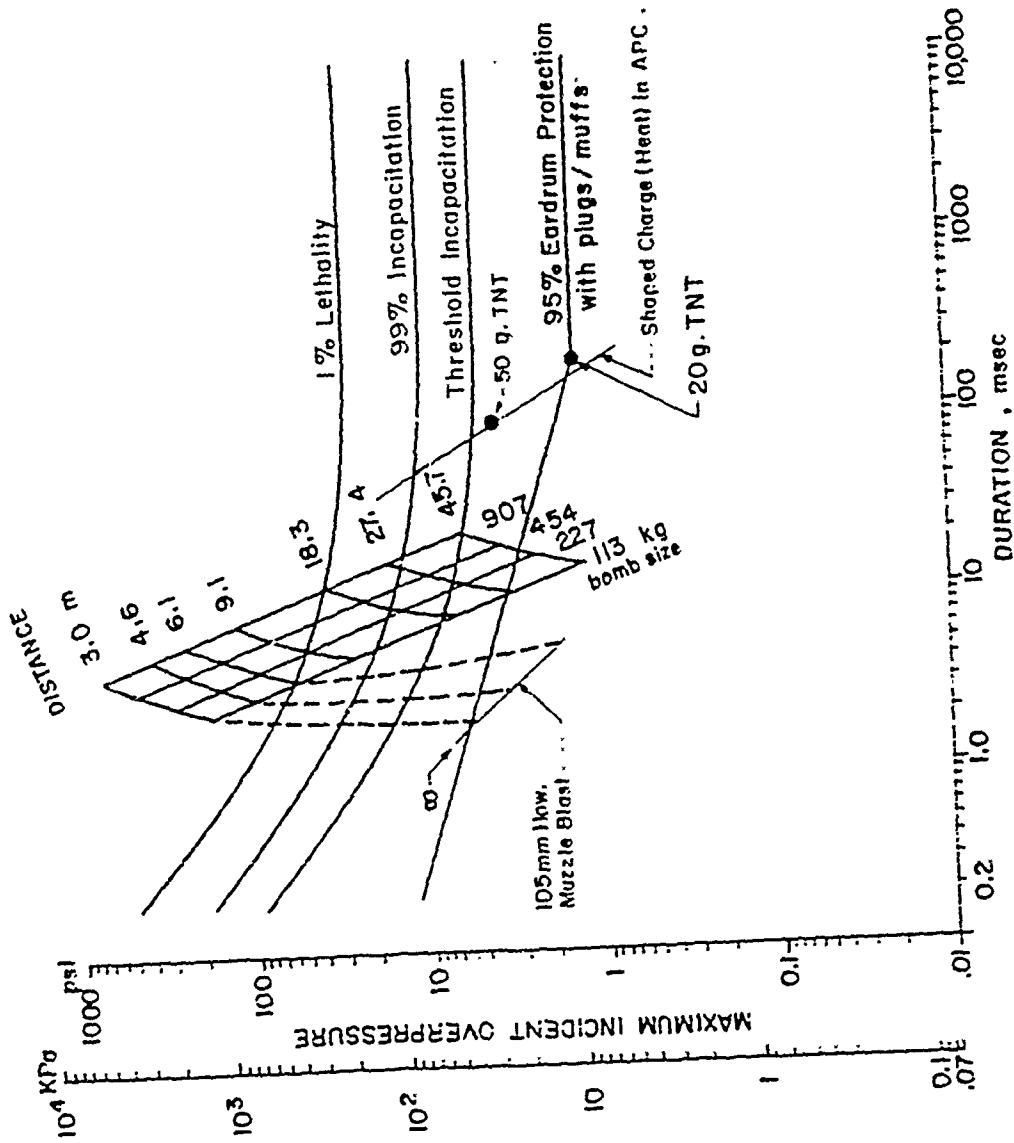


Figure 12. Lethality and Incapacitation curves predicted for a 70-kg man applicable to the free stream situation.