

**The Case for Transient Air Embolism from Lung Injury as a
Mechanism for Blast-Related Brain Injury and Its Implications**
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Air embolism (AE) has long been regarded as the primary cause of death for persons who die within a short time following exposure to blast overpressure (primary blast injury). However, the role of air embolism in those persons who survive has received little attention. A review of the literature with an eye towards looking for signs that transient, possibly sub-clinical, air embolism plays a role in the development of mTBI/PTSD from primary blast has yielded a strong indication that its role may be substantial. Initial attempts to treat patients with blast-related mTBI/PTSD with hyperbaric oxygen therapy (HBOT) have produced significant remission of symptoms in all cases and complete remission in some (Harch, Hoffman, et al 2009; Harch, Fogarty, et al 2009; Wright 2009). Those results are consistent with air embolism as a causative factor. It is recommended that the role of air embolism in the development of mTBI/PTSD and other effects resulting from primary blast injury be researched as quickly as reasonably possible.

This paper explores the following three hypotheses:

1. There is a significant range of exposure to levels of air blast between “no effects” and “lethal” where air embolism subsequent to the air blast is not only possible, but probable.
2. The resulting, often sub-clinical, arterial bubbles are not harmless.
3. The nature of the conflicts in Iraq and Afghanistan is such that conditions conducive to the production of sub-clinical, arterial bubbles occur frequently.

The Defense Atomic Support Agency (Bowen 1968) published a family of curves which are still widely cited for predicting survival for a 70-Kg man in various blast overpressure situations. Figure 1 shows the overpressures for a 2.5 ms duration blast overpressure wave that are required to produce lung damage when the person is in an open area and when the person is close to a reflective surface. The reflective surface location is much more dangerous because the gases pile up, creating pressures up to several times higher than the incident wave. Also shown are the pressures calculated to have occurred in a 1988 bus bombing (Katz 1989). The overpressure in the bus was calculated to last 2 to 3 ms and falls in the range of pressures that Bowen’s curves predict would cause serious lung injury. Most of the people on the bus survived. However, 38% of the survivors required treatment for blast lung. Any exposure to blast overpressures above the “lung damage threshold” can be expected to create the possibility of the lungs leaking air into the arterial circulation. Indeed, Mason (1971) outfitted a dog with a Doppler bubble detector on the carotid artery, exposed the dog to an LD50 air blast, and then observed families of echoes (presumed to be air bubbles) going up the carotid in synchronization with the dog’s respiration for about 30 minutes post-blast. This is consistent, based on several decades of experience, with the duration of arterial bubbles typically observed in diving air embolism cases. In Mason’s dog, carotid blood flow was also observed to drop temporarily to near zero following each group of echoes. The dog struggled for the first day, then appeared to recover fully. As a result, Mason reported that his “silent” bubbles did not produce lasting physiological sequelae. However, the dog was sacrificed at 5 days post-injury and there are indications that blast-related brain injury

may require 2 to 3 weeks to become manifest (Wright 2009). Nonetheless, the notion that “silent bubbles” are not harmful seems to have taken hold. As far as we have been able to determine, the literature regarding primary blast injury has been silent on the topic of arterial microbubbles ever since.

The dichotomy between the air blast literature and diving literature regarding the clinical significance of AE is also striking. In a Review Article in the *New England Journal of Medicine*, DePalma (2005) is silent on the risks of non-occlusive AE and doesn't even mention HBOT as a possible element in the treatment of blast lung. Richmond (1991) states that the presumed cause of death for persons dying quickly from primary blast is considered to be AE, but there are no air emboli found in those who survive. It is extraordinarily rare for the line between survival and non-survival to be so clearly delineated. Contrarily, in diving (sport, commercial and military), any indication of AE sufficient to produce clinical symptoms is considered a medical emergency requiring recompression and hyperbaric oxygen as quickly as possible.

That bubbles associated with non-occlusive air embolism, also referred to as microbubbles, are harmful has now been recognized in several other clinical situations. Microbubbles were first recognized as a medical hazard in open heart surgery several decades ago (Gallagher 1973; Rodigas 1982). Cardiopulmonary bypass machines are connected to the systemic circulation through the ascending aorta, carrying microbubbles mainly to the brain. The clinical consequences include major and minor neurologic injury and neurocognitive deterioration (Barak 2008). Microbubbles are also associated with diminished neurologic executive function in divers with decompression sickness (Cianci and Slade 2006). Microbubbles originate in the lines and tubing of hemodialysis machines and circulate in the blood stream until lodging in the capillary beds of various organs, mainly the lungs (Barak 2008). Cernak (2001) has also shown that LD50 air blasts (440 kPa e.g. 65 psi for 50 ms) to the thoracic region only with the head shielded consistently produce brain damage in mice.

The likelihood of lung injury as a significant component in the family of injuries produced by blast is strongly dependent on the nature the blast pressure profile (See Figures 2, 3 & 4).

- Blast overpressure profiles are considered “short” when the length of the pressure wave is less than the length of the thorax parallel to the direction of wave travel (White, 1971). For humans, we, therefore define (somewhat arbitrarily) blast waves as short when the length of the overpressure wave is less than about 1 foot. Thus, any blast wave lasting less than about 0.9 ms is considered “short”. Short blast waves produce moving chest wall loading, but do not completely encircle the chest, and relatively high pressures are required to produce lung injury.
- Blast overpressure profiles are considered “long” when the length of the pressure wave is greater than the length of the thorax parallel to the direction of wave travel. For humans, overpressure waves are considered “long” when the length of the overpressure wave is greater than about 2 feet (time > 1.8 ms). “Long” blast waves completely encircle the thorax and produce very rapid chest compression. (15 psi overpressure converts to about 1 ton per square foot). As the compression period gets longer, the pressures required to produce damage quickly become lower. “Long” blast waves are damaging to lungs at MUCH lower pressures than are “short” blast waves (See Figure 4). It is also noted that these pressures are substantially lower than are the blast pressure profiles being employed

in some current work looking at comparisons between direct blast injury to the brain and brain injury from collisions (Moore 2009).

The same munition that would produce a short duration blast in an open area can quite easily produce a long duration blast when detonated in a confined space due to the lower rate at which the blast gases can disperse. In the current wars in Iraq and Afghanistan, an unusually large fraction of operational blast exposures have occurred in confined spaces: city streets, building interiors, and the interiors of armored vehicles penetrated by ordnance. Consequently, we believe that the case for transient air embolism as a mechanism for the blast-related brain injuries seen in the current wars is far stronger than has been realized to date.

Hyperbaric oxygen therapy (HBOT) is considered the definite treatment for air embolism (Horrocks 2001). Experience is now showing that chronic mTBI/PTSD resulting from blast exposure responds well to HBOT, albeit under a much different therapy profile from acute embolism (Harch and Fogarty 2009; Wright 2009). That is not conclusive, but certainly supports the hypothesis of air embolism, sub-clinical or simply unrecognized, as a possible cause.

We make the following recommendations:

1. Try to confirm Mason experiment.
2. Make sure researchers understand the difference between “short” & “long” blast overpressures.
3. Establish “sentinel” signs for subtle AE (burst eardrums, mucosal edema, cognitive disruption lasting more than a few minutes, any loss of consciousness, etc.).
4. Consider a field trial where if air embolism is suspected, the person is referred for HBOT at treatment depths currently utilized for acute air embolism.

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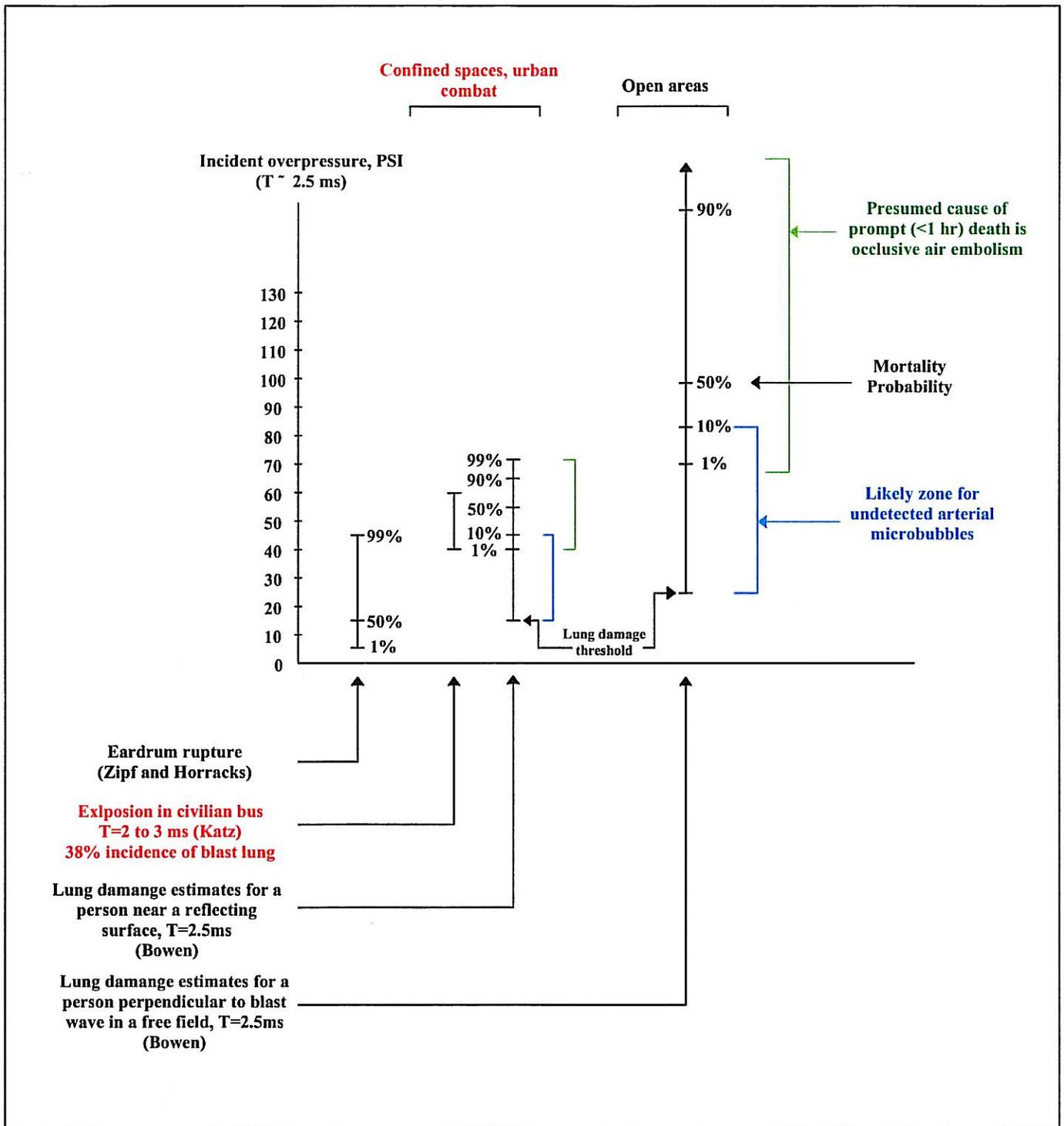


Figure 1:
Probable Effects of a 2.5ms Blast Overpressure Wave In
Confined Spaces and Open areas.

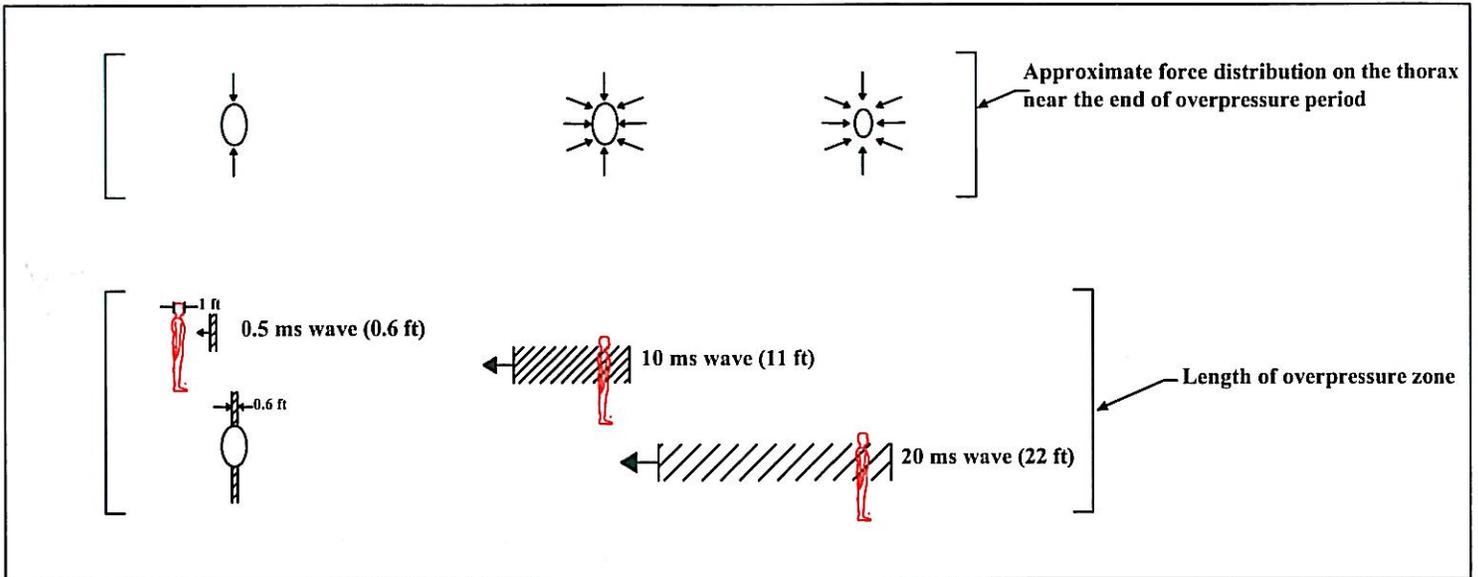


Figure 2:
Approximate Lengths of Overpressure Zone Relative to a Typical Human in a Free Field Situation. (Blast wave assumed to be travelling at the speed of sound, 1,100 ft/second)

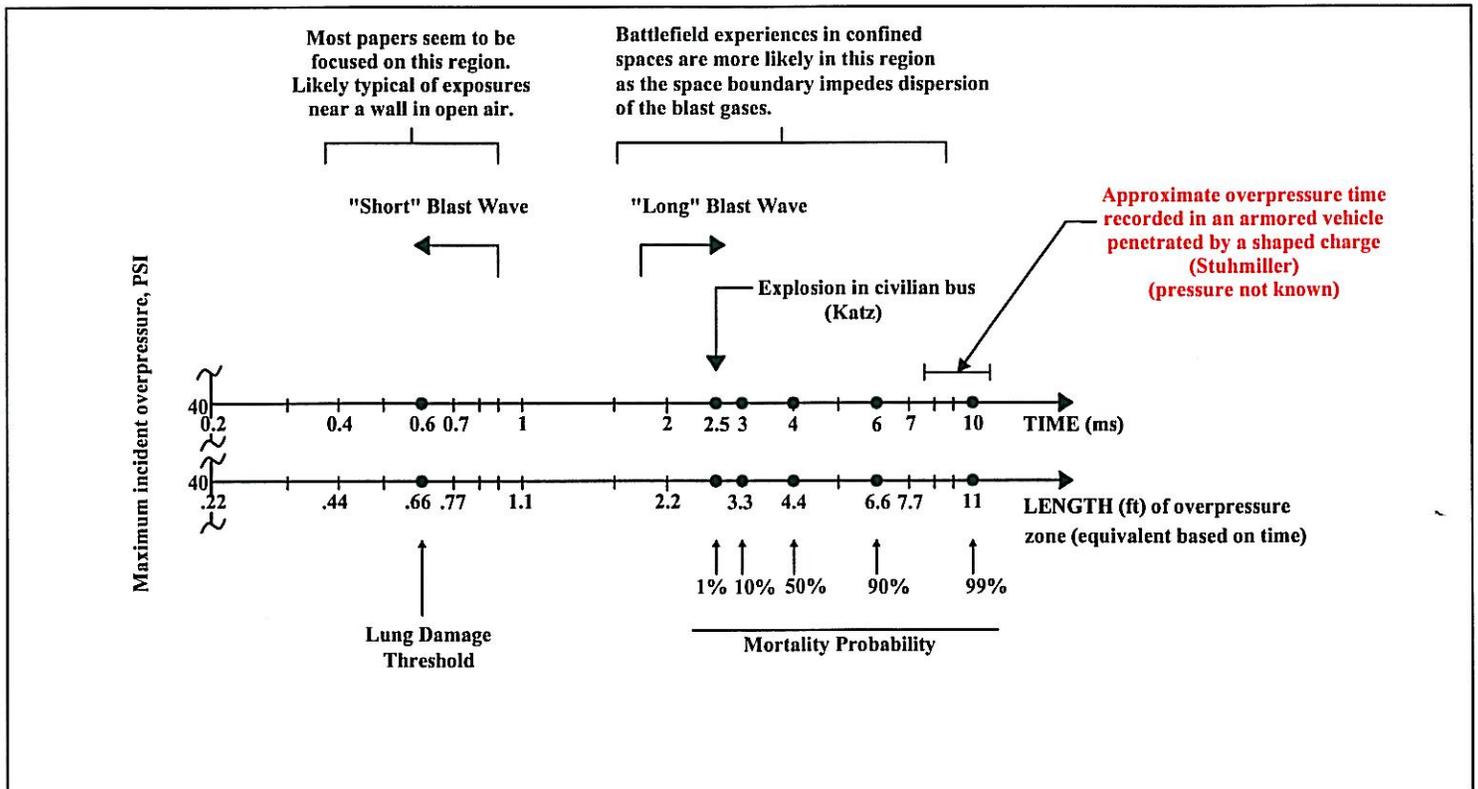


Figure 3:
Effect of Duration on Lethality of Blast Overpressure for Blast Wave Against a Reflective Surface for a 40 PSI Incident Pressure Wave. (Bowen)

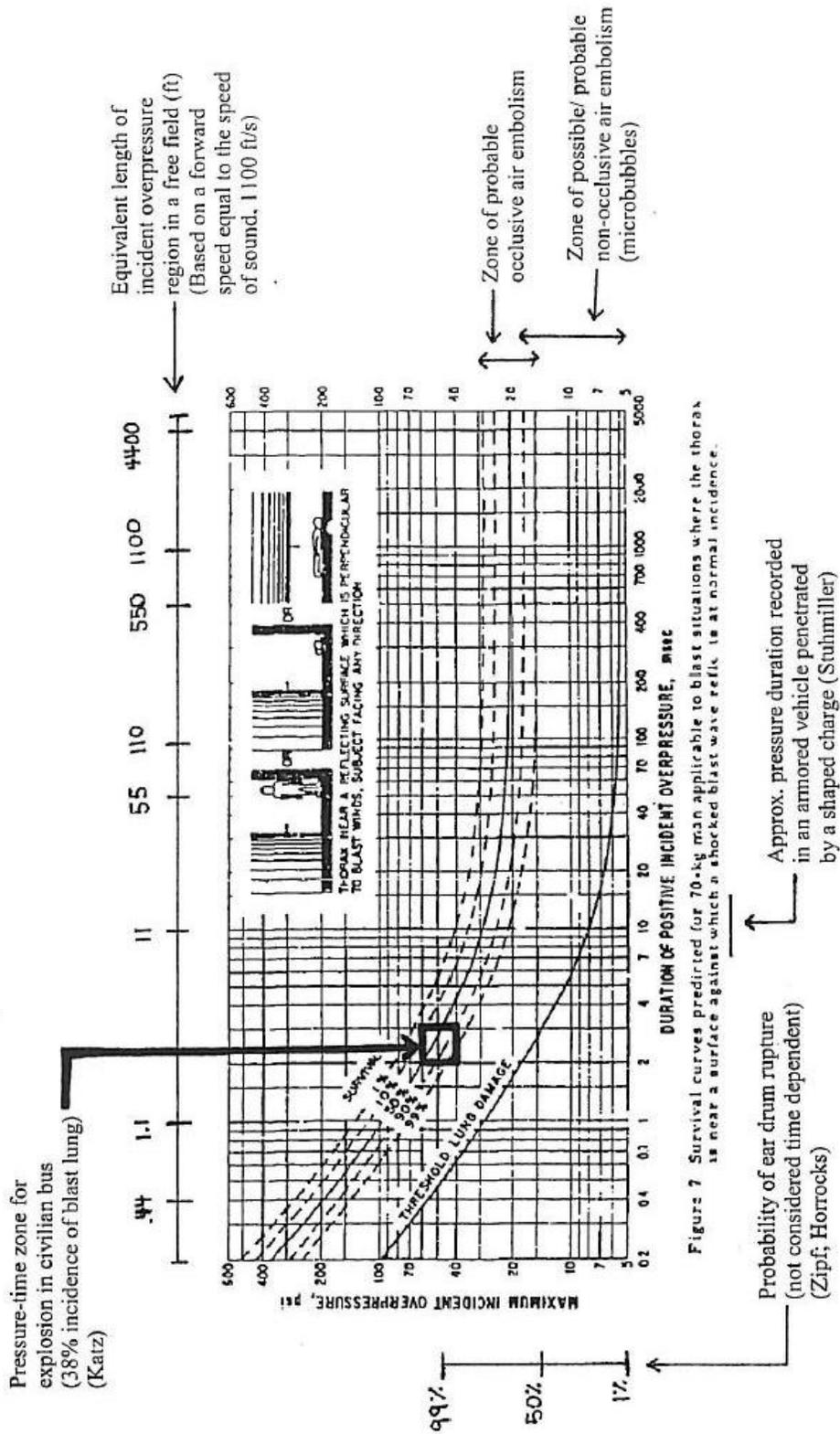


Figure 7 Survival curves predicted for 70-kg man applicable to blast situations where the thorax is near a surface against which a shocked blast wave reflects at normal incidence.

Figure 4: Effect of Duration and Incident Pressure on Lethality of Blast Overpressure against a Reflective Surface