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Assessing the Blunt Trauma Potential of Free Flying Projectiles for Development and Safety Certification of Non-Lethal Kinetic Energy Impactors.

by
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Introduction

The primary performance objective for non-lethal, anti-personnel kinetic energy impact projectiles is to reliably deter or incapacitate without causing injuries that require medical treatment beyond simple first aid or which leave permanent damage. Unlike many lethal weapons, non-lethal weapons must have upper limits (in order to remain non-lethal) for the impact parameters that govern the terminal effects. The development and safety certification of kinetic energy, non-lethal impactor weapons requires documented and testable criteria to ensure that lethality is limited. Presently there are no firmly established, universal design criteria to ensure that the terminal effect will be below the threshold of unacceptable injury against a specified range of the populace.

Predicting the terminal physiological effect of non-penetrating impact by a free flying projectile on a human being is difficult. Projectile properties, the location of impact, the angle of incidence, and the physiology of the target determine the overall effect. Different parts of the human body are notably more susceptible to blunt trauma injury than other parts, thus the same level of impact to different parts of the body can have a vastly different physiological effect. The only variables that can routinely be controlled in advance of impact on a human by a non-lethal projectile are the projectile properties. A better understanding of the combined influence of projectile properties on the mechanism of projectile/target interaction and on the resulting terminal effect will aid in designing projectiles that are effective yet have a low overall probability of being lethal or causing a serious and/or permanent injury. U.S. impact testing of animals by non-penetrating projectiles, and other blunt impact studies such as from the middle 1960's to the late 1980's resulted in insight into the mechanisms of injury by blunt trauma, and proposed criteria. Distribution restrictions on some of the past work made it unavailable to some projectile designers, and no study is known where all of the available data are compared. These data have now been reviewed and correlated in an attempt to derive more meaningful guidelines for the design and proof-testing of non-lethal projectiles. The present paper is based on releasable data from a report prepared by Battelle in 1997 for the Office of Special Technology.[1]

Some of the projectile properties that influence terminal effect are: terminal kinetic energy, terminal momentum, impact contact area, cross-sectional density, shape, and compliance. Many prior studies of the terminal effect of non-penetrating, non-lethal projectiles ignored these projectile properties except for kinetic energy. In two studies, the diameter and mass of the projectile and the mass of the target were used to fit predictive equations to the experimental results of animal testing. In other studies, impacts on animal surrogates (goats, swine, dogs, and baboons) were used to set kinetic

energy thresholds for lethality and severe injury. In other studies, measurements of impacts were correlated to the mechanism of injury and the extent of a blunt trauma injury; however, these did not relate measured impact parameters to projectile properties.

Key Prior Work

A number of studies of relevance are briefly cited to show the type of past work; these are not discussed in chronological order. Cooper et al. related chest deflection to lethality and developed models for predicting chest deflection as a function of projectile kinetic energy, diameter and target mass or chest thickness.[2] Viano, for General Motors, developed a model called the Viscous Model for predicting the likelihood of severe injury to soft tissue from compression due to blunt impact. The Viscous Model is based on the experimentally measured term CV_{max}, described later in the text.[3] Cuadros, working with Viano, has extended the CV_{max} studies to include impacts from commercially-available, non-lethal projectiles.[4] In conjunction with the Institute for Preventive Sports Medicine and St. Joseph Mercy Hospital, Ann Arbor Michigan, Viano had used the Viscous Model to assess the hazards of baseball impacts to the chest and to test the degree of protection offered by commercial chest protectors.[5] Several organizations including the Land Warfare Laboratories[6], the Lovelace Foundation, and the Swedish Research Institute of Defense[7] performed tests, where animal surrogates and/or human cadaver skulls were impacted in various locations by an assortment of blunt projectiles, including experimental and then commercially-available projectiles. The damage produced by the impacts was correlated to projectile energy, velocity and sometimes projectile diameter and target mass. The U.S. Army Biomedical Laboratory at the Edgewood Arsenal developed a four parameter model for predicting the lethality of a blunt impact to the thorax region and for predicting the likelihood of liver fracture due to an impact to the liver region.[8]

The Biomedical Laboratory performed a series of blunt impacts with free flying, non-compliant cylinders on goats. The group related the projectile properties of mass, velocity and diameter with the target mass to the experimentally observed terminal effect for an impact to the side of the thorax over the lungs and for an impact to the side of the thorax over the liver. They noted the terminal effects of each test on a scatter plot of ln WD versus ln MV^2, (where M is the mass of the projectile in g, V is the velocity in m/sec., W is the target mass in kg., and D is the diameter of the projectile in cm). It was noticed that the impacts with similar terminal effect were grouped together and that by drawing two parallel, discriminate lines, these terminal effects could be separated into three distinct groups. These were then defined using the parameter MV^2/WD.[8]

The Edgewood Arsenal work also took data, from several sources, for blunt impacts to other surrogate animals impacted with a variety of projectiles. These data were plotted as ln WD versus ln MV^2, then overlaid with the discriminate lines discovered from the goat testing. It was found that the model agreed well with the independent test data, if certain eccentricities in some of the data points were considered.[8] Edgewood Arsenal also fit a probability function to the data plotted as ln (WD) vs. ln (MV^2), for non-lethal and lethal impacts to the thorax.[8] The probability function is used to predict percent chance of death due to an impact to the thorax based on the parameter MV^2/WD. For example, an impact in the lung area with an $MV^2/WD = 1000$ has a predicted probability of lethality of only 1%, however, an identical impact over the liver area is in the zone were 50% percent of impacts caused liver fracture in the tested goats.

The previously mentioned Viscous Model is based on studies of steering wheel compression of the chest of unembalmed cadavers in simulated automobile accidents. Viano noted that damage to the heart was a function of two parameters: the percent compression that occurred, and the rate at which the compression occurred. He noted that small compression of the chest could cause fatal damage to the thoracic organs when the compression occurred over a short time period. He called this the viscous effect and said that during slow compression of the chest, the body tissue can deform without damage. However, when the compression occurs quickly, the tissue cannot deform rapidly enough to prevent damage. Studies performed with cadavers, animals and a Hybrid III anthropomorphic simulator led to the development of the term CV_{max}. This is the maximum of the instantaneous product of the fractional chest compression, C, times the rate of compression, V. When the rate of tissue compression exceeds some threshold, the damage mechanism changes from one consisting of a simple crushing/shearing to one involving a more complicated visco-elastic response (rate-dependent response). This is very similar to dynamic loading where all loading on a structure will have the same effect if the product of peak pressure and total impulse is constant. Thus, a dynamic load can have the same effect as a much greater static load. An analogy is easily made to CV_{max} were V is analogous to peak pressure and C is analogous to total impulse. Therefore it is reasonable to assume that CV_{max} will also correlate with fracture of the ribs.

Viano correlated the damage to human cadavers and surrogate animals by a blunt impact with the experimentally measured CV_{max} from identical impacts on a Hybrid III anthropomorphic simulator. He concluded that impacts to the chest which produce a CV_{max} equal to 1 m/s had a 25% chance of causing severe damage to the thorax, and a CV_{max} of 1.3 m/sec. to have a 50% chance of causing severe damage to the thorax. Viano considers a CV_{max} of 1 m/sec. to be a threshold for human tolerance to blunt trauma of the chest.[3] An initial attempt to measure CV_{max} for the impact of several non-lethal weapons have been made, however, there is a large variation in the measured data.[4] It does not appear that a suitable method to accurately measure CV_{max} for non-lethal projectiles has been found and substantiated. This theory, though is supported by other work, and shows promise.[1,2,7]

In fact, Clemedson recognized (in the late 1960's) the significance of the rate of compression on the severity of injury. Studies with rabbits exposed to primary blasts revealed that the rate of chest compression has a much stronger influence on the extent of pulmonary injury than the amplitude of compression. It was noted that for chest compression (in rabbits) occurring at less than 15 ft/sec surface speed, compressions as great as 20 mm caused no pulmonary lesions. However, pulmonary lesions of increasing severity occurred for 5 mm compressions when the velocity of compression increased from 15 ft/sec. to 35 ft/sec. When the rate of compression was increased to 50 to 65 ft/sec, a 5-mm compression had a 50% chance of lethality. Compressions of 5 mm in excess of 65 ft./sec. had a greater than 50% chance of lethality. If the amplitude of compression was increased to 15 mm, a compression velocity of 65 ft/sec. produced 100 percent lethality. A further increase in the amplitude of compression to 20 mm at 65 ft/sec. had a high probability of being instantaneously lethal.[8]

It is interesting to note that if you calculate CV_{max} for the case of 50% lethality, (using a rabbit chest dimension of 75 mm and assuming a constant velocity for the compression), you get $CV_{max} = 1.3$ m/sec. This agrees with Viano's estimate of 50%

chance of severe injury for CV_{max} = 1.3 m/sec. The value 75 mm is the approximate chest dimension of some New Zealand rabbits used in a study by Lau et al.

Cooper and Taylor also made observations about the rate dependence of tissue damage due to blunt trauma compression. They explained this phenomenon of additional injury, beyond that caused by the crush mechanism, in terms of longitudinal pressure waves which they called stress waves. They said the stress waves are propagated in front of, and beyond the points of compression, when the rate of tissue compression exceeds some threshold. They measured overpressure in the right ventricle caused by the stress wave immediately after impact and the subsequent compression of the heart, by the deflection of the rib cage, when swine were impacted in the thorax with 140 gram 3.7 cm diameter projectiles at 30 to 64 m/sec. They also measured the depth of chest deflection and calculated the velocity of deflection using high-speed photography. Cooper fitted two equations to these data, one for calculating normalized chest deflection and one for calculating actual chest deflection. The two equations for predicting chest deflection are based on terminal kinetic energy and the diameter of the projectile and the mass or chest thickness of the target.[2] In another set of tests swine were prepared four to five weeks prior to the tests by surgically attaching small silver spheres to several locations on the heart and aorta. Cine and flash radiography were used to measure, with millisecond resolution, the displacement and velocity of displacement of the animals' hearts following impact.[9] Cooper did state, "Most impacts at 'high' velocity (say > 30 m/s) are with projectiles of low mass resulting in short duration, small displacement of the body wall at high velocity. Under these circumstances, stress waves may contribute significantly to the injury mechanism."[10] Cooper also stated that the stress waves produce injury at the micro-vascular level in regions of discontinuity such as the air/tissue interfaces of the lungs, stomach, and intestines.[10] This may be the effect responsible for the formation of pulmonary lesions from exposure to a primary blast.

Some Comparisons

Cooper's model for chest deflection can be related directly to the Edgewood Arsenal model. Multiplying the Edgewood Arsenal parameter MV^2/WD by ½ and dividing by 1000 g/kg converts it to the variable E/WD in Cooper's equation for calculating normalized chest deflection = $0.4\left(1-e^{-0.95(E/WD)}\right)$, where E = kinetic energy of the projectile in joules, W = mass of the target animal in kg., and D = diameter of the projectile in cm. This allows chest deflection, calculated as a function of kinetic energy and projectile diameter, to be related to the predicted lethality using the Edgewood Arsenal model. The implication of this is that if the ratio of kinetic energy, KE, to diameter, D, is held constant for a series of projectiles, they will all have the same predicted lethality by the Edgewood Arsenal Model and the same predicted depth of chest deflection by Cooper's model. Two questions to ask are: Under what conditions could this be valid? Do other models of lethality support the contention of identical lethality when KE/D is held constant?

The first condition that must be met for either of these two models to be valid is that the projectile impact be non-penetrating. This places limits on kinetic energy that is projectile dependent for a given target. The projectile properties of cross sectional area and density, shape and compliance influence the tendency of a projectile to penetrate. Cooper's expression for normalized chest deflection approaches a maximum of 40% as the ratio of E/D increases, therefore correlation with the Edgewood Arsenal model should

not be made near values of 40% relative chest compression. Also the impacts should be in a region were they produce a non-lethal outcome. It is meaningless to compare lethal impacts, as lethality does not have a relevant severity scale. Next, the meaning of KE/D must be considered.

With no other constraints, the constant KE/D means very little. At any given KE the value for momentum can vary widely. At constant KE, when momentum is maximized and the terminal velocity drops below some threshold, whole body displacement of an unrestrained target will occur before significant tissue damage by compression occurs. On the other hand, when momentum is minimized, the inertia of the projectile becomes so low that the projectile is rapidly stopped by the target, thereby delivering all its kinetic energy before any significant displacement of the body wall occurs. Therefore, kinetic energy thresholds are probably only valid within a specified range of momentum values.

Momentum is directly related to the impulse from the impact and the duration of the target/projectile interaction. The impulse and duration of the impact influence the mechanism of target/projectile interaction and thus the physiological response to the impact. At a constant energy, increasing the momentum of the projectile increases the impulse and the duration of the impact. The greater impulse means that the impacted tissue is exposed to pressure under the projectile for a longer period of time. The greater momentum also means that the duration of the impact is greater, thus the transfer of energy from the projectile to the target is spread out over a greater length of time. This favors injury by a simple crush and shear mechanism. As the momentum is decreased, at constant KE, the impulse and the time of projectile target interaction decrease. This means the tissue is compressed for a shorter length of time and that the same amount of energy is transferred to the target in a shorter length of time. Since energy and work have the 'same units', minimizing momentum maximizes the power, which is work per unit time, of the impact. This favors injury by Viano's viscous model. Since both models (Edgewood Arsenal and Cooper) were derived semi-empirically from experimental results of blunt impact, and because the mechanism of injury and the efficiency with which energy is transferred from the projectile to the target is largely momentum dependent, these models are most likely only valid for projectiles with similar momentum, kinetic energy and diameter to those used by the two research groups.

Furthermore since the body is made up mostly of water, its viscosity increases as the rate of deformation increases. This has a synergistic effect as the momentum decreases at constant kinetic energy. The increase in viscosity causes the body to apply a greater force of resistance to the projectile. Thus, further decreasing the projectile target interaction time and the impulse with a subsequent increase in the rate of energy transfer from the projectile to the target. This increase in resistance to flow by the body as the momentum of the projectile decreases will cause the actual depth of deflection to deviate from that predicted by Cooper's equation. Cooper's equation says that chest deflection is directly related to kinetic energy. Kinetic energy and work have the same units of force times distance. Since the force is the body's resistance to flow, deflection by the projectile decreases as the resistive force of the body increases. Therefore the two models are also probably only valid when the impact velocity is within the range of velocities used in the testing.

Discussion

From the cited references it is apparent that the physiological effects of blunt trauma are due mainly to two simultaneous injury mechanisms. The first mechanism is crush and shear. This mechanism dominates the injury when the displacement or compression of tissue occurs at relatively low velocity. This low velocity displacement creates injuries by crushing organs and applying shearing forces to arteries, veins, bones, and connective tissues. The second mechanism is the previously described viscous mechanism of damage. Viscous damage is basically crush injury with time dependence. When the crush or compression occurs rapidly, the tissues being compressed cannot deform rapidly enough to relieve the sudden increase in hydrostatic pressure. This results in micro-vascular injury beyond that which would have been expected from a long duration crushing injury of the same displacement. As mentioned, this time dependence is analogous to shock loading of a structure. Additionally, the pressure pulse that develops from the high pressure in front of the projectile, if of sufficient amplitude, will cause further damage as it propagates beyond the volume of tissue that was displaced. The damage done by the pressure pulse, beyond the volume of tissue that is directly displaced or compressed by the projectile, is accentuated in regions of discontinuity such as the air/tissue interfaces of the lungs, stomach, and intestines.[10]

When a non-penetrating projectile hits a body it deflects the body wall, compressing and displacing organs that are in its path. If the deflection occurs very slowly the total amplitude of the deflection can be rather large before any significant physiological damage is done. Also, the pressure under the projectile which is initially low increases in a somewhat linear fashion as the amplitude of deflection increases. For the case of a slow deflection the degree of damage tends to be directly related to the amplitude of the displacement. As the velocity of the displacement increases, the total amplitude of the displacement that can occur before physiological damage decreases and the instantaneous pressure under the projectile increases. The degree of physiological damage begins to deviate from being linearly related to displacement amplitude. At some point as the velocity of displacement and the pressure under the projectile increase beyond a threshold, the physiological damage becomes exponential with respect to displacement. At and beyond this point small displacements of the body wall, that under milder conditions would cause no damage, can cause significant physiological damage.

We need to keep in mind that our interest is in non-lethal impacts that do not cause serious or permanent physiological injury. The impact from a non-lethal weapon thus should not cause damage by the viscous mechanism, as there is a high potential to produce a significant amount of tissue death. The damage by a lower velocity crush and shear mechanism, produced by a non-lethal weapon impact, must be below the threshold at which large vessels are ruptured, bones are broken, and any internal organs are fractured. In order to design non-lethal projectiles that produce the desired terminal effect two things are needed. First, a better understanding of how the projectile properties and the terminal parameters determine the mechanism and the magnitude of the projectile/target interaction is needed. This will allow projectiles to be designed to produce an impact with the desired terminal effect. Second, a better understanding of the physiological threshold to the combined effects of blunt trauma resulting from the different trauma mechanisms is needed. This will allow projectiles to be "tuned" so that their terminal effect falls below the threshold for serious or permanent injury.

To a large extent, the kinetic energy determines the depth of deflection of the body wall. However, momentum also has an influence on deflection depth. This is because the momentum at a given kinetic energy has a strong influence on the duration of the impact. Short duration impacts (lower momentum) cause the deflection depth to decrease due to the viscous effects of body fluids. More important though is the dependence of wounding mechanism on the duration of the impact. Short duration impacts favor wounding (if any) by the "Viscous Mechanism" and long duration impacts favor wounding (if any) by a crush and shearing mechanism. Both these mechanisms, viscous and crush and shear, occur for every impact. The severity of the damage by these mechanisms, at a given kinetic energy, is to a large extent determined by the projectile momentum. The importance of the relation between momentum and kinetic energy was observed from fragmentation studies on goats where the degree of incapacitation from a fragment hit was related to $MV^{3/2}$, which is the square root of the product of momentum and kinetic energy. This is also similar to shock loading where the product of peak pressure and total impulse is constant for an identical effect. High momentum favors crush and shearing, low momentum favors viscous injury. Construction of the projectile can also affect the mechanism of projectile/target interaction. Blunt projectiles spread the initial impact force over a larger area of the target. This helps to decrease pressure maxima, and the possibility of localized viscous injury due to the rapid compression of tissue from the high pressure developed at the tip of the sharp ogive. The blunt ogive also minimizes the likelihood of penetration. A compliant projectile is also beneficial as it helps to increase the projectile/target interaction time and it helps to absorb some of the energy of impact, lowering the pressure under the projectile, which allows for a greater terminal energy, which is beneficial from an aerodynamic point of view.

In order to know why one projectile produces a severe wound and another does not you must know the mechanism of each interaction. The mechanisms of projectile/target interactions can be elucidated from studies of impacts where the results are related to the projectile and not to a potential physiological response. A uniform and instrumented test medium can be used to measure the effect of changing one (if possible) projectile parameter over a range of values. For instance the kinetic energy, diameter, compliance and shape of a projectile can be held constant as the momentum (mass times velocity) of the projectile is changed. Conversely the momentum can be held constant and the kinetic energy varied. Of course, in a similar manner the effects of varying the other projectile parameters can be investigated. The measured data from the impacts can be correlated to projectile properties and later to a physiological response.

Ideally, the physiological response could be predicted using a computer model of a human target. Thresholds resulting from the computer model could be verified with limited animal testing. The instrumented test medium could be a three-dimensional array of pressure sensors dispersed in modeling clay, ballistic gelatin, or a high viscosity oil. Pressure measured as a function of time after impact and location of transducer relative to impact site could be correlated with the degree of deflection and the velocity of deflection measured by high-speed photography. Alternatively, a block of ballistic gelatin could be prepared with a dispersion of micro-spheres containing a die activated by a component in the gelatin. Any volume within the gelatin where the pressure exceeded the rupture strength of the micro-spheres would be made visible by the dye. Knowledge of the

rupture strength of the micro-spheres and the dispersion of the dye in the gelatin could be correlated to projectile parameters and a physiological response.

Recommendations

Future testing of non-lethal projectiles should use advances in materials, computer modeling, and instrumentation to avoid the need for additional animal or perhaps even cadaver testing. Proper instrumentation and modeling would allow greater insight into non-penetrating type, non-lethal injuries. Knowledge of how the physical properties and parameters of a projectile relate to the physiological response from their use against a human target will allow weapons designers to design projectiles to produce a desired terminal effect. The better understanding of the physiology of blunt trauma, that will result from the knowledge of the projectile target interaction mechanism, will aid the in developing safety certification of non-lethal weapons and in setting impact thresholds for weapons designers.

References

1) Janice M. Milosh, Jeffrey M. Widder, and Donald J. Butz, "Design Criteria for Non-Lethal Ballistic Projectiles, and Assessment of the Prototype 'Sticky Shocker'", Battelle final report Task Order 102, September 30, 1997.

2) Cooper, G. J., B. P. Pearce, M. C. Stainer, and R. L. Maynard, "The Biomechanical Response of the Thorax to Nonpenetrating Impact with Particular Reference to Cardiac Injuries," *J. of Trauma* 22, 994-1008 (1982).

3) Viano, D. C., and I. V. Lau, "A Viscous Tolerance Criterion for Soft Tissue Injury Assessment," *J. Biomechanics*, Vol. 21, No. 5, p. 387-399 (1988).

4) Cuadros, J. H., "Terminal Ballistics of Non-Lethal Projectiles," 14th International Symposium on Ballistics, Quebec, Canada, 26-27 September 1993.

5) Viano, D. C., et al., "Mechanics of Fatal Chest Injury by Baseball Impact: Development of an Experimental Model," *Clinical Journal of Sports Medicine*, Vol. 2, No. 3, p. 166-171, (1992).

6) Egner, D. C., The Evaluation of Less-Lethal Weapons, U.S. Army Human Engineering Laboratory Technical Memorandum 37-77 (December 1977).

7) Clemedson, C., G. Hellstrom, and S. Lindgren, "The Relative Tolerance of the Head, Thorax, and Abdomen to Blunt Trauma," Annals New York Academy of Sciences, Vol. 152 (1968).

8) Clare, V. R., A. P. Mickiewicz, J. H. Lewis, and L. M. Sturdivan, Blunt Trauma Data Correlation, Edgewood Arsenal, AD No. A012761 (May 1975).

9) Cooper, G. J., R. L. Maynard, B. P. Pearce, M. C. Stainer, and D. E. M. Taylor, "Cardiovascular Distortion in Experimental Nonpenetrating Chest Impacts," *J. of Trauma* 24, 188-200 (1984).

10) Cooper, G. J., and D. E. M. Taylor, "Biophysics of Impact Injury to the Chest and Abdomen," *J R Army Med Corps* 135; 58-67 (1989).