DEVELOPMENT OF A MANNEQUIN FOR ASSESSMENT OF BLAST INCAPACITATION AND LETHALITY

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Summary

A growing number of conflicts have now seen the use of enhanced blast weapons that create significant and relatively long duration overpressure. This overpressure, or blast wave, is the principal cause of injury and/or lethality. Since peace keeping and peace making missions are increasingly being conducted in areas where these novel weapons could be used against the dismounted infantryman, there is an immediate need to increase the level of protection against such threats. This requires initially the development of a reliable procedure to assess the performance of protection systems. Defence R&D Canada – Valcartier initiated the development of a biofidelic and robust mannequin called the Mannequin for the Assessment of Blast Incapacitation and Lethality (MABIL) that will allow personal protection systems to be quantitatively evaluated in terms of injury potential. The research approach and injury assessment capabilities are presented here. During the primary phase of the project, priority was given to auditory injury and to head and torso injuries induced by global body acceleration and impact. Associated injury mechanisms and tolerance information were reviewed from published literature. Prototypes were fabricated and evaluated under blast loading conditions. Experimental results and their potential influence on the following development phases of MABIL are presented.

1. Introduction

Enhanced blast weapons are designed to use sustained overpressures, or blast waves, as the principal injury and/or lethality mechanism. This is in marked contrast to more 'traditional' weapons that use fragmentation as the primary kill mechanism. As a result, enhanced blast weapons represent a significant threat as current personal protective equipment for the dismounted infantryman is designed to stop fragmentation/ballistic threats not blast overpressure. With the proliferation of these types of weapons, the risk of encountering them during peacekeeping and peace making mission has increased significantly in recent years. There is, therefore, an immediate need to increase the level of protection for the dismounted infantryman against such weapon systems.

An extensive research program was initiated by Defence R&D Canada Valcartier to improve the survivability of protected soldier against enhanced blast weapons. An important part of this program consists in the development of a biofidelic and robust Mannequin for the Assessment of Blast Incapacitation and Lethality (MABIL) that will allow personal protection systems to be quantitatively evaluated in terms of injury potential. The MABIL development project is divided into five phases. The first three phases will aim at adding new features to MABIL by addressing specific types of injury (Table 1) while the last two phases will focus on the integration of the various components. A parallel activity is focusing on the development of the protection system itself.

Table 1: MABIL's injury assessment capabilities.

Phase	Injury Type
1	a) Head injuries caused by global body acceleration and impact
	b) Torso injuries caused by global body acceleration and impact
	c) Middle and inner ear injuries caused by blast overpressure
2	a) Lungs injuries caused by blast overpressure
	b) Thermal burns affecting head and torso regions
	c) Eye injuries caused by flash
3	a) Airways and bowel injuries caused by blast overpressure
	b) Heart, spleen, bladder, liver and kidneys injuries caused by blast overpressure
	c) Head and torso injuries caused by secondary fragmentation
	d) Eye injuries caused by flying debris and blast overpressure
	e) Injuries caused by chemical products (e.g. burns and intoxication)

Initial findings of the first phase of development are presented in the following sections. It includes a description of the preliminary design requirements followed by a review of injury mechanisms and tolerance information.

2. Background

Explosive devices can produce various types of injury. They can be divided into four categories depending on the mechanisms involved:

Primary blast injuries are caused by the direct interaction of the blast wave (overpressure) with the body. When a pressure wave impacts an individual, it produces distributed forces acting on the impinged body surface causing shock wave propagation (stress/strain) through the body tissue. This can cause disruption if tolerance levels are exceeded. This type of injury is mainly observed in gas containing organs such as the lungs and airways, the gastrointestinal tract, and the auditory system. These organs are believed to be more susceptible to primary blast injury because of the interaction of the shock wave with the interface of high density (tissue) and low density (air) resulting in higher stress concentration [Maynard and Coppel 1997].

Secondary blast injuries are produced by projectiles striking the victim. These projectiles consist of bomb fragments or other objects thrown by the blast. Small objects travelling at high velocity typically result in penetrating injuries (unprotected) or localised blunt trauma (behind protection). Associated injury mechanisms consist of damage to the structures (e.g. bone) close to the projectile pathway as well as tissue rupture and laceration. Larger objects, such as bricks, propelled by the blast may result in a variety of non-penetrating injury patterns (e.g. contusion, dislocation, fracture, concussion, traumatic amputation, etc.) while the precise injury outcome is a function of the impact location on the body and the mass, shape, velocity and compliance of the projectile.

Tertiary blast injuries are related to the displacement of the body. Injuries are caused by rapid acceleration (i.e. inertial loading) or when the victim impacts solid objects. The severity and type of injuries depend primarily on the body characteristics and orientation, launching/impact velocity, and the characteristics of the solid objects. Tertiary injuries correspond typically to the non-penetrating injury patterns described previously.

Quaternary blast injuries include other injury mechanisms such as thermal injury (e.g. skin burn) from radiant and convective heat of the explosion, and toxicity (i.e. inhalation of combustion products). Specific injury types and severity are mainly a function of the explosive device used.

3. Design Requirements

A preliminary review of the requirements defined for MABIL suggests that the injury assessment capabilities can be divided in two distinct categories. The first category includes injuries caused by global body acceleration and impact. Assessment of these injuries requires the reproduction of body kinematics. Thus, a device replicating primary human body characteristics such as shape, dimensions, inertia and weight is mandatory. The capability to withstand impact and high acceleration is also required. The second category encompasses all the other types of blast injuries defined in Table 1. Global body motion is not required and should be avoided to reduce the risk of damaging the surrogate and its instrumentation. The proposed MABIL concept can be described as one system with two possible configurations:

1. The first configuration is a complete mannequin representing the main characteristics of a human body. It is based on the technology of Anthropomorphic Test Devices (ATD) commonly used to evaluate the performance of protection systems in automotive safety research.
2. The second configuration consists of a simplified physical model of the head and torso with the measurement systems required to assess the risk of blast injuries. It is proposed to mount this model on a rigid structure to maintain its position and orientation during testing.

4. Injury Mechanisms and Tolerance Information

4.1 Head Injury Caused by Body Acceleration and Impact

Head injuries from global body acceleration (inertial loading) and impact occur as the result of tissue stress or strain caused by the application of a mechanical force. The main parts of the head affected are the scalp, the skull, and the brain and its vasculature. In the context of blast loading, these injuries are associated with the secondary and tertiary categories as defined previously. Impact-induced injuries are associated with short-duration impulsive loading and high peak acceleration while injuries resulting from inertial loading are caused by pure linear and/or angular acceleration pulse of longer duration [Little 1993].

For this project, priority was given to skull fractures and brain injuries since damages to the scalp are typically associated with lower trauma severity. Skull fracture is the result of an applied force exceeding the structural limit of the bone material which varies as a function of the impact location, gender, age, load distribution, loading rate, and bone thickness/composition. They occur from impact only since inertial loading alone cannot produce skull fracture. Brain injuries occur when an external force, resulting from inertial loading or impact, causes tissue to rupture from excessive distortion. Specific injury mechanisms involved are a function of the load characteristics. Table 2 presents common types of brain injury mechanisms as identified by Melvin and Lighthall. The last three mechanisms listed in Table 2 are believed to be involved in both impact and non-impact situations. Resulting injuries are typically divided into two categories, focal and diffuse, based on the type of damage. Focal injuries are more localized to regions of the brain where tissues are subjected to tensile or compressive stresses. Diffuse injuries refer to brain swelling, concussion, and damages to neural, axonal, and micro-vascular structures [Bandak 1997]. While most head injury models use the occurrence of skull fracture as a predictor of brain injuries, it has been demonstrated that damage to tissues contained inside the skull may happen with or without direct impact to the head and thus, in absence of skull fracture [Newman 1998]. In fact, researchers

Table 2: Brain injury mechanisms [Melvin and Lighthall 2002].

Brain Injury Mechanism
Skull deformation/fracture
Movement against rough interior surface of the skull
Infarction or pressure
Contrecoup (at the opposite side of the impact point)
Motion of the brain hemispheres relative to the skull and each other
Rupture of bridging vessels

were able to produce traumatic brain injuries experimentally with animal specimens from pure inertial loading (no impact) [Ommaya 2003].

Characteristics and proposed injury tolerance values of the main impact and non-impact injury models developed over the years are summarized in Table 3. These models have been validated for specific conditions, typically associated with car crashes, which may or may not be appropriate for blast loading applications. This table also shows that injury functions developed to predict traumatic brain injury are based on temporal responses of linear acceleration, rotational acceleration, or a combination of both. The maximum linear acceleration with dwell limit is a typical example of a requirement used in test standard for evaluating headgear. Although not reported here, there are numerous other helmet standards characterized by the type of headform, energy level, impacting surface, criteria, etc. [Newman 2002]. These standards are not necessarily correlated to actual injury data but they have shown to provide a net safety benefit.

Table 3: Summary of brain injury models.

Loading Type	Injury Prediction - Parameter	Tolerance Value	Injury Outcome	Measurement	Reference
Impact	Maximum Linear Acceleration	180 g	< 5% risk of skull fracture	Hybrid III head linear accelerations at CG	[Mertz and Prasad 1997]
	Maximum Linear Acceleration with Dwell Time	a < 400 g time @ 200g < 2 msec time @ 150g < 4 msec	n.a.	DoT headform linear acceleration at CG	[NHTSA 1988]
	Severity Index	1200	n.a.	NOCSEA headform linear accelerations at CG	[NOCSAE 1997]
	Head Injury Criterion (15 ms-HIC)	700	5% risk of AIS ≥ 4 brain injury < 5% risk of skull fracture	Hybrid III head linear accelerations at CG	[Mertz and Prasad 1997]
	Power Index	12.8 kW	50% risk of mild traumatic brain injury	Hybrid III head linear and rotational accelerations at CG	[Newman, Shewchenko et al. 2000]
No-impact	GAMBIT	0.5	5% risk of AIS ≥ 4 brain injury	Head linear and rotational accelerations at CG	[Newman 1998] [Kramer and Appel 1990]
	Angular acceleration + angular velocity change	α>8000 rad/s^2, ω > 75 rad/s α>12500 rad/s^2, ω > 60 rad/s α>15000 rad/s^2, ω > 150 rad/s	Concussion (AIS 2) Acute subdural hematoma (AIS 5) Diffuse axonal injury (AIS 5)	Head rotational acceleration at CG (sagittal plane).	[Thibault and Gennarelli 1990]
	Angular acceleration + angular velocity change	α>4500 rad/s^2, ω > 30 rad/s α>4500 rad/s^2, ω > 70 rad/s	Bridging vein disruption Gliding contusion	n.a.	[Glaister 1997]

4.2 Torso Injury Caused by Body Acceleration and Impact

Thoracic injuries from global body acceleration and impact are caused by the same mechanisms described in the previous section, i.e. tissue rupture resulting from the application of an external mechanical force. These injuries are also associated with the secondary and tertiary categories.

Loading characteristics have a significant effect on the biomechanical response of the chest, and thus, on injury outcome. Furthermore, complexity of the chest structure and internal organs required development of very specific models. For example, a known impact, i.e. characterized by the mass, velocity, loading area, compliance etc., at mid-sternum level will produce a certain type and severity of injury while the same impact on the side of the chest will result in a different injury outcome. For that reason it was chosen to limit, at least for the initial development phase of MABIL, the range of impact conditions to frontal loading.

The biomechanics of torso injury have been studied extensively over the past years to assist in the development of protection systems for automotive safety and defence and law enforcement applications. The first area involves typically low velocity-large mass impacts while the second area is associated with high velocity-low mass impacts. Research on torso injuries caused by distributed loading has also been conducted to evaluate the performance of restraint systems such as seat belts and airbags.

For focal impact as experienced in car crash situations, Viano and Lau suggested that the type of impact injuries to the torso is a function of the velocity of deformation [Viano and Lau 1988]. Crushing injuries were found to occur at velocities below 4.5 m/s where the peak compression was identified as a good predictor of injury. Viscous type injuries were observed at velocity between 4.5 m/s and 30 m/s where the viscous response (VC), i.e. the product of the sternal velocity by the chest compression, was found to be an effective predictor of injury. For non-penetrating ballistic impacts (e.g. bullet defeated by body armour), the same biomechanical parameters were identified but with different threshold values [Cooper and Pearce 1981; Cooper and Pearce 1982; Cooper and Maynard 1986; Bir 2000; Tam, Dorn et al. 2000].

In the absence of focal impact the chest loading mechanism is different. The load is distributed on the surface of the chest and the relative motion between different tissues causes disruption from stretching, bending, and distortion. This is believed to be similar to the loading produced by restraint systems used in the automotive industry, i.e. seat belts and airbags. Currently, motor vehicle safety standards use chest acceleration criterion to evaluate performance of restraint systems. The human tolerance value for severe chest injury (AIS=4) has been found to correspond to a peak spinal acceleration of 60 g sustained for 3 ms or longer when measured with the 50th percentile Hybrid III test dummy. The recent addition of airbag protection systems in vehicles has resulted in different injury mechanisms related to the deployment of such devices. The most important being the membrane type loading to the chest from frontal airbag which may present certain similarities with the blast related non-impact loading mechanism. By studying airbag-induced injury to children, Mertz and Weber found a relationship between dummy responses and animal injury severity [Mertz and Weber 1982]. Their results suggest that a sternal compression rate of 10 m/s measured with the 50th percentile Hybrid III dummy corresponded to 50% probability of severe chest injury (AIS=4). Table 4 summarizes the major findings related to the torso injury models that may be applicable for MABIL.

Table 4: Summary of torso injury models.

Loading Type	Injury Prediction - Parameter	Tolerance Value	Injury Outcome	Measurement	Reference
Impact	Chest compression	C_{max} = 34%	25% risk of AIS≥4	Hybrid III 50th male Chest compression	[Viano 1997]
	Chest compression	C_{max} = 20%	50% risk of sternum fracture and myocardial bruising	Swine Chest compression	[Cooper and Pearce 1982]
	Viscous response	VC_{max} = 1.0	25% risk of AIS≥4	Hybrid III 50th male Chest compression or sternal + thoracic spine accelerations	[Viano 1997]
	Viscous response	VC_{max} > 0.6	AIS = 2	BABT Torso Rig Backface deflection + acceleration	[Bir 2000]
No-impact	Spinal acceleration + duration	A_{max} > 60 g for >3 ms	AIS =4	Hybrid III 50th male Upper spine linear accelerations	[Cavanaugh 2002]
	Chest velocity	V_{max} > 7 m/s V_{max} > 10 m/s	50% risk of AIS =3 50% risk of AIS =4	Hybrid III 50th male Chest compression or sternal + thoracic spine accelerations	[Mertz and Weber 1982]

4.3 Ear Injury

The primary interest of the current work consists in identifying injury models associated with blast related trauma to the ear. In addition, it is expected that impulse noise-induced hearing loss may become relevant when protection systems offer sufficient overpressure attenuation but not necessarily

enough to prevent permanent or temporary hearing loss. While not a lethal injury, hearing loss can be extremely incapacitating particularly from the point of view of situational awareness, let alone the disorientation and discomfort associated with this type of injury.

The ear is considered the most susceptible organ of the human body to blast overpressure. Damage is observed primarily at the tympanic membrane and the middle ear (i.e. the air space where the ossicles are located). Haemorrhages and eardrum (tympanic membrane) perforation are the initial injury mechanisms while fracture, dislocation or disruption of the ossicles are observed at higher pressure levels [Garth 1997]. Injury criteria for eardrum rupture is usually defined in terms of peak static pressure (overpressure) measured by a pressure transducer oriented side-on to the blast wave. Although perforation of the tympanic membrane was observed at pressure level as low as 35 kPa (5 psi), the accepted threshold value is 104 kPa (15 psi or 1 atmosphere) which corresponds to a 50% risk of eardrum rupture [Horrocks 2001]. In addition to the peak pressure, other parameters have been shown to affect the type and severity of ear injury. Using experimental data, Richmond and Axelsson were able to predict the occurrence and nature of eardrum rupture as a function of the peak incident overpressure and duration of positive incident overpressure for a person facing the blast source in free-field [Richmond and Axelsson 1990]. James and Pickett proposed another injury model using experimental results obtained with cadaver specimens [James and Pickett 1982]. Pressure was measured at the level of the tympanic membrane but no meatus and pinna were present. This model uses peak overpressure and positive impulse at the level of the eardrum to predict injury. The main advantage of this model is that it could be used for performance evaluation of protection systems because measurements were conducted *in situ*. Table 5 presents a summary of the ear overpressure injury models described above.

Impulse noise-induced hearing loss is typically characterized with temporary threshold shift (TTS) or permanent threshold shift (PTS). These parameters quantify the hearing sensitivity shift (in decibels) after exposure to a sound. A temporary shift lasts less than 6 months while a permanent shift is still present after 6 months. A summary of noise-induced injury functions is presented in Table 5. Most functions use peak pressure, impulse duration, and the number of times the impulse noise is being repeated to predict the occurrence of injury [RTO-NATO 2003]. The major difference between them resides in the calculation method used to evaluate impulse duration. The French Committee for Weapon Noises recommended another criterion. It uses the pressure time history, filtered with an A-weighting filter, over a period of 8 hours to establish the TTS. Finally, a mathematical model of the ear was developed and validated by the US Army Research Laboratory (ARL) to be used for predicting risk and severity of impulse noise-induced hearing loss. The model is designed to reproduce the nonlinear behaviour of the human ear and its components. Injury prediction approach is based on mechanical fatigue at the level of the basilar membrane [Rouhana, Webb et al. 1998]. The model was found to respond similarly to the human ear to critical parameters of the insult (peak pressure, noise frequency content) and shows good correlation with a range of test data.

5. Experimental Trials

The main objective of the first series of experimental trials was to characterize the physical response of the surrogates in a blast loading environment. Measurement requirements were based on injury models described in the previous section.

Table 5: Summary of ear injury models.

Loading Type	Injury Prediction - Parameter	Tolerance Value	Injury Outcome	Measurement	Reference
Overpressure	Ear overpressure (facing blast wave)	P_{max} = 104 kPa	=50% risk of eardrum rupture	Static pressure, transducer oriented side-on to the blast wave.	[Horrocks 2001]
	Ear overpressure + Duration	P_{max} = 90 kPa, d<6ms P_{max} = 65 kPa, d>6ms P_{max} = 300 kPa, d<6ms P_{max} = 200 kPa, d>6ms	50% risk of minor eardrum rupture 50% risk of major eardrum rupture	Static pressure, transducer oriented side-on to the blast wave.	[Richmond and Axelsson 1990]
	Ear overpressure + Impulse	P_{max} > 130 kPa, I > 12 Pa/s P_{max} > 190 kPa, I > 16 Pa/s	50% risk of eardrum rupture 100% risk eardrum rupture	Static pressure, transducer located into ear canal.	[James and Pickett 1982]
Impulse Noise	Effective exposure level (MIL-STD-1474D)	L_M = 140 dB	TTS_2 = 25 dB	Static pressure, transducer oriented side-on to the blast wave.	[MIL-STD-1474D 1997]
	Effective exposure level (Pfander criterion)	L_P = 160 dB	TTS_2 = 25 dB	Pressure transducer located at head level, oriented side-on and facing up.	[Chan, Ho et al. 2001]
	Effective exposure level (Smoorenburg criterion)	L_S = 166.2 dB	TTS_2 = 25 dB	Pressure transducer located at head level, oriented side-on and facing up.	[Chan, Ho et al. 2001]
	8-hour equivalent A-weighted sound exposure level	L_{Aeq8} = 85 dB	TTS_2 = 25 dB	Pressure transducer located at head level, oriented side-on and facing up.	[Chan, Ho et al. 2001]
	Auditory Risk Units (ARU)	ARU = 200	0% TTS	Pressure transducer at ear level, oriented normal to eardrum.	[Rouhana, Webb et al. 1998]

5.1 Physical Models

A 50th percentile male Hybrid III mannequin shown in Figure 1 was instrumented with linear (Endevco 7264B-2000) and rotational (Endevco 7302BM4) accelerometers at the head CG and linear accelerometers (PCB 350B03) at the upper spine. In addition, three headforms shown in Figure 2 were positioned on rigid posts and instrumented with two pressure transducers (Endevco 8510C-50 and 8510C-100). One pressure transducer was located in the mouth and the other was positioned at the level of the eardrum in a simplified ear canal based on the geometry of a life-size ear model. Each headform was also equipped with a photodiode (UDT Sensors PIN-10AP) located at eye level. Although measurement of light intensity was not part of the first phase objectives, it was included because of availability of sensors. These headforms were based on the external shape of the mid-size facially-featured headform specified in test standard CSA-Z262.2-M90 for face protectors and visors for ice hockey players. This headform was proposed

Figure 1: Hybrid III Mannequin.

Figure 2: Instrumented headform.

because it is readily available while being representative of an average human head. In addition, it has anatomical details, allows easy installation of helmets, and provides a good interface with other head protection systems. Pressure transducers (PCB 113B51) were also positioned at various locations beside the physical models to characterize the blast event.

5.2 Test Set-up

Position of the headform was fixed at 5 m from the charge for all tests while the Hybrid III dummy was located successively at 5 m, 4 m, and 3 m from the charge. Headforms and charge heights were set to 1.5 m from the ground. The Hybrid III dummy was placed in a standing position. It was supported under the arms and was free to move backward. High explosive (5kg C4) charges were used and the physical surrogates were not protected. In this first project phase, high explosive was used to provide a reference for future comparison with various enhanced blast charge compositions. Also, since the initial objectives were to evaluate the surrogates' response and to assess current injury criteria, it was not found necessary to reproduce the enhanced blast profile characteristics.

6. Results

Figure 3 shows peak side-on pressure values as a function of the distance from the charge. This information can be used to quantify input load on the physical surrogates. It is observed that peak pressure does not vary significantly for standoff distance greater than 5 m.

Note that injury threshold values shown in the following figures are indicated for reference only. They are not expected to be applicable directly since the injury models were not developed for the current loading conditions.

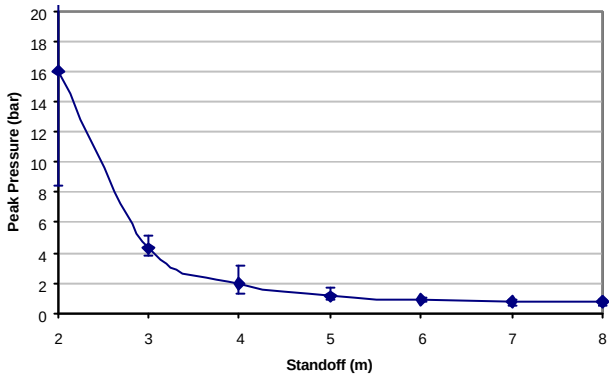

Figure 3: Peak side-on pressure vs. standoff distance form the charge.

6.1 Head Acceleration

The Hybrid III head acceleration results are summarized in the following figures. Despite the limited number of repetitions, it is possible to identify general trends. As expected, resultant linear acceleration increases with shorter standoff distances, Figure 4. It is suggested that the risk for injury is significantly greater at a standoff distance of 3 m. The same trend is also depicted in Figure 5 which shows HIC values as a function of the distance from the charge. In these figures, data points correspond to single test results.

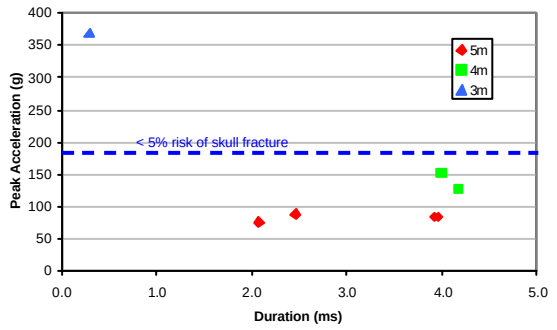

Figure 4: Peak head linear acceleration and duration.

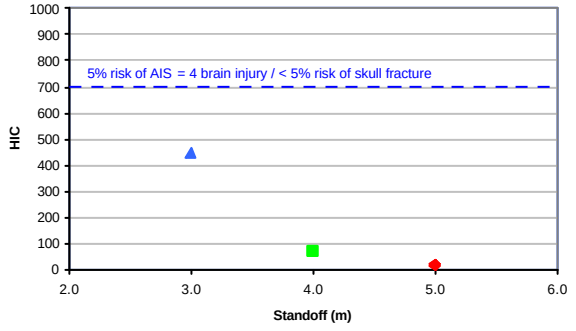

Figure 5: HIC values as a function of the standoff distance.

Rotational accelerations results are provided in Figure 6 with injury thresholds values proposed by [Thibault and Gennarelli 1990]. The response obtained at a standoff distance of 3 m was rejected because of faulty signals. As opposed to the previous figures, a 4 m standoff corresponds to a relatively high risk of injury.

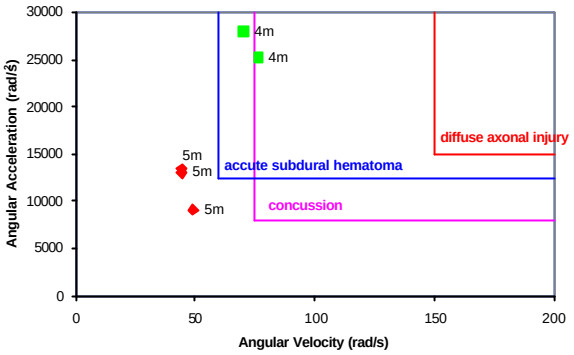

Figure 6: Peak head rotational acceleration and velocity (injury thresholds as per [Thibault and Gennarelli 1990]).

6.2 Torso

In this test series, only the spine acceleration was monitored. No value of chest deflection measurement was recorded. Therefore, only one injury model can be used for comparison with test data. Figure 7 shows the peak linear spine acceleration and the corresponding duration above 60 g for the Hybrid III mannequin standing at 3 m, 4 m, and 5 m from the charge. Again, peak values obtained at 3 m are significantly higher and correspond to greater risk of injury.

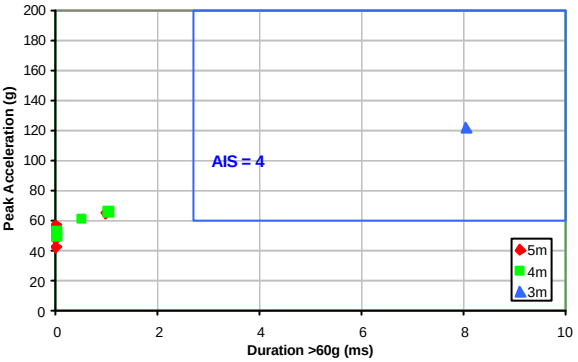

Figure 7: Peak spine acceleration and corresponding duration above the 60 g limit.

6.3 Ear

While proposed injury models include impulse-noise hearing loss, only results for ear injury caused by overpressure are reported here. Based on the injury function developed by Richmond and Axelsson, Figure 8 shows that for all cases there is a 50% risk of moderate to major eardrum rupture. On the other hand, when using the injury function proposed by James and Pickett, all cases correspond to a complete rupture of the eardrum, Figure 9. This suggests that the ear canal model used in these experiments over amplify the pressure in comparison with the human response.

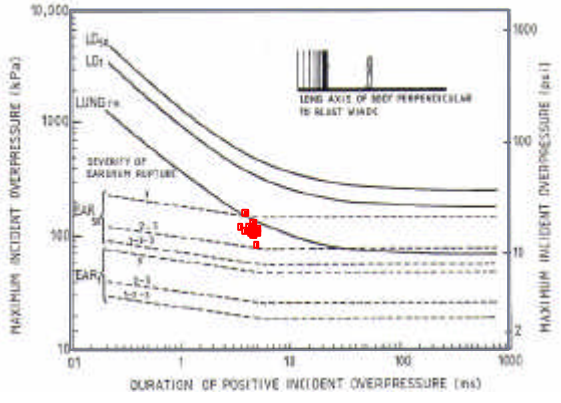

Figure 8: Injury prediction for eardrum rupture, modified from [Richmond and Axelsson 1990]. Experimental results are indicated with red squares.

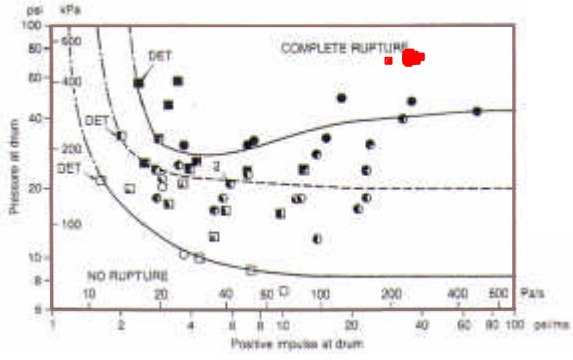

Figure 9: Eardrum injury prediction, modified from [James and Pickett 1982]. Experimental results are indicated with red squares.

Figure 10 compares the peak values recorded from the reference transducer (side-on) positioned beside the headform and two pressure gauges installed in the headform, one in the ear canal (side-on) and one in the mouth (face-on). The error bars represent minimum and maximum values recorded. As expected, the pressure sensor installed in the mouth recorded peak pressure about 2 times greater than the reference value. It is also clear from this graph that the ear canal amplifies significantly the pressure signals which confirms that injury models based on free-field pressure measurements can not be used with pressure measured at the level of the eardrum.

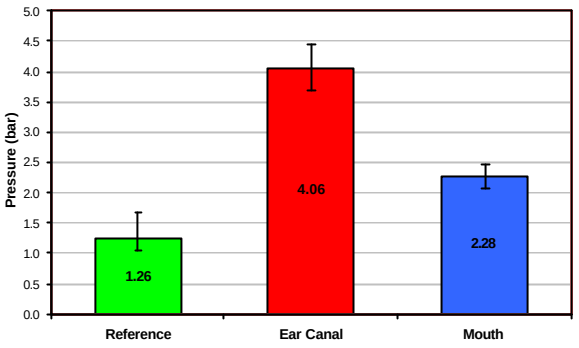

Figure 10: Effects of pressure transducer orientation and location.

Other results were analyzed to further quantify the effect of the ear canal on the pressure signal recorded. Thus, for the same orientation with respect to the blast wave (i.e. side-on) the transducer responses were compared to identify the amplification ratio provided by the ear canal. Figure 11 shows that the reference transducer and the pressure gauge located in the mouth of the headform provided approximately the same peak values. In comparison with the transducer located at the level of the eardrum, the mean peak value is 3.9 times greater than the average peak reference value. When the ear canal is oriented face-on to the blast, the signal is amplified 5.9 times in comparison with the reference signal, Figure 12.

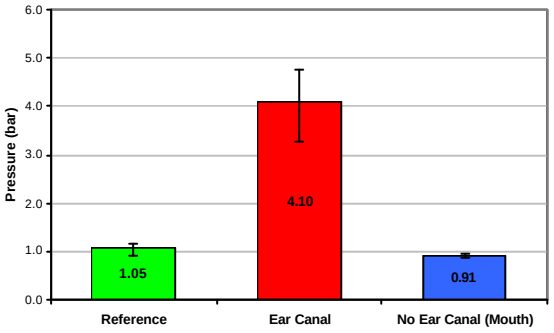

Figure 11: Effects of ear canal.

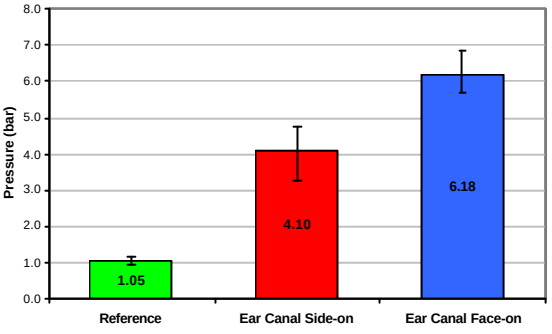

Figure 12: Effects of ear canal orientation.

7. Conclusions

In most cases, injury functions described in the previous sections were not developed for the conditions under which MABIL is expected to be used. Although, assuming that injury mechanisms do not change, one could presume that the biomechanical parameters proposed to predict injury (e.g. head rotational acceleration, chest deflection, etc) remain applicable for blast loading conditions. Re-evaluation of the structure biofidelity and tolerance values using biological test specimens and epidemiological data must then be considered for these new loading conditions. In absence of such data, accurate injury prediction will not be possible but relative ranking of protection systems may be feasible.

In this first phase, the development of MABIL was based on existing physical models and injury assessment functions. Based on current knowledge, these functions represent what is believed to be the most appropriate ways of evaluating potential damages associated with the first set of injuries defined in Table 1. The proposed injury models were organized by body region and by type of loading. For

head and torso injuries, a distinction shall be made when reviewing experimental data between impact and no-impact loading since it may involve different injury mechanisms. For ear injury, it is suggested to consider blast and impulse noise simultaneously. Higher pressure will likely result in blast ear injury while lower pressure will correspond to temporary or permanent hearing loss.

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