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Blast Headform Development

- Litereture Review -

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Biokinetics Report No.: R07-09
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Contract No.: W7701-062565/001/QCL
Scientific Authority: Kevin Williams, (418) 844-4000 ext 4238

Defence R&D Canada – Valcartier
Contract Report
DRDC Valcartier CR 2007-234
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Canada

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September, 2007

This report constitutes a deliverable for Task 1, Traumatic Brain Injury Review, of the project for the Development of a Novel Biofidelic Headform for Blast Induced Brain Trauma Assessment.

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Abstract

Changes to the threat types, their exposure rates and improvements to protective equipment have changed the injury patterns that are seen in the battlefield, with brain injuries becoming more prevalent. Although a considerable number of publications exist that address the effects of blast injury to the lungs, the mechanism(s) by which brain injuries are caused by exposure to blast is largely unknown. It is still not fully understood how a blast shock wave might damage head/brain structures such as cerebral tissues or blood vessels. A better understanding of the effects of blast overpressure on the head is required such that a surrogate headform can be produced for the design and evaluation of head protective systems for blast loads.

A literature review was conducted to further the general understanding of blast effects on cerebral tissues and provide insight into possible headform assessment methods that can be used to correlate surrogate headform responses to real brain injuries. Based on the findings of the literature review, it is anticipated that a surrogate blast headform and its associated injury criteria will be based on detailed measurements of wave propagation through soft tissue surrogates and other structures that are represented within the surrogate headform.

Résumé

Une évolution des menaces posées par les armes à effet de souffle, une augmentation de leur taux d'exposition, de même que des améliorations du matériel de protection du personnel militaire ont changé le type de lésions observées sur les champs de bataille, avec des blessures traumatiques au cerveau devenant de plus en plus répandues. Bien qu'il existe un nombre considérable de publications qui traitent des lésions d'effets de souffle primaires au poumon, les mécanismes par lesquels l'onde de choc causerait de telles blessures au cerveau sont peu connus. En fait, il n'a jamais été établi avec certitude comment ces ondes endommageraient des structures de la tête et/ou du cerveau telles que les tissus ou les vaisseaux sanguins cérébraux. Une meilleure compréhension des effets du souffle sur la tête est essentielle afin de permettre la production d'un modèle de tête pour effet de souffle qui permettrait l'élaboration et l'évaluation des systèmes de protection de la tête humaine exposée à différentes charges explosives.

Une revue détaillée de la littérature a été effectuée afin de bien comprendre les effets du souffle sur les tissus cérébraux et d'identifier les méthodes possibles d'évaluation de modèles de tête pour effet de souffle qui seront utilisées pour corréler les réponses de ces modèles aux blessures cérébrales typiques vues lors d'explosions. Sur la base des résultats de cette revue de littérature, on prévoit qu'un modèle de tête pour effet de souffle et ses critères de blessures associés seront basés sur des mesures détaillées de la propagation de l'onde de choc à travers les substituts synthétiques de tissu mou et autres structures qui sont représentés dans le modèle.

Executive Summary

The occurrence of brain injuries has increased significantly during recent conflicts, likely due to the changed nature of modern combat. Improvements to ballistic protection offered by the latest generation of ballistic armour, including the helmet, have changed the injury patterns seen in the battlefield, with brain injury becoming more prevalent. Changes to the types of threats seen in the battlefield (IEDs, RPGs, AV mines, etc.) along with greater exposure rates have also contributed to the increased number of brain injuries.

A literature review was conducted to assess the state of knowledge pertaining to blast related brain injury and to define the characteristics required of a surrogate headform that would be suitable for assessing blast-related injuries. The injury mechanisms were categorized from primary to quaternary, with primary injuries being related strictly to the blast shockwave traversing through the skull and brain.

The effects of primary blast forces on the head/brain can result in injuries that vary in severity from concussion to death. The severity of injury depends on the magnitude of blast and a person's proximity to the blast origin. Surrounding structures, such as walls, must be considered in addition to proximity as they can have an amplifying effect on the blast forces.

Few attempts have been made to assess the effects of pure primary blast forces on the central nervous system. Kinetic energy transfer however, is a proposed mechanism of brain injury in which shockwaves traveling through the brain result in pressure increases in the brain tissues and motion of the brain relative to the skull. The most direct method to monitor for the shockwave propagation is the use of pressure and displacement sensors.

It may be important to represent the different type of tissues and fluid filled compartments found in the brain because they have a mitigating effect on shock wave induced strains. However, for practical, technical and manufacturing reasons, it may not be feasible to model all of these complex brain structures. Therefore, for the purposes of the initial blast headform development, a homogeneous brain surrogate material can be adopted that would provide an averaged brain response to blast loading. This approach has been used to create brain surrogates by other researchers.

Although, the current literature review has provided some initial guidelines for the development of a blast headform, additional research is required to assess head/brain characteristics that would better define the biofidelity requirements of a blast headform.

Ed Fournier, David Sullivan, Tim Bayne, Nicholas Shewchenko, Lucie Martineau "Blast Headform Development – Literature Review", Biokinetics Report R07-09, 2007. Produced for DRDC Valcartier. DRDC CR2007-234.

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Sommaire

L'incidence des lésions cérébrales a augmenté considérablement lors des récents conflits, vraisemblablement causée par la nouvelle nature des combats. L'amélioration de la protection balistique offerte par la dernière génération de veste pare-éclats et casque de combat a eu pour effet de modifier le profil des blessures observées dans les champs de bataille avec notamment un accroissement des traumatismes cérébraux. Les nouvelles menaces (engins explosif improvisés, RPG, mines anti-char, etc.) combinées à un taux d'exposition supérieur ont aussi contribué à l'augmentation des blessures au cerveau.

Une revue de la littérature a été effectuée afin d'évaluer le niveau actuel des connaissances sur les blessures au cerveau causées par l'effet de souffle ainsi que pour définir les caractéristiques requises d'un modèle de tête pouvant servir à évaluer le risque de blessure occasionnées par l'effet de souffle. Les mécanismes de blessure furent catégorisés de primaire à quaternaires. Les blessures primaires étant reliées strictement à l'onde de choc se propageant à travers le crâne et le cerveau.

Les effets de souffle primaires sur la tête et/ou le cerveau peuvent causer des blessures d'une sévérité variant de la commotion cérébrale au décès. La sévérité des blessures dépend de l'amplitude de l'explosion et de la distance entre la victime et la source de l'explosion. Les structures avoisinantes, tel qu'un mur ou un plafond, doivent également être considérées car elles peuvent amplifier les forces engendrées par l'effet de souffle.

Quelques tentatives furent élaborées afin d'évaluer les effets primaires d'une explosion sur le système nerveux central où le transfert d'énergie cinétique a été proposé comme mécanisme de blessure au cerveau. Cette hypothèse suggère que l'onde de choc se déplaçant à travers le cerveau augmente le niveau de pression dans les tissus cérébraux et engendre un déplacement relatif du cerveau par rapport au crâne. La méthode la plus directe pour mesurer la propagation de l'onde de choc consiste à utiliser des capteurs de pression et de déplacement.

Il peut être important de représenter les différents types de tissus et compartiments remplis de fluide présents dans le cerveau puisqu'ils ont un effet d'atténuation sur les déformations causées par l'onde de choc. Cependant, pour des raisons pratiques, techniques et de fabricabilité, il peut être impossible de modéliser toutes ces structures complexes du cerveau. De ce fait, pour les besoins du développement initial du modèle de tête pour effet

de souffle, un matériau homogène sera utilisé pour représenter le cerveau ce qui permettra de fournir une réponse médiane du cerveau soumis aux effets de souffle. Cette approche a également été utilisée par d'autres chercheurs pour créer des modèles de cerveau synthétiques.

Cette revue de littérature a fourni quelques données initiales permettant d'orienter le développement du modèle de tête pour effet de souffle. Toutefois, des recherches supplémentaires sont requises pour déterminer les caractéristiques de la tête et du cerveau qui pourraient mieux définir les critères de biofidélité d'un modèle de tête pour effet de souffle.

Ed Fournier, David Sullivan, Tim Bayne, Nicholas Shewchenko, Lucie Martineau. "Blast Headform Development – Literature Review", Biokinetics Report R07-09, 2007. Produit pour DRDC Valcartier. DRDC CR 2007-234.

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1. Introduction

The occurrence of brain injuries has increased significantly during recent conflicts[1-5], likely due to the changed nature of modern combat. High-explosive devices such as land mines, rocket propelled grenades, improvised explosive devices (IEDs) and thermo-baric weapons are now potential threats to the military, law enforcement personnel, and first responders. Current combat helmets have been designed primarily to defeat ballistic threats and to attenuate low-level impacts, not to protect against blast weapons. The improvement in ballistic protection offered by the latest generation of ballistic vests, plates, and helmets has also changed the injury patterns in much the same way that the introduction of airbags in automobiles led to higher rates of lower limb injuries amongst crash survivors.

Although a considerable number of publications exists that address the histopathological and pathophysiological effects of blast injury to the lungs, the mechanism(s) by which brain injuries are caused by exposure to blast is a relatively new field of study. It is still not fully understood how a blast shock wave might damage head structures such as cerebral tissues or blood vessels. However, the effects of cerebral tissue disruption in survivors can be devastating with chronic headaches, sensitivity to light or noise, impaired memory, reductions in problem-solving abilities, and behavioural changes. A better understanding of the effects of blast overpressure on the head is therefore required in order to design a test device capable of evaluating the performance and assisting in the design of improved head protection systems.

Current head surrogates and test methods were developed to assess the performance of protective devices designed to address head injury mechanisms associated with direct contact of the head with solid objects. They do not consider blast overpressure effects on the head. There are a few injury criteria for non-contact acceleration injuries based on the linear and rotational acceleration. Although these criteria may provide insight into some aspects of blast induced brain injury, they do not capture the effects of shock wave propagation through the structures of the brain.

It is anticipated that the blast headform and associated injury criteria will be based on detailed measurements of wave propagation through tissue surrogates and other structures that are represented within the surrogate headform. These measurements will provide insight into the loading of the brain structures that are required to make a direct link with emerging injury research on micro-structural changes to tissues caused by exposure to blast.

The objectives of the literature review summarized herein are to: further the general understanding of blast effects on cerebral tissues; and, provide insight into possible headform assessment methods that can be used to correlate

surrogate headform responses to real brain injuries. Such a correlation will undoubtedly provide guidance in the definition of a surrogate headform's construction and geometry consideration that are essential to obtaining a biofidelic response.

2. Literature Review

High-explosive blasts, from both conventional weapons such as rocket propelled grenades (RPG), landmines and bombs[6] as well as unconventional IED threats[7,8], are an aspect of modern warfare that soldiers are now facing and must be protected against.[9]

Soldiers and medical personnel must be prepared for the potential injuries that can result from events involving high-explosive blasts. Brain injuries are an especially important threat as they may potentially be life-threatening or life-altering without being visually apparent. Clinical data for brain injury resulting solely from exposure to blast waves is quite limited. Thus, epidemiology and test data obtained in experiments using biological specimens (e.g., animals, cadavers) are also reviewed to identify injury mechanisms and biomechanical predictors of injury (e.g. acceleration, overpressure) associated with blast wave propagation through the head. A review of existing physical headform models used in blast testing is also presented.

2.1 Injury Mechanisms in a Blast Scenario

There are four basic types of injury mechanisms observed in blast events.

2.1.1 Primary Blast Injuries

Primary blast injuries generally affect air-fluid interfaces and gas-containing organs (e.g., lungs, gastrointestinal tract and middle ear). They are caused by barotraumas induced by a short duration high-pressure shockwave, mathematically described as a Friedlander Wave Form, passing through the tissues.[10] A still unresolved issue is whether or not the blast wave can directly affect the central nervous system (CNS), as these injuries are not always physically apparent and might present the same symptoms as other medical conditions that are commonly diagnosed in the soldier, such as operational stress (e.g., post-traumatic stress disorder).[9,11]

2.1.2 Secondary Blast Injuries

Secondary blast injuries can affect any anatomical region of the victim through both blunt and penetrating trauma caused by debris/objects dispersed by the blast wind. The debris involved in this type of injury are either primary fragments (components of the explosive device) or secondary fragments (those that result from the explosion).[12] These types of injuries can affect any part of the CNS.[9]

2.1.3 Tertiary Blast Injuries

Tertiary blast injuries are caused by the blast wind propelling the individuals through the air and into other objects. Generally, tertiary injuries occur to people close to the detonation site. These types of injuries can cause extensive blunt trauma to the CNS, including skull fractures, open and closed head and spinal cord trauma, contusions of brain tissues (e.g., coup-contrecoup effect), concussions, as well as peripheral nerve injuries.[9]

2.1.4 Quaternary Blast Injuries

Quaternary injuries include any injuries that can not be classified as primary, secondary or tertiary. These types of injuries can include complications of existing conditions (e.g., in women who are pregnant or in patients receiving anticoagulants), hypoxia and/or asphyxiation, inhalation of toxic materials leading to poisoning, radiation exposure, as well as chemical or thermal burns.[12]

2.2 Polytraumatic Nature of Brain Injury

While the four mechanisms of injuries potentially experienced in a blast event are distinct in their clinical presentation, injuries will nevertheless tend to be polytraumatic. Indeed, the blast wave causing the primary blast injury to different internal organs is also responsible for accelerating the casualty into an object, thus resulting in a tertiary injury.[13,14] Similarly, these injuries might be further complicated by the presence of penetrating wounds and burns. Thus, some blast-exposed soldiers may even present with injuries consistent with the four mechanisms of injury. Research has recently been undertaken on the effects of blast exposure on the CNS, as very little is known regarding the interaction of the CNS and blast waves.[7,9] This topic is of particular concern as primary blast injuries are not necessarily physically apparent, and injuries involving the CNS have the potential to be debilitating or even life-threatening.[15]

2.3 Load Characterization

Primary blast injuries depend not only on how a blast wave interacts with an individual but also on the magnitude of the blast force and the proximity of the individual to the explosion.[13] The severity of tissue damage sustained by the victim and consequently the severity of injury decreases with the cube of the distance from the source of the blast[9,12]. Whether the event occurs in an open area, such as a field, or in a confined environment, such as a building or vehicle, can significantly influence the magnitude of the primary blast injury in gas-containing organs as the additive effect of reflections and reverberations can cause an increase in pressure differential.[9,12,16] For

example, people located farther than 6 m from an open-air detonation site of a 1 kg 20 kg trinitrotoluene (TNT) charge will likely not have blast injuries in any of their gas-containing organs.[17] In contrast, detonation of smaller charges in enclosed spaces may increase the rate of mortality by two-fold compared to that resulting of detonations in open spaces[18]. Whether or not brain tissues are affected by different blast loading conditions in the same manner as air-filled organs (i.e., as a function of charges and distances) has yet to be established.

2.4 Blast Injury Patterns

Few attempts have been made to assess the effects of pure primary blast forces on the CNS (for a review, see ref. [7]). While minimal research has been done regarding this topic, some studies have identified potentially susceptible cerebral regions as well as possible mechanisms of primary blast injury. Within these studies different strategies have been used to diagnose brain injury, each providing different indicators of injury that are important to recognize in order to make effective comparisons between the different studies. The injuries resulting from primary blast mechanisms can range from concussion to death.

2.4.1 Anatomical Sites of Brain Injury

Some research findings have shown that the area of the brain most commonly injured in a primary blast event is the cerebral cortex on the side facing the blast.[7,19] In these studies, the ultrastructural[a] and biochemical changes in the hippocampus of rats exposed to either whole-body or local chest blast loading was demonstrated.[7,19] Interestingly, cognitive deficits were observed not only experimentally but also in combat veterans with reduced hippocampal volume.[20]

Kaur et al.[21] reported structural alterations in the epithelium of the choroid plexus[b] following a single blast exposure, suggesting that this vascular structure is very vulnerable to a blast wave despite being deeply embedded in the cerebrospinal fluid ventricular system.

Optic tract fibre degeneration observed in rats exposed to blast overpressure exceeding 104 kPa was followed by axonopathy[c] at the level of the midbrain,

[a] Structures of the brain too small to be seen with a light microscope.

[b] The epithelium is the outer membranous tissue of the choroid plexus which is the area on the ventricles of the brain where cerebrospinal fluid is produced.

[c] A disorder which disrupts the normal functioning of the axons.

midbrain-diencephalic junction[d], and the thalamus, suggesting that humans looking directly into an oncoming blast wave would place both eyes at risk.[22]

Other studies have hypothesized that the pineal gland[e] and cerebella cortex[f] might also be injured based on the symptoms and the inherent susceptibility of these particular regions of the brain to injury.[23,24]

It is noteworthy that while the effects of non-penetrative blast on the different brain structures were apparent within hours of the blast event, the anatomy of the brain appeared normal in rats when the survival interval was increased to 28 days.[21,25] While these findings might suggest the reversible nature of blast-related brain injury, the presence of long-term behavioural and cognitive impairments was not assessed.

Victims without secondary or tertiary blast injuries but who were suspected to have sustained neurological damage as a result of primary blast injury showed impairments in memory and had short-lasting apnea. These symptoms are attributable to damage of the temporal lobe and the brain stem, respectively.[16,26] Symptoms such as headaches, sleep disturbances, sensitivity to light and noise, mood changes as well as disturbances in attention span, memory, speech and problem-solving skills have also been documented in soldiers.[9] Studies have also hypothesized that the temporal lobe of the brain is more prone to axonal shearing due to primary blast injury.[16]

2.4.2 Injury Severity

Traumatic brain injuries (TBI) ranging from very low-grade (i.e. mild-TBI or MTBI) to lethal, have been documented and hypothesized to result from primary blast forces effects on the brain. One frequently reported low-grade brain injury seen in soldiers is concussion (i.e., MTBI).[27,28] Casualties with MTBI might not show external signs of physical trauma due to the effectiveness of their personal body armour in counteracting threats. MTBI in Canadian Forces casualties is sometimes masked by high levels of trauma caused by IEDS or RPGs. Indeed, where survival is of prime importance, doctors may not look for blast injuries if there are no signs such as ruptured ear drums or blast lung.[29]

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[d] The junction between the midbrain and the diencephalon, the latter being part of the optic track of the CNS.

[e] A small endocrine gland located deep in the anterior part of the brain, and thought to be associated with sleep patterns.

[f] The cortex (outer layer) of the cerebellum comprises the anterior inferior aspect of the brain and modulates information flowing through the deep nuclei of the cerebellum.

Despite not having external signs of injury, victims may present significant behavioural symptoms and neurological deficits subsequent to the blast event[6,14,16,30], as their body armour might not protect them against the barotraumas of primary blast injury.[31] Clinical signs of primary blast-related TBI can include but are not limited to cerebral/subdural haemorrhage, oedema, contusion, chromatolytic changes in neurons, and diffuse axonal injury. Non lethal TBI can yield serious complications that might eventually lead to death.[4,7,14,15] While these findings suggest that the severity of TBI following exposure to blast waves might vary, they also reinforce the fact that the brain must be fully protected to reduce any adverse effects of neuronal injury on body functions.

2.4.3 Indirect Mechanisms of Injury

Experimental studies in which neurotrauma was caused by focusing blast waves on the middle thoracic region (i.e., over the lungs) while protecting the animal's head suggest an indirect mechanism of injury.[32,33] This pulmonary blast injury-induced neurotrauma might be related to the rise in pressure in the thoracic region, due to its compression as a result of the shockwave, which might then result in intense venous backpressure in the brain. The increased cerebral blood pressure can then lead to a rupture of the blood vessels in the meninges and brain tissues thus causing intracerebral haemorrhages.[15,30,34]

It has also been shown that hyperexcitation of the brain tissues through afferent nerve impulses from the injured lungs might lead to adverse cellular impairments, this phenomenon being further compounded by the ischemia observed following lung injury.[35] Another potential injury mechanism is the barotraumas caused by acute gas embolism in cerebral blood vessels. The emboli enter the arterial circulation after severe damage to the lungs from blast overpressure resulting in alveolar rupture. The embolus then enters the circulatory system and can cause ischemia or infarction of the brain and spinal cord.[11,13,30]

Another potential injury mechanism found in several animal studies showed that high velocity projectiles hitting the extremities can induce brain injury through the creation of transient, high-frequency oscillating shock waves that propagate in all directions in the tissues with a velocity close to that of sound in water.[34,36] While these waves are unlikely to cause structural distortions of the brain due to their short duration, they might create high-pressure differentials within the blood vessels and cause haemorrhages and minor blood-brain barrier dysfunctions when they reach the CNS [36]. The authors hypothesized that the shock waves are likely to induce brain injury through an increased release of neurotransmitters and autocoids from damaged peripheral tissues.

2.4.4 Direct Mechanisms of Brain Injury

While the direct effects of blast wave exposure on the brain are not easily discriminated from indirect effects, several studies have observed pathophysiological changes in the brains of animals exposed to blast forces at levels below the threshold for induction of macroscopic lung injury.[7,19,37] Despite damage being proven in animals, only a handful of reports have shown in humans that a brain injury might have been caused directly by exposure to blast waves. Nevertheless, a few mechanisms of direct primary blast-related brain injury have been proposed.

It has been hypothesized that blast-related neurotrauma might involve the transfer of kinetic energy to the CNS. Shock waves would travel to the brain via different body structures (e.g., bones, muscles, viscera) resulting in increased pressure in the brain capillaries and neurons.[7,30,33,38] Alternately, a blast wave propagating through the brain and skull might cause a movement of the brain relative to the skull thus causing injury. The kinetic energy transferred to the brain, causing movement, can cause brain injuries such as concussion, cerebral contusion or diffuse axonal injury (DAI).[7,30,33,38] The movement of the brain relative to the skull can also cause more serious damage to the blood vessels bridging the brain surface to the dural venous sinus, initiating pathological changes such as subdural haematoma, cerebral haemorrhage or oedema.[11] The most common areas that will be affected through this mechanism will be the superficial gray matter of the inferior, lateral, and anterior part of the frontal and temporal lobes.

DAI, which occurs at higher angular accelerations than concussion and more readily in the coronal plane, is believed to be an injury primarily related to the movement of the brain at the time of loading. Margulies et al.[39] investigated DAI resulting from rotational accelerations of the skull using physical models comprising a real adult human skull and two baboon skulls, all filled with a silicone gel. The adult skull and one baboon skull incorporated a surrogate falx fabricated from polyurethane (MP1880, J. P. Stevens & Co., Inc. MA). The second baboon skull did not incorporate the falx. Strains were measured by the deformation of a grid embedded in the brain surrogate. The presence of the falx was found to greatly reduce the measured strains. Combining experimental and physical model results, a local threshold for critical strain in the coronal plane of ε=0.094 was proposed.

Considering the different potential brain injury mechanisms, it is important to mimic as much as possible the skull-brain complex in order to effectively and accurately evaluate blast effects on the head.

2.4.5 Diagnosis of Brain Injury

Many different strategies have been employed in the experimental and clinical studies reviewed to assess the effects of blast waves on the brain. The strategies and techniques comprise measuring cognitive status along with standard imaging and neurological evaluation techniques, as well as invasive sensor techniques and quantification of novel neurophysiological markers.

In suspected cases of neurological trauma in humans, a Glasgow Coma Scale (GCS) is often employed to establish a baseline and evaluate progress during recovery.[15,40] Standard medical imaging techniques that were employed, in the studies reviewed, include Computed Tomography (CT) and Magnetic Resonance Imaging (MRI). CT scans were used to determine the presence of skull fractures, cerebral oedema and cerebral haemorrhage, whereas functional MRIs with greater diagnostic ability were used to detect DAI as it is more sensitive than CT scans for this purpose.[7,15,30] Standard neurological evaluation techniques such as electroencephalograms (EEG) were used to evaluate both human as well as experimental TBI. EEG's were used to evaluate the acute as well as chronic neurological issues observed in the subjects in multiple studies. [6,7,26,30,33,41]

Attempts have been made to establish diagnostic methods for assessing the presence of neurotrauma based on alterations in the levels of various neurophysiologic markers. Examples of biomarkers currently being investigated include eicosanoids, nitric oxide, reactive oxygen species and neurofilament proteins. All of these neurophysiologic markers appear promising for the diagnosis of TBI and are detectable in blood samples.[6,19,30,33] However, it should be noted that much of the ongoing research on the diagnostic value of neurophysiologic markers is still in the development phase and is carried out in animals. Until clinical studies are conducted on humans, one must be careful in assuming that these markers will also be effective for diagnosing blast-induced TBI in humans. Understandably, none of these assessment methods are directly applicable to correlate surrogate headform responses to real brain injuries. Nevertheless, biomarker techniques may prove to be amenable for use in the field. With controlled loading conditions, comparative studies could be developed to establish a correlation between injury outcome and surrogate headform response under replicated conditions.

Another evaluation technique for assessing neurotrauma that was reviewed involved the use of invasive sensors. In this study, the movement of a blast wave through a rat was measured using a miniature fibre optic pressure transducer (FISO FOP-MIV pressure sensor, FISO Technologies Inc., QC) placed inside the brain of a rat.[38] The rats were placed in a compressed air-driven shock tube and subjected to a blast wave. While this study did not

provide a threshold for injury based on pressure readings observed in the brain, it did provide a potential technique for evaluating blast wave propagation through the brain as it has been theorized that the pressure integrated over time (i.e., impulse) is a key factor in altering the biophysical properties of neurons.[42] This method also represents a way to evaluate energy transfer to the brain and possibly, better understand the mechanisms of injury involved with primary blast.

Yet another evaluation technique that has been used in the past and has been proven to be effective is the use of accelerometers. Accelerometers have been used by the automotive industry for many decades to evaluate the potential for head injury in car crashes. Accelerometers have been used in cadaver studies and in surrogate headforms where they have proven to be both durable and effective for assessing impact severity and correlating headform response to injury. The drawback of using automotive headforms and accelerometers is that the impulses seen in the automotive environment involve physical contact or inertial loads of some sort and are of longer duration and higher energy. An additional limitation of this method is the assumed correlation between rigid body kinematics as incorporated into the Head Injury Criterion (HIC) and Severity Index (SI) and the anatomical disruption of the brain that results from blast loading. Nevertheless, acceleration measurements are still useful and may be more appropriate for an assessment of secondary and tertiary blunt impact effects.

While there are currently many methods to evaluate neurotrauma, it is likely that a combination of these different tools might be the most effective manner to evaluate primary blast injuries to the brain.

2.5 Blast Injury – Case Studies

Despite the large amount of literature regarding blast injuries and the research currently being undertaken on the effects of blast waves on the brain, there are only limited data suggesting that human brain injury might have directly resulted from primary blast forces. As the US remains at war in Iraq and IED's are an ever present danger to soldiers, reports of brain injuries following exposure to explosive devices are becoming more frequent.[7,14,16,27,40] Many of the studies suggest that brain injury might be due to indirect mechanisms as various penetrating wounds were also reported. In the absence of physical alterations in brain structures, the diagnosis of primary blast-related TBI remains a challenge because symptoms are common to other medical conditions (e.g., operational stress). Despite the scarcity of literature, the circumstances of two recent cases in which primary blast–related TBI is likely to have occurred were found while gathering information for this review.

The first case study depicts the injuries that occurred when a dismounted soldier was caught in the blast-arc of a shoulder mounted rocket launcher during a training exercise.[16] The soldier briefly lost consciousness and was brought to a first aid station where he complained of bad headaches, blurred vision and leg pain. He was subsequently taken to a local emergency department. All CT scans were negative and the soldier did not show adverse focal neurological findings. The soldier was then transferred to another hospital for observation where he continued to complain about headache, sensitivity to light, nausea, ringing in the ears, dizziness and depressed cognitive status for the ensuing 24 hours, after which period he began to improve. The soldier was released 11 days after the event and continued to improve, but minor symptoms persisted for 4 months after the event. The authors of the case study hypothesized that the soldier suffered a chronic left peripheral vestibular lesion.

The second study describes the experiences of the crew of two Swedish armoured personnel carriers hit by antitank missiles[43]. The level of overpressure sustained during the event appeared to be lower than that required to induce lung or gastrointestinal injuries. Interviews were carried out 6-12 months after the event, giving the soldiers a relatively long period of time to compare their experiences with other soldiers involved in a similar event or even to process their experience in different ways (e.g., forgotten or suppressed memories). The recollections reported by more than half of the soldiers that were not physically injured (i.e., no penetrating wounds or fractures) may indicate the presence of concussion (e.g., loss of consciousness, headaches, nausea) caused by the blast overpressure. The incapacitation reported by some of the soldiers may also indicate that certain brain functions (e.g., memory, awareness of time, reaction time) were temporarily blocked or altered.

These case studies suggest the existence of primary blast-related injury to the CNS. However, many gaps in the information must be filled especially regarding the injury mechanisms and thresholds for humans, to fully comprehend and ultimately evaluate the injury mechanisms involved with primary blast injury to the CNS.

2.6 Performance Requirements for the Surrogate Headform

As the blast headform is meant to evaluate the effectiveness of helmets in preventing primary blast injuries, it is important to effectively and accurately represent the important aspects and regions of the human head. Considering the paucity of information available on primary blast TBI research, assumptions must be made using the rich source of literature on ballistic impacts to the head.

2.6.1 Regions and Properties of the Skull to Be Replicated

Research carried out to determine the level of complexity needed in the skull base to achieve a biofidelic response following blunt impacts has shown that the irregular geometry of the skull base must be represented in physical headform models as the movement of the brain relative to the skull has been proposed to be a mechanism of injury.[7,30,33,38] This would allow for the best representation of the complex interactions between the brain and the skull due to the role of the base of the skull geometry in reducing brain displacement[44]. These displacement effects are likely to be an important aspect during tertiary blast loads when the head undergoes substantial movement from wind blast or blunt impacts. The importance of skull geometry on primary blast loads is not currently known but is thought to effect blast transmission patterns and magnitudes in the brain.

While this review suggests that the irregular shape of the skull base may affect the response of a skull, physical and manufacturing limitations may limit the level of detail that can be incorporated in the design of a fully enclosed blast headform.

The neck, which decouples the head from the torso, provides a rotational constraint thereby affecting the angular accelerations and should be included in the blast headform design.

Some surrogate models developed for testing ballistic impacts have incorporated skin-simulating materials in their designs.[45,46] Preliminary results using a mathematical model in which heads, with and without the scalp, were exposed to a blast wave (300 kPa) were comparable (J.-S. Binette, personal communication), suggesting that it might not be required to simulate the skin in a headform designed for the assessment of primary blast loading.

Cernak et al.[33] proposed that following exposure to an explosion, the shock waves might travel to the brain via the cranium and induce injury. Thus, it appears important that the simulated bone materials selected for the blast headform possess physical characteristics comparable to those of the human skull. The properties of the human skull[47-50] are presented in Table 1 along with various plastic materials with similar properties.

Table 1: Properties of some materials considered for simulating the skull.

Properties	Units	Material									
		Skull	DuraForm GF	DuraForm PA	P400 ABS	E-shell 100	E-shell-200	R5-R11	Nanocure RC25	Y8 Photopolymer	TC 854 Polyurethane
Density (Avg.)	g/cm^3	1.4	1.4	0.97	1.05	-	-	-	-	-	1.2
Tensile Strength (Avg.)	MPa	57.0	38	44	34	48	58	50	46	27	69.4
Modulus of Elasticity	GPa	9.6	-	-	-	2.4	2.4	1.4	4.9	0.5	-
Tensile Modulus	GPa	5.3	5.9	1.6	2.5	-	-	-	-	-	-
Compressive Strength	MPa	96.0	-	-	-	-	-	-	-	-	-
Izod Notch	J/m		96	214	107	21	81	35	16	54	60.7
Hardness	Shore D		-	-	105	83	83	85	93	78	84

As mentioned previously, a region of the brain injured in a primary blast event appears to be the cerebral cortex on the side that faced the explosion.[7,19] However, the thicknesses of the different bones that make up the skull (i.e., facial bones and cranium) can vary between 5 mm and 8 mm. Not surprisingly, skull thickness has been shown to have a direct bearing on the biofidelity of the headforms used for ballistic impacts.[51] Preliminary tests performed using a mathematical model of a head subjected to a blast overpressure (i.e., 300 kPa) showed that the propagation of the blast wave through the brain varied as a function of the density and thickness of the bones (J.-S. Binette, personal communication). The testing done to date shows that the different thicknesses of the human skull should be simulated in designing the blast headform in order to elicit the most biofidelic response possible.

2.6.2 Regions and Properties of the Brain to Be Replicated

In addition to the brain itself, it may be necessary to model the soft tissues and fluids associated with the brain. Studies have shown that both the white and the grey matter of the CNS should be modelled as these cerebral tissues have unique properties.[52] Furthermore, the inclusion of the ventricles in any surrogate model assessing impact-related TBI is important as studies have shown that these cerebrospinal fluid-filled cavities may provide strain relief for the solid structures of the brain.[44] [53] Additionally, cerebral vasculature could influence the deformation response of the brain to impact as shown by Parnaik et al. [54]

While thicker skull bones afford protection against skull fracture and open head injury, the layer of cerebrospinal fluid that circulates in the subarachnoid space, delimited by the meninges, protects the brain from potential injury resulting from the brain impacting the inside of the skull (i.e., concussion). Some research has also shown that a slip, vs. a non-slip interface, between the brain and skull and an irregular shaped skull base provides more realistic brain kinematics despite predicting larger displacements than found *in vivo*.[44]

Another set of structures found in the brain that separate it into regions is the falx and tentorium. The falx and tentorium have been shown to reduce strains and inhibit movement of the brain inside the skull.[55] Despite being shown to have important characteristics in the complex structure of the brain there has been no mechanical property characterization for these structures, making them difficult to replicate. Researchers developing finite element brain models typically characterize these structures as an extension of the skull but they are really comprised of dura mater.

As stated previously, it is important to replicate as closely as possible the regions of the brain that are particularly susceptible to primary blast injury (see Section 2.4.1). Preliminary results using a mathematical model to assess the potential role of the folds of the cerebral cortex in the transfer of shock waves do not support the use of complex folding patterns in designing the brain (J-S Binette, personal communication).

Due to the lack of comparable data using primary blast-related studies, it is unclear which of the above-mentioned parameters should be modeled in a blast headform. For technical and manufacturing reasons, it may not be feasible to model all of these complex brain structures along with their unique properties. One can assume that a homogeneous brain surrogate material that provides an averaged response could lead to consistent behaviour under blast loading conditions. This approach has been used to create brain surrogates in previous ballistic or impact models.[56-60] For example, Sarron et al. used a cadaver skull filled with a synthetic material to evaluate the behind armour blunt trauma on the skull.[61] Room-temperature vulcanizing (RTV) silicone (Rhodorsil® 585 RTV, Rhodia, Cranbury, NJ) was cast into a skull to represent the cerebral contents. The main function of the RTV was to pot a hydrophone used to identify the occurrence of bone fracture and to measure the brain pressures and forces at discrete locations. Sarron et al. make no conclusions about the applicability of the RTV used and whether it was a successful synthetic brain representation.

Thali et al. [59] constructed a skin-skull-brain model to investigate gun shot wounds. The model comprised ballistic gelatin to simulate the brain in an artificial polyurethane skull covered with silicone to represent the scalp. Embedded in the silicone were synthetic fibres to simulate the collagen and

fat of the scalp. While this model was suitable for the assessment of gun shot wounds, its applicability to impact assessment is inappropriate based on other research efforts. Brands et al. [57,58] compared the viscoelastic behaviour of porcine brain tissues to those of edible bone gelatin and silicone gel.[58] The dynamic modulus of the gelatin was between 50 and 2000 times higher than that of the porcine tissue depending on the concentration of the gelatin solution used. Furthermore, the gelatin behaved as a nearly perfect elastic solid within the loading rates tested (0.1 to 16 Hz) and was too stiff to be used to simulate brain tissues. The silicone gel used was Sylgard® 527 A&B (Dow Corning, Midland, MI). This material's dynamic modulus was similar to that of the brain tissue for strains up to 1% and loading rates up to 10 Hz. For loading rates up to 260 Hz, the silicone gel became stiffer, thus resulting in lower strains being measured than those in the porcine brain tissue. While the silicone's response remained linear for strains up to 10%, it became non-linear at higher strains.

In a follow-up study, Brands et al. [58] extended the material characterisation of Sylgard® with additional shear and stress relaxation experiments. The shear tests that included material strains from 1% to 20% exhibited dynamic strains in the range of 258 Pa to 15.2 kPa at loading rates of 0.16 Hz and 1150 Hz respectively. The stress relaxation experiments revealed that Sylgard® behaves as a linearly viscoelastic solid for strains up to 50% and loading frequencies up to 461 Hz. It was also found that the porcine brain tissue exhibited a 30% shear softening at 20% strain and that its time-dependent behaviour did not depend on strain level.

In the previous two studies, it was found that the phase lag of the applied loading increased more for Sylgard® at frequencies above 1 Hz than the phase angle for brain tissue. For this reason, the silicone would not predict the response of brain tissue exactly if used to simulate brain tissue in a physical headform. Brands et al. [57,58] suggested in both studies that Sylgard® 527 could be used to simulate brain tissue in a mechanical head model to obtain a qualitative impression of the brain's response during impact loading, although the timing and the amount of strain may differ than that in real cerebral tissue. Based on failure patterns and brain tissue rate-sensitive characteristics, Zhang et al.[62] have also recently reported that Sylgard® gel is an appropriate simulant to enhance the understanding and mechanisms of ballistic brain injury. While there is no information in the literature on the transfer of shock waves through silicone gel such as Sylgard®, this material appears the best candidate to mimic brain matter.

3. Current Headforms Used In Blast Testing

Several headforms that have been used for the assessment of blast loading are briefly described in the following sections. A list of potential measurement transducers that can be incorporated into a blast headform to assess blast loading is also presented.

3.1 Hybrid III

The Hybrid III anthropomorphic test device (ATD) was developed by General Motors in 1973 for use in the evaluation of automotive occupant safety. The skull of the headform is manufactured from cast aluminum and the exterior skin is made from a vinyl rubber.

The exterior anthropometry of the Hybrid III headform is reasonably accurate from the basic plane upward. However, the headform does not include a representative neck or nape due to the presence of a flexible segmented neck. The mass characteristics of the headform are similar to those of a human providing good replication of rigid body head kinematics. However, the physical properties of the aluminum skull are not representative of those of a human skull, and therefore the transmission and reflection of a blast wave is not expected to be similar to that of a human head.

One of the main attributes of the headform is its durability even when subjected to large loading conditions. This durability permits testing of the headform in extreme conditions while ensuring confidence that the response of the headform will not change from test to test. Standard instrumentation is available to permit the measurement of linear and angular accelerations in three orthogonal directions. The Hybrid III headform also integrates with a biofidelic neck resulting in realistic head movement as a result of loading to the head.

The Hybrid III headform has been used to assess the blast mitigation properties of helmets in conjunction with various injury assessment functions such as HIC. A 50th percentile Hybrid III headform is shown in Figure 1.

Figure 1: Hybrid III Headform

3.2 Manikin for Assessing Blast Incapacitation and Lethality (MABIL)

MABIL was developed by DRDC Valcartier for the evaluation of new personal protection concepts developed for the protection against blast threats. MABIL consists of a solid urethane head, based on the Canadian Standards Association's (CSA) headform anthropometry, with detailed ear and facial features and a simplified torso representation (Figure 2).

The headform is instrumented with two pressure sensors, one in the mouth and one in the ear canal. It is also fitted with a photodiode situated in the eye to measure light intensity from the blast. The MABIL headform, however, does not have any means to assess the impact that is imparted to the head as a result of a blast. The MABIL program relied on the Hybrid III headform and neck for global body accelerations and impact measurement.

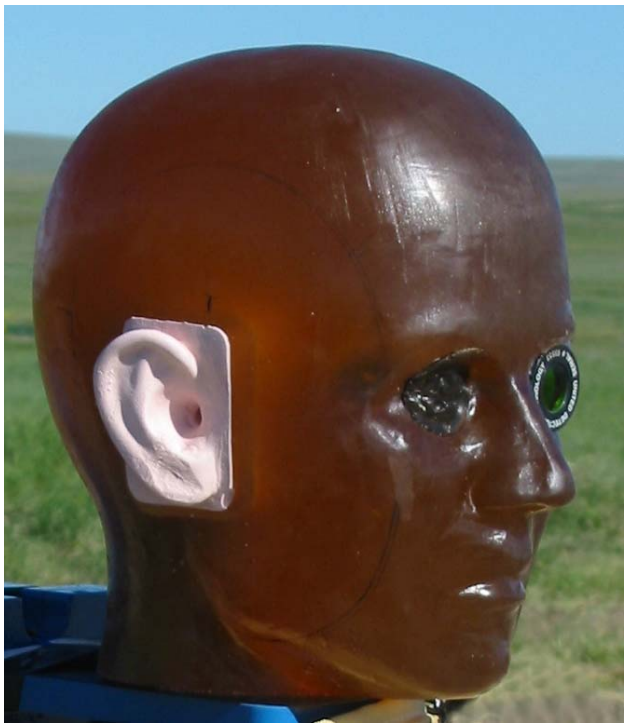

Figure 2: MABIL Headform

3.3 DERAMan Head

The Dynamic Event Response Analysis Man (DERAMan) head was designed and developed by researchers at Britain's Defence Evaluation and Research Agency.

The DERAMan head is constructed with similar geometrical features to that of a human head as it includes a skull and a soft gelatinous brain. The head is mounted on a compliant neck (see Figure 3). The head is fitted with 40 piezoelectric polymer pressure sensors located within the brain, 45 piezoelectric ceramic pressure sensors on the inside surface of the skull, two accelerometers and one three dimensional force gauge for a total of 90 inputs.

While the DERAMan head has been used in blast trials, results have yet to be released.

Figure 3: DERAMan Headform

3.4 Johns Hopkins Blast Headform

A headform is being developed for the assessment of head loading in a blast environment at the Applied Physics Laboratory of Johns Hopkins University, (J. Roberts, personal communication). However, no literature has been published on the headform at the time of this literature review.

3.5 Transducers for Assessing Headform Response

Other than the accelerometers which have been discussed previously in Section 2.4.5, several measurement transducers have been identified in a previous report for potential application in a surrogate blast headform with a gelatinous brain-simulating material[63]. The sensors were embedded in low durometer RTV and subjected to a high-speed, low mass projectile strike. Six of the seven sensors showed promise for use in a gelatinous medium and are listed in Table 2 below.

Table 2: Measurement transducers suitable for use in a gelatinous medium.

Manufacturer	Instrument	Model Number	Measurement Range	Acceleration	Local Strain	Global Strain	Pressure	Wave Propagation
PCB	Accelerometer	357A08	± 1000 g	✓				✓
Sonometrics	Displacement Transducer	n/a	n/a		✓	✓		
PCB	Pressure Sensor	138M160	0 – 69.0 MPa				✓	✓
FISO	Fibre Optic Pressure Sensor	FOP-M-PK-C2-F1-M2-R4-St	0 – 6.9 MPa				✓	✓
Dynasen Inc.	PVDF Pressure Sensor*	PVF2-11-25-EK	0 – 30.0 GPa				✓	✓
Dynasen Inc.	Piezo Pressure Pin*	CA-1135 – 0.5″ long	0 – 30.0 GPa				✓	✓
* requires calibration *in situ*								

4. Summary and Recommendations

A literature review was conducted to assess the state of knowledge surrounding head/brain injury related to blast loading. The injury mechanisms were categorized from primary to quaternary. Although none of the papers that were reviewed could say with certainty, the regions of the brain that are affected by primary blast include:

- the cerebral cortex on the side facing the blast;
- the vascular structure of the epithelium of the choroid plexus which is embedded in the cerebrospinal fluid of the ventricular system;
- the optic track when facing an oncoming blast;
- the pineal gland and cerebellar cortex; and,
- the temporal lobe and the brain stem with the temporal lobe being susceptible to axonal shearing.

The effects of primary blast forces on the head/brain can result in injuries that vary in severity from very low grade to lethal. The severity of injury depends on the magnitude of blast and a person's proximity to the blast origin. Surrounding structures, such as walls, must be considered in addition to proximity as they can have an amplifying effect on the blast forces.

For the purposes of the surrogate blast headform, only loading mechanisms directly involving the head will be considered. Blast loading of the thoracic region, for example, that results in a rise in pressure that may lead to intense venous backpressure in the brain resulting in a rupture of the blood vessels, will not be considered.

Kinetic energy transfer has been proposed as a direct mechanism of brain injury in which shockwaves traveling through the brain would result in pressure increases in the brain capillaries and neurons or the shock wave can induce a relative motion of the brain relative to the skull. In a surrogate headform these pressure increases or relative movements can be monitored with pressure sensors or shear/displacement crystals embedded in the brain medium.

DAI occurs more readily in the coronal plane and is related to angular acceleration of the head that occurs at the time of loading resulting in movement of the brain relative of the skull. Under these angular displacement conditions the falx and tentorium have been shown to greatly reduce the induced strains in the brain. The inclusion of the falx and or the tentorium must therefore be considered in the design of the surrogate blast headform for assessing injuries resulting from secondary and tertiary blast

effects. Impendence mismatch between these membranes and the brain tissue may also influence blast wave propagation.

Many different strategies have been used to assess the effects of blast waves on the brain. Techniques include measuring cognitive status, standard imaging and neurological evaluation techniques, as well as invasive sensor techniques and quantification of neurophysiological markers. Except for the use of sensors none of the techniques are suitable for use with the blast headform. Pressure sensors embedded in the brain of rats have been used to study the movement of blast waves through the brain. Although no threshold for injury was proposed it is believed that the integral over time of the pressure pulse, resulting in impulse, is a key factor in altering the properties of neurons. This method also represents a way to evaluate energy transfer to the brain and is easily incorporated into a surrogate headform.

Accelerometers are yet another sensor that can easily be incorporated in a headform. These sensors have been used for decades to evaluate the potential for closed head injury in car crashes. Although the impulses in the automotive environment are of much longer duration than the blast environment, a headform's acceleration under blast conditions is an effective measurement for assessing the insult imparted to the head during secondary and tertiary blast effects and should be incorporated into the blast headform design.

In addition to the pressure sensors and accelerometers, a transducer that may be suitable for measuring both local and global strain has been identified (displacement transducer: Sonometrics Corporation, London, ON) and will be evaluated in a blast loading environment.

A proposed mechanism of injury in blunt impact or blast wind events considers the relative movement of the brain relative to the skull. To effectively represent this movement, the irregular geometry of the skull base must be included in the headform design. Similarly to the falx indicated above, the geometry of the skull base can affect the response of the skull/brain interaction when subjected to rotational acceleration. This specific movement may be technically difficult to measure. However, shear/displacement crystals embedded in the brain and fixed to the inside of the skull may be able to discern these relative movements.

A blast shock wave must first travel through the cranium before interacting with the brain. Thus, it is important that the materials selected to simulate the skull bone have mechanical and impedance properties comparable to those of the human skull. Additionally, the varying thickness of the skull, which has been shown to have a direct bearing on the headform biofidelity, must also be considered in the fabrication of the surrogate head. Mathematical modelling

has indicated that it may not be necessary to simulate the softer tissues of the scalp for primary blast loads.

Studies have suggested that it may be important to represent both the white and grey matter of the CNS and that the ventricles should be included as they have a mitigating effect on the induced strains. Additionally, the cerebral vasculature could influence the deformation response of the brain while the layer of cerebrospinal fluid that circulates in the subarachnoid space can pad the brain from impacts with the skull. Until such time that the practical, technical and manufacturing limitations are overcome, it may not be feasible to model all of these complex brain structures along with their unique properties. Therefore, for the purposes of the initial blast headform development, a homogeneous brain surrogate material can be adopted with the objective of providing an averaged brain response to blast loading. Several researchers have used this approach to create brain surrogates.

A material that has commonly been used to simulate the brain under shock loading is Dow Corning's silicone gel Sylgard® 527 A&B. Although silicone is not biofidelic under all loading conditions, this material would provide a qualitative assessment of the brain response to impact loading. While no information was found regarding the transfer of shock waves through the Sylgard®, this material appears the best candidate to mimic brain matter, and is recommended for use in the fabrication of the blast headform.

The body of knowledge regarding the brain's response to blast loading is limited, and the mechanisms by which brain injuries are caused are a burgeoning field of study. Although the current literature review has provided some initial guidelines for the development of a blast headform, additional research is required to assess head/brain characteristics that would enhance the biofidelity of a blast headform. Suggested areas of research are listed below:

- Mechanical characterization of both human brain tissues and potential surrogate brain tissues under blast loading conditions (i.e. strain magnitude, phase and relaxation).
- Identification of injury mechanisms, levels of injury severity and threshold and the underlying physical mechanisms contributing to the injury outcome.
- Studies to correlate injury outcome with the physical response of the head/brain to blast insult.
- Test programs to evaluate the blast surrogate headform, throughout its development, under blast conditions and where possible validate its response.

- Identification of headform characteristics (e.g., inertia and center of gravity location) would improve the blast headforms kinematic response to secondary and tertiary blast loading.
- Assessment of the effects of the neck interface with the headforms response. This would include proper location of the occipital condyle and joint resistance.

Additional sources of information that may be applicable to the design of a blast headform and need to be investigated further are:

- Reports on blast studies previously prepared under projects granted by the Technical Support Working Group. These reports have not been made available for this review.
- Reports to be prepared under the Defence Advanced Research Projects Agency's Prevent Blast Program. These projects seek to characterize insult with injury mechanisms and outcome.
- Publications from the Applied Physics Laboratory's at Johns Hopkins University describing their efforts in developing a blast headform.

The mechanism(s) by which brain injuries are caused by exposure to blast is a relatively new field of study. It is still not fully understood how a blast shock wave might damage head structures such as cerebral tissues or blood vessels. However, the effects of cerebral tissue disruption in survivors can be devastating with chronic headaches, sensitivity to light or noise, impaired memory, reductions in problem-solving abilities, and behavioural changes. A better understanding of the effects of blast overpressure on the head is therefore required in order to design a test device capable of evaluating the performance and assisting in the design of improved head protection systems.

Current head surrogates and test methods were developed to assess the performance of protective devices designed to address head injury mechanisms associated with direct contact of the head with solid objects or high inertial loads as a result thereof. They do not consider blast overpressure effects on the head. There are a few injury criteria for non-contact acceleration injuries based on the linear and rotational acceleration. Although these criteria may provide insight into some aspects of blast-induced brain injury, they do not capture the effects of shock wave propagation through the structures of the brain. The development of a new headform for the study of blast loading is therefore warranted.

5. References

1. Ambum B. Brain Injuries Lead Iraq War Injuries. *United Press International*, 2004.

2. Jaffe G. An Army Surgeon Says New Helmet Doesn't Fit Iraq. *The Wall Street Journal*, 2004.

3. Jontz S. More Head Injuries in Afghanistan, Iraq Push Improvements in Protective Gear. *Stars and Stripes*, 2004.

4. Okie S. Traumatic Brain Injury in the War Zone. *New England Journal of Medicine* 2005;352(20):2043-2047.

5. Wagner C. Brain Injuries High Among Iraq Casualties. *ARNEWS: Army News Services*, 2003.

6. Cernak I, Wang Z, Jiang J, Bian X, Savic J. Cognitive Deficits Following Blast Injury-Induced Neurotrauma: Possible Involvement of Nitric Oxide. *Brain Injury* 2001;15(7):593-612.

7. Taber KH, Warden DL, Hurley RA. Blast-Related Traumatic Brain Injury: What is Known. *Journal of Neuropsychiatry and Clinical Neuroscience* 2006;18(2):141-145.

8. Nelson TJ, Clark T, Stedje-Larsen ET, Lewis CT, Grueskin JM, Echols EL, et al. Close Proximity Blast Injury Patterns from Improvised Explosive Devices in Iraq: A Report of 18 Cases. *The Journal of Trauma, Injury, Infection, and Critical Care* 2007;62:1-6.

9. Finkel MF. The Neurological Consequences of Explosives. *Journal of the Neurological Sciences* 2006;249(1):63-67.

10. Stuhmiller JH, Phillips YY, Richmond DR. The Physics and Mechanisms of Primary Blast Injury. In: Zatchuck R, Jenkins DP, Bellamy RF, Quick CM, editors. *Textbook of Military Medicine. Part 1. Warfare, Weapons, and the Casualty,* . Washington DC: TMM Publications, 1990:241-270.

11. Mayorga MA. The Pathology of Primary Blast Overpressure Injury. *Toxicology* 1997;121(1):17-28.

12. DePalma RG, Burris DG, Champion HR, Hodgson MJ. Current Concepts: Blast Injuries. *The New England Journal of Medicine* 2005;352(13):1335-1342.

13. Argygros GJ. Management of Primary Blast Injury. *Toxicology* 1997;121(1):105-115.

14. Warden D. TBI During Wartime: The Afghanistan and Iraq Experience. Washington, DC: Walter Reed Army Medical Center, 2006:36.

15. Yilmaz S, Pekdemir M. An Unusual Primary Blast Injury* Traumatic Brain Injury Due to Primary Blast Injury. *American Journal of Emergency Medicine* 2007;25(1):97-98.

16. Sylvia FR, Drake AI, Wester DC. Case Report: Transient Vestibular Balance Dysfunction after Primary Blast Injury. *Military Medicine* 2001;166(10):918-920.

17. Stein M, Hirshberg A. O:Medical Consequences of Terrorism. The Conventional Weapon Threat. *Surgical Clinics of North America* 1999;79(6):1537-52.

18. Leibovici D, Gofrit ON, Stein M, Shapira SC, Noga Y, Heruti RJ, et al. Blast Injuries: Bus versus Open-air Bombings--a Comparative Study of Injuries in Survivors of Open-air versus Confined-space Explosions. *Journal of Trauma, Injury, Infection, and Critical Care* 1996;41(6):1030-5.

19. Saljo A, Bao F, haglid KG, Hansson H-A. Blast Exposure Causes Redistribution of Phosphorylated Neurofilament Subunits in Neurons of the Adult Rat Brain. *Journal of Neurotrauma* 2000;17(8):719-726.

20. Yehuda R. Linking the Neuroendocrinology of Post-traumatic Stress Disorder with Recent Neuroanatomic Findings. *Seminar Clinics of Neuropsychiatry* 1999;4(4):256-65.

21. Kaur C, Singh J, Lim MK, Ng BL, Yap EP, Ling EA. Studies of the Choroid Plexus and its Associated Epiplexus Cells in the Lateral Ventricles of rats following an Exposure to a Single Non-penetrative Blast. *Archives od Histology and Cytology* 1996;59(3):239-48.

22. Petras JM, Bauman RA, Elsayed NM. Visual System Degeneration Induced by Blast Overpressure. *Toxicology* 1997;121(1):41-9.

23. Kaur C, Singh J, Lim MK, Ng BL, Ling EA. Macrophages/microglia as 'Sensors' of Injury in the Pineal Gland of Rats following a Non-penetrative Blast. *Neuroscience Research* 1997;27(4):317-22.

24. Kaur C, Singh J, Lim MK, Ng BL, Yap EP, Ling EA. The Response of Neurons and Microglia to Blast Injury in the Rat Brain. *Neuropathology and Applied Neurobiology* 1995;21(5):369-77.

25. Kaur C, Singh J, Lim MK, Ng BL, Yap EP, Ling EA. Ultrastructural Changes of Macroglial Cells in the Rat Brain following an Exposure to a Non-penetrative blast. *Annals Academy of Medicine Singapore* 1997;26(1):27-9.

26. Suneson A, Axelsson H, Hjelmqvist H, Medin A, Persson JKE. Physiologic Changes in Pigs Exposed to a Blast Wave from a Detonating High-Explosive Charge. *Military Medicine* 2000;165(2):119-126.

27. Jontz S. Head Injuries Push Improvements in Gear. *Stars and Stripes-European Edition* 2004.

28. Scott SG, Belanger HG, vanderploeg RD, Massengale J, Schloten J. Mechanism-of-Injury Approach to Evaluating Patients With Blast-Related Polytrauma. *The Journal of the American Osteopathic Association* 2006;106(5):265-270.

29. *BLAST & IED Health Effects and Injury Mitigations Symposium*. Toronto, ON, Canada, 2006.

30. Cernak I, Savic J, Ignjatovic D, Jevtic M. Blast Injury from Explosive Munitions. *The Journal of Trauma, Injury, Infection, and Critical Care* 1999;47(1):96-103.

31. Mellor SG, Cooper GJ. Analysis of 828 Servicemen Killed or Injured by Explosion in Northern Ireland 1970-84: the Hostile Action Casualty System. *British Journal of Surgery* 1989;76(10):1006-10.

32. Cernak I, Malicevic Z, Prokic V, Zunic G, Djurjevic D, Ilic S, et al. Indirect Neurotrauma Caused by Pulmonary Injury: Development and Prognosis. *International Review of the Armed Forces Services* 1997;70:114-120.

33. Cernak I, Wang Z, Jiang J, Bian X, Savic J. Ultrastructural and Functional Characteristics of Blast Injury-Induced Neurotrauma. *The Journal of Trauma, Injury, Infection, and Critical Care* 2001;50(4):695-706.

34. Knudsen SK, Oen EO. Blast-Induced Neurotrauma in Whales. *Neuroscience Research* 2003;46(3):377-386.

35. Cernak I, Savic J, Malicevic Z, Zunic G, Radosevic P, Ivanovic I, et al. Involvement of the Central Nervous System in the General Response to Pulmonary Blast Injury. *The Journal of Trauma, Injury, Infection, and Critical Care* 1996;40(3 Suppl):S100-4.

36. Suneson A, Hansson HA, Seeman T. Pressure Wave Injuries to the Nervous System Caused by High-energy Missile Extremity Impact: Part II. Distant Effects on the Central Nervous System--a Light and Electron Microscopic Study on Pigs. *The Journal of Trauma, Injury, Infection, and Critical Care* 1990;30(3):295-306.

37. Axelsson H, Hjelmqvist H, Medin A, Persson JK, Suneson A. Physiological Changes in Pigs Exposed to a Blast Wave from a Detonating High-explosive Charge. *Military Medicine* 2000;165(2):119-26.

38. Chavko M, Koller WA, Prusaczyk WK, McCarron RM. Measurement of Blast Wave by a Miniature Fiber Optic Pressure Transducer in the Rat Brain. *Journal of Neuroscience Methods* 2007;159(2):277-281.

39. Marguiles SS, Thibault LE, Gennarelli TA. Physical Model Simulations of Brain Injury in the Primate. *Journal of Biomechanics* 1990;23(8):823-836.

40. Neal CJ, Bell RS, Armonda R, Ling GSF, Ecklund JM, Moores L, et al. Craniocerebral Trauma Evacuated from Operation Iraqi Freedom: A Report from Two Stateside Military Hospitals. Washington, D.C.: National Capital Neurosurgery Consortium, 2005.

41. Raina AK, Baheti M, Haldar A, Ramulu M, Chakraborty AK, Sahu PB, et al. Impact of Blast Induced Transitory Vibration and Air-Overpressure/Noise on Human Brain- an Experimental Study. *International Journal of Environmental Research* 2004;14(2):143-149.

42. Kodama T, Hamblin MR, Doukas AG. Cytoplasmic Molecular Delivery With Shock Waves: Importance of Impulse. *Biophysical Journal* 2000;79(4):1821-1832.

43. Medin AL. Blast Overpressures, Gases and Fumes from a HEAT Warhead Caused Temporary Incapacitation to the Crews of Two Attacked Armoured Personnel Carriers. An Experience from the Swedish UN Mission in Bosnia 1994. *FOA-R-97-00626-310--SE* 1997:27 p.

44. Ivarsson J, Viano DC, Lovsund P. Influence of the Lateral Ventricles and Irregular Skull Base on Brain Kinematics Due to Saggital Plane Head Rotation. *Journal of Biomechanical Engineering* 2002;124(4):422-431.

45. Biermann P, Ward E, Cain R, Carkhuff BG, Merkle A, Roberts J. Development of a Physical Human Surrogate Torso Model for Ballistic Impact and Blast. *Journal of Advanced Materials* 2006;38(1):3-12.

46. Jussila J, Leppaniemi A, Paronen M, Kulomaki E. Ballistic Skin Simulant. *Forensic Science International* 2005;150(1):63-71.

47. Lynnerup N. Cranial Thickness in Relation to Age, Sex and General Body Build in a Danish Foensic Sample. *Forensic Science International* 2001;117(1-2):45-51.

48. Hubbard RP. Flexure of Layered Cranial Bone. *Journal of Biomechanics* 1971;4(4):251-263.

49. McElhaney JH, Fogle JL, Melvin JW, Haynes HR, Roberts VL, Alem NM. Mechanical Properties of Bone. *Journal of Biomechanics* 1970;3(5):495-511.

50. van den Bosch HLA. Chapter 2: The Deformable Headform. *Crash Helmet Testing and Design Specification*. Eindhoven, The Netherlands: University Press, 2006:23-34.

51. Viano DC, Bir C, Walilko T, Sherman D. Ballistic Impact to the Forehead, Zygoma, and Mandible: Comparison of Human and Frangible Dummy Face Biomechanics. *The Journal of Trauma, Injury, Infection, and Critical Care* 2004;56(6):1305-11.

52. Zhang L, Yang KH, King AI. A Proposed Injury Threshold for Mild Traumatic Brain Injury. *Journal of Biomechanical Engineering* 2004;126(2):226-236.

53. Ivarsson J, Viano DC, Lovsund P, Aldman B. Strain Relief from the Cerebral Ventricles During Head Impact: Experimental Studies on

Natural Protection of the Brain. *Journal of Biomechanics* 2000;33(2):181-189.

54. Parnaik Y, Beillas P, Demetropoulos CK, Hardy WN, Yang KH, King AI. The Influence of Surrogate Blood Vessels on the Impact Response of a Physical Model of the Brain. *Stapp Car Crash Journal* 2004;48:259-77.

55. van den Bosch HLA, Leensen MWBM, Klomp NHM, Sauren FAAHJ, Wismans JSHM. ASME, editor. Development of an Improved Dummy Head for Use in Helmet Certification Tests. ASME Symposium on Crashworthiness, Occupant Protection and Biomechanics in Transportation; 2000; Orlando, FL. ASME.

56. Thali MJ, Kneubuehl BP, Zollinger U, Dirnhofer R. The "Skin-Skull-Brain Model": A New Instrument for the Study of Gunshot Effects. *Forensic Science International* 2002;125(2-3):178-189.

57. Brands DWA, Bovendeerd PHM, Peters GWM, Wismans JSHM, Paas MHJW, van Bree JLMJ. SAE, editor. Comparison of the Dynamic Behavious of Brain Tissue and Two Model Materials. 43rd Stapp Car Crash Conference; 1999; San Diego, CA.

58. Brands DWA, Bovendeerd PHM, Peters GWM, Wismans JSHM. Stapp, editor. The Large Shear Strain Dynamic Behaviour of In-Vitro Porcine Brain Tissue and a Silicone Gel Model Material. Stapp Car Crash Conference; 2000; Atlanta, GA. SAE.

59. Thali MJ, Kneubuehl BP, Dirnhofer R. A "Skin-Skull-Brain Model" for the Biomechanical Reconstruction of Blunt Forces to the Human Head. *Forensic Science International* 2002;125(2-3):195-200.

60. Thali MJ, Kneubuehl BP, Zollinger U, Dirnhofer R. A Study of the Morphology of Gunshot Entrance Wounds, in Connection with their Dynamic Creation, Utilizing the "Skin-Skull-Brain Model". *Forensic Science International* 2002;125(2-3):190-194.

61. Sarron J, Dannawi M, Faure A, Caillou J, Da Cunhal J, Robert R. Dynamic Effects of a 9mm Missile on Cadaveric Skull Protected by Aramid, Polyethylene or Aluminum Plate: An Experimental Study. *The Journal of Trauma, Injury, Infection, and Critical Care* 2004;57(2):236-243.

62. Zhang J, Yoganandan N, Pintar FA, Guan Y, Gennarelli TA. Experimental Model for Civilian Ballistic Brain Injury Biomechanics Quantification. *Journal of Biomechanics* 2006.

63. Fournier E, Bayne T, Shewchenko N. Review of Instrumentation for Blast Headform. Ottawa: Biokinetics and Associates Ltd., Report No. R06-15, March 23, 2006.

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6. List of Symbols, Abbreviations, Acronyms and Initials

ATD	anthropomorphic test device
CNS	central nervous system
CSA	Canadian Standards Association
CT	Computed Tomography
DAI	diffuse axonal injury
DERAMan	Dynamic Event Response Analysis Man
DRDC	Defence Research Development Canada
EEG	electroencephalogram
GCS	Glasgow Coma Scale
HIC	Head Injury Criterion
IED	improvised explosive device
MABIL	mannequin for assessing blast incapacitation and lethality
MRI	magnetic resonance imaging
MTBI	mild traumatic brain injury
RPG	rocket propelled grenade
RTV	room temperature vulcanizing
TBI	traumatic brain injury
TNT	trinitrotoluene

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Publishing: DRDC Valcartier 2459 Pie−XI Blvd North, Québec, Quebec, G3J 1X5
Performing: Biokinetics and Associates Ltd.
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2. SECURITY CLASSIFICATION (Overall security classification of the document including special warning terms if applicable.)

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3. TITLE (The complete document title as indicated on the title page. Its classification is indicated by the appropriate abbreviation (S, C, R, or U) in parenthesis at the end of the title)

Blast Headform Development − Literature Review − (U)
(U)

4. AUTHORS (First name, middle initial and last name. If military, show rank, e.g. Maj. John E. Doe.)

Ed Fournier, David Sullivan, Tim Bayne, Nicholas Shewchenko, Lucie Martineau

5. DATE OF PUBLICATION (Month and year of publication of document.)

November 2007

6a NO. OF PAGES (Total containing information, including Annexes, Appendices, etc.)

35

6b. NO. OF REFS (Total cited in document.)

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7. DESCRIPTIVE NOTES (The category of the document, e.g. technical report, technical note or memorandum. If appropriate, enter the type of document, e.g. interim, progress, summary, annual or final. Give the inclusive dates when a specific reporting period is covered.)

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SDA05−181

9b. CONTRACT NO. (If appropriate, the applicable number under which the document was written.)

W7701−062565/001/QCL

10a. ORIGINATOR'S DOCUMENT NUMBER (The official document number by which the document is identified by the originating activity. This number must be unique to this document)

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(U) Changes to the threat types, their exposure rates and improvements to protective equipment have changed the injury patterns that are seen in the battlefield, with brain injuries becoming more prevalent. Although a considerable number of publications exist that address the effects of blast injury to the lungs, the mechanism(s) by which brain injuries are caused by exposure to blast is largely unknown. It is still not fully understood how a blast shock wave might damage head/brain structures such as cerebral tissues or blood vessels. A better understanding of the effects of blast overpressure on the head is required such that a surrogate headform can be produced for the design and evaluation of head protective systems for blast loads.
A literature review was conducted to further the general understanding of blast effects on cerebral tissues and provide insight into possible headform assessment methods that can be used to correlate surrogate headform responses to real brain injuries. Based on the findings of the literature review, it is anticipated that a surrogate blast headform and its associated injury criteria will be based on detailed measurements of wave propagation through soft tissue surrogates and other structures that are represented within the surrogate headform.

(U) Une évolution des menaces posées par les armes à effet de souffle, une augmentation de leur taux d'exposition, de même que des améliorations du matériel de protection du personnel militaire ont changé le type de lésions observées sur les champs de bataille, avec des blessures traumatiques au cerveau devenant de plus en plus répandues. Bien qu'il existe un nombre considérable de publications qui traitent des lésions d'effets de souffle primaires au poumon, les mécanismes par lesquels l'onde de choc causerait de telles blessures au cerveau sont peu connus. En fait, il n'a jamais été établi avec certitude comment ces ondes endommageraient des structures de la tête et/ou du cerveau telles que les tissus ou les vaisseaux sanguins cérébraux. Une meilleure compréhension des effets du souffle sur la tête est essentielle afin de permettre la production d'un modèle de tête pour effet de souffle qui permettrait l'élaboration et l'évaluation des systèmes de protection de la tête humaine exposée à différentes charges explosives.
Une revue détaillée de la littérature a été effectuée afin de bien comprendre les effets du souffle sur les tissus cérébraux et d'identifier les méthodes possibles d'évaluation de modèles de tête pour effet de souffle qui seront utilisées pour corréler les réponses de ces modèles aux blessures cérébrales typiques vues lors d'explosions. Sur la base des résultats de cette revue de littérature, on prévoit qu'un modèle de tête pour effet de souffle et ses critères de blessures associés seront basés sur des mesures détaillées de la propagation de l'onde de choc à travers les substituts synthétiques de tissu mou et autres structures qui sont représentés dans le modèle.

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(U) surrogate headform; brain injury; skull; mechanism of blast injury

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