# Behind Helmet Blunt Trauma: Pathophysiological understanding and relevance of specifying helmets

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**Abstract.** A helmet is one of the most important elements of a soldier's personal protective equipment (PPE). Its primary purpose is to protect the head from fragments and bullets, and secondary, to protect against blunt impacts. In France, as well as other countries, helmets are still specified to prevent ballistic penetrating traumatic brain injuries (TBI) and attenuate blunt trauma. The pathophysiology of this type of blunt TBI, described as behind helmet blunt trauma (BHBT), remains scarcely studied.

In the first part of this study, we provide a detailed overview of the basic pathophysiology of TBI and review current literature on the specific pathophysiological consequences of BHBT, which range from superficial skin abrasions to extensive skull and brain damage. The severity of these injuries depends on the mechanical properties of the projectiles, as well as those of the helmet, which theoretically absorbs part of the energy at impact, thereby attenuating TBI.

In the second part of this study, we discuss the mechanical phenomena (acceleration, rotational motion, etc.) happening on impact, which lead to BHBT-related injuries. Then, we provide a review of literature on the various methods used to quantify these mechanisms through multiple criteria, the use of physical surrogates – instrumented or not – and numerical models. Some of these methods are currently employed by certain countries to assess the protecting capabilities of helmets against BHBT.

Finally, based on this review, we critically assess the relevance of these specifications, considering the architectural constraints they impose on the system and their actual protective effect.

## 1. INTRODUCTION

Nowadays, combat helmets are designed to stop 9 mm bullets. But by doing so, the energy of the bullet is transmitted to the helmet, causing a deformation of its shell. Even though this bullet is not a realistic threat on military ground, it is still used to specify helmets, as it allows us to compare them. Up until now, military helmets were not able to stop bullets from shoulder weapons. However, new helmets have been developed with the aim of stopping this kind of threat. In addition, to improve helmets, their mass must be reduced without compromising the protection. In order to do so, the tendency is to switch the shell's material, from aramid to ultra-high molecular weight polyethylene (UHMWPE), which has a lower areal density (AD) than the former. This implies an increase of the backface deformation (BFD), which in turn raises the risk of behind helmet blunt trauma (BHBT), therefore explaining why evaluating the BFD seems necessary. This is even more important to take into consideration now that shoulder weapons are becoming a tested threat.

Loftis et al. reported 92 989 US civilians injured at the head from 2013 to 2015 [1]. They highlight the fact that even if penetrating wounds are not so common (6%), they lead to complex injuries (involving skull, meninges, brain and vessels), which tend to be particularly lethal (54% of them). On the other hand, blunt injuries are more common (90%), but less lethal (3%) of them. Plaisted reports that among the 400 000 US soldiers who suffered traumatic brain injuries (TBI) due to blunt impacts (car accident, fall, parachuting, etc.) between 2000 and 2019, 83% presented mild TBI, 10% presented moderate TBI, 1% presented severe TBI and only 1.3% presented a penetrating wound [2]. This reflects the fact that head injury is still a major threat for the soldier's health, therefore emphasizing the importance of protecting the head.

Regarding French feedback on injured soldiers, over an 11-year period (2012-2023, covering conflicts such as operations Pamir in Afghanistan (2002-2014), Barkhane in Sahel (2014-2022) and Chammal in Iraq and Syria (since 2024)), 408 soldiers were listed as injured, and 52 (13%) of them succumbed to their wounds [unpublished personal data]. Regarding those 52 soldiers, 7 (13%) of them only had an injury on the head and neck area, and 25 (48%) others suffered at least an injury on the head and neck area. Even though blast injuries are the most common threat on modern battlefields, accounting for 87 recorded skull injuries, ballistic impacts still resulted in 18 skull injuries. This data highlights that bullets remain a relevant threat and that head injuries must be carefully

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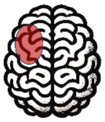
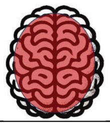
considered, especially since the number of soldiers who avoided a head injury thanks to their helmet is not given in this study. In addition, this feedback comes from a context of asymmetric conflicts, which means that the epidemiology could differ significantly in high intensity conflicts. Furthermore, since French helmets from this feedback were made of aramid, those numbers regarding injured and dead soldiers could have been higher if helmets were made of UHMWPE, assuming that the BFD generates major head injuries. However, it remains unknown whether ballistic head injuries which happened while wearing a helmet were due to bullet penetration or BFD, therefore questioning if BFD presents a significant injury risk. This brings up the question of whether or not current helmet specifications remain relevant. Indeed, this requirement seems to hinder mass reduction, which is an important aspect nowadays, especially as helmets are increasingly equipped with accessories such as night vision goggles with their batteries and communication headsets for example, all of which significantly add to the total weight. For instance, a special operations helmet's shell, which weighs approximately 700-800 g, may reach around 3 kg when fully equipped. In fact, some countries might remove this specification after observing no injury - or only marginal cases - due to BFD.

Therefore, the aim of this study is to provide a detailed overview of the basic pathophysiology of TBI as well as the specific pathophysiological consequences of BHBT, and to discuss the mechanical phenomena (acceleration, rotational motion, etc.) happening on impact, which lead to BHBT-related injuries. The aim is to critically assess the relevance of the current helmet's specifications.

## 2. ANATOMY AND PATHOPHYSIOLOGY OF TRAUMATIC LESIONS OF THE HEAD

Head trauma can lead to various types of injuries. Broadly, they are divided into traumatic lesions of the skull and soft tissues from TBI, which include primary injuries (direct consequence of the trauma on brain tissues) and secondary injuries (resulting from secondary pathophysiological mechanisms such as impaired cerebral blood flow and inflammation reactions). Although this work excludes penetrating ballistic injuries, BHBT also includes open injuries. In addition, head injuries are often associated with maxillo-facial trauma. This review will focus on skull injuries, TBI, and/or a combination of both. These different lesions are summarized in Table 1.

**Table 1.** Definitions of the main type of head injuries

| Category | Type of Injury | Description |
|---|---|---|
| **Superficial lesions**<br> | Scalp contusions / cephalhematoma<br>Lacerations of the scalp | Contusion or hematoma of the scalp, without skin opening<br>Breaching of the skin, generally resulting un massive bleeding |
| | Skull fracture<br>Depressed fracture<br>Open fracture<br>Skull base fracture | Simple (one fracture line) or comminuted (multiple fracture lines) without vault deformation<br>Comminuted fracture with sinking vault<br>Fracture associated with scalp laceration<br>Can result in dural opening with nasal or ear cerebrospinal fluid leak |
| **Focal brain injuries**<br> | Penetrating TBI | Breaching of the scalp, skull, and dura mater, exposing the brain directly to the outside |
| | Cerebral Contusion | Localized haemorrhagic injury of the brain parenchyma, often caused by direct impact |
| | Epidural Hematoma | Accumulation of blood between the dura mater and the skull, usually due to arterial rupture |
| | Subdural Hematoma | Accumulation of blood between the dura mater and the arachnoid, often caused by venous rupture |
| | Subarachnoid Haemorrhage | Presence of blood in the subarachnoid space, which may result from traumatic brain injury |
| **Diffuse brain injuries**<br> | Cerebral Oedema | Focal or diffuse excessive accumulation of fluid in the brain, increasing the intracranial pressure |
| | Concussion | Temporary alteration of brain function without visible structural injury |
| | Diffuse Axonal Injury | Damage to brain axons caused by acceleration-deceleration forces, and secondary injuries, leading to prolonged coma |

### 2.1 Skull and soft tissues injuries

Blunt head traumas are frequently associated with skull and soft tissues injuries. Soft tissues are highly vascularized, meaning that lacerations can often result in profuse bleeding requiring prompt haemostasis. Skull fractures increase significantly the risk of an intracranial hematoma [3]. It is important to distinguish blunt trauma from penetrating TBI which associates lacerations at the same site of soft tissues, skull fracture and the dura matter, exposing the brain parenchyma to the outside.

### 2.2 Traumatic brain injuries

TBI correspond to a structural injury and/or functional impairment of the brain resulting from a trauma [4].

#### *2.2.1 Primary Brain Injuries*

Primary brain injuries are classified into focal and diffuse injuries, which often occur together. They are most commonly observed in moderate and severe TBI.

Focal brain damage results from lacerations, compression, concussion and shearing forces, at the primary impact site (coup). Damage may develop in tissues opposite to the impact (contrecoup) or near the coup site due to the secondary impacts when the brain rebounds against the skull [5]. In addition to the transmitted energy of the impact, cavitation, which corresponds to the vaporization of water at a constant temperature due to reduced pressure, has also been proposed as another mechanism of focal lesion in blunt TBI [6]. This hypothesis suggests that a rapid increase in acceleration may lead to an increase of the intracranial pressure (ICP) at the impact location and a decreased pressure at the opposite side of the cerebrum (contrecoup), which can cause the development of cavitation bubbles. When they burst or collapse, they can create local tissue damage [7–9]. However, this hypothesis has not been confirmed by any clinical data to date.

In contrast, diffuse brain injuries primarily result from non-contact forces of rapid deceleration and acceleration, causing shearing and stretching injuries to neuronal axons, glial cells, and blood vessels. If diffuse insults, such as blast TBI, are a major source of diffuse brain injuries, focal impacts can also lead to these types of consequences. The diffuse axonal injury (DAI) represents the main diffuse structural brain injury. It is characterized by widespread lesions in subcortical and deep white matter, often involving the frontal and temporal white matter, corpus callosum, and brainstem. These structural and functional impairments in neurons lead to various clinical consequences, including bilateral neurological deficits and/or delayed awakening from coma, categorized according to the Adams classification. The severity of axonal injury and neuronal degeneration significantly impact long-term TBI prognosis [10]. The majority of DAI in TBI does not result from a direct lesion of the axons at the time of primary injury, but from a secondary injury mechanism [11]. Diffuse brain injuries also lead to a delayed traumatic chronic encephalopathy, a neurodegenerative disease [12].

#### *2.2.2 Secondary Brain Injuries*

Secondary brain injuries arise from mechanical, biochemical, cellular, and physiological changes that begin during the primary injury and can extend from hours to years afterward. Those injuries include excitotoxicity, mitochondrial dysfunction, release of reactive oxygen species and lipid peroxidation, neuroinflammation, axonal degeneration, glial scar and apoptotic cell death [13,14].

Post-traumatic intracranial hypertension (ICH) is the primary driver of secondary brain injuries. It is defined as an increase in ICP above 20 mmHg, resulting from an imbalance between the volumes of the intracranial steady space (the cranium is a rigid box) and its components (brain parenchyma, cerebrospinal fluid (CSF), and cerebral blood volume) [15]. Due to the exponential relationship between intracranial volume and pressure [16], additional volumes from primary injuries and secondary injuries result in an exponential increase in ICP. It may potentially lead to a vicious circle: ICH worsen secondary injuries (in particular cerebral oedema) which, in turn increase ICH. ICH results in two main processes. Firstly, pressure gradient within the cranio-spinal cavity can lead to displacement of brain parenchyma (cerebral herniations) which can lead to severe cerebral impairment. In particular, the compression of the brain stem can result in cardiac and or respiratory arrest. Secondly, the increase in ICP progressively reduces the cerebral perfusion pressure (CPP = Mean Arterial Pressure - ICP), impairing oxygen and nutrient delivery to neurons, promoting secondary brain injuries, and leading to brain death when cerebral blood flow (CBF) is severely compromised.

While the severity of TBI is related to the severity of the impact, there is no direct correlation between primary and secondary injuries [5]. Thus, head trauma can result in moderate primary brain injuries but severe secondary brain injuries.

Clinically, TBI can manifest as a reduced level or loss of consciousness, altered mental state, memory loss, neurologic deficits (persistent or transient), and/or intracranial lesions. TBI severity is classified based on clinical symptoms, in particular the consciousness level, as assessed using the Glasgow Coma Scale (GCS) which ranges from 15 (normal consciousness) to 3 (deep coma). A GCS ≤ 8 corresponds to a coma [17]. TBI is considered mild when GCS ≥13 [18] (sometimes considered ≥14), moderate when 9 ≤ GCS ≤ 12, and severe when GCS ≤ 8 [19,20].

The severity of head injury can be classified according to the Abbreviated Injury Scale (AIS), a tool used in severe trauma settings to quickly assess and communicate the overall severity of trauma and expected clinical outcomes (see Table 2).

**Table 2.** AIS classification in head injuries [21,22]

| AIS | Head injury | Lesions |
|---|---|---|
| **1** | Headache or dizziness | Superficial lesions |
| **2** | Unconscious less than 1 h | Closed fracture |
| **3** | Unconscious 1 - 6 h | Comminuted and/or depressed fracture |
| **4** | Unconscious 6 - 24 h | Open skin (skin opening), massively depressed fracture, small focal lesions |
| **5** | Unconscious more than 24 h | Penetrating TBI (dural opening), large focal lesion, diffuse lesions |
| **6** | Non-survivable | Massive destruction of both cranium (skull), brain, and intracranial contents (crush) |

## 3. CLINICAL CONSEQUENCES OF BHBT

Literature regarding mechanisms and clinical consequences of military BHBT is extremely scarce. BFD can lead to several types of injuries, including skull fractures, focal brain injuries and diffuse brain injuries.

Key factors can significantly influence skull responses. The predominant fracture types in BHBT are linear and depressed, influenced by factors including impact velocities, contact areas, and loading regions [22,23]. For instance, broader contact areas typically result in linear fractures, while more localized impacts often cause depressed fractures [24]. In terms of impact severity, broad contact areas larger than 13 cm² generally lead to linear fractures, whereas smaller, localized impacts usually result in depressed fractures [24]. A study on helmeted post mortem human subjects (PMHS) impacted by 9 mm bullets showed that impacts at 460 m/s primarily produced linear fractures, while impacts with velocities around 440 m/s can lead to both linear and depressed fractures [25].

Focal brain injuries likely result mainly from mechanical loading conditions. Both skull deformations and relative skull-brain motions can induce focal brain injuries, although their precise contribution in the primary mechanism of the blunt ballistic impacts remains unclear. These injuries typically occur directly beneath the impact site. Skull deformation generates compressive stresses in the brain, leading to injuries such as cerebral contusions and haemorrhages [26]. Skull fractures may also rupture meningeal vessels, potentially causing epidural hematomas. Post-impact skull movement can induce additional injuries (contrecoup) due to skull-brain collisions because of inertia and inflict additional injuries [27]. The precise contribution of skull deformation versus relative motion to injuries is not yet clear.

The specific mechanisms for diffuse injuries in BHBTs are not well-defined, necessitating more comprehensive research. Changes in ICP during impacts are probable critical indicators of TBI severity but has not been precisely studied in military BHBT, and establishing a direct correlation between initial ICP changes and injury severity in humans remains extremely challenging. Mechanical stress enhances the vulnerability of neurons to secondary injuries, even in cases of mild TBI [28].

## 4. HELMET EVALUATION REGARDING BHBT

### 4.1 Biomechanics of the head

Biomechanics is the study of a biological system using mechanics. Regarding TBI, biomechanics consists in studying the head's movements and mechanical loads applied to the brain, in order to predict head injuries based on the mechanical phenomena [29].

Upon impact, dynamic mechanical forces act on the skull and the brain, generating both linear and rotational movements of the head (see Figure 1). The overall acceleration of the head can be described using three-dimensional linear and rotational accelerations, measured at its centre of gravity [29]. Linear acceleration compresses the brain against the skull at the opposite side of the impact location. This creates a volume variation of the brain tissue, and consequently, intracranial pressure fluctuations, and eventually cavitation, which can cause brain injuries. Rotational acceleration, on the other hand, puts the brain tissue in rotation, generating internal shear, as different regions undergo varied inertial movements due to local density variations, which can also cause brain injuries [30,31, 32,33].

The mechanism behind diffuse injuries in BHBT, especially DAI and cerebral haemorrhages, probably result from a combination of angular and linear accelerations. The relative contribution of linear and rotational

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accelerations resulting from an impact depends on multiple factors, including the direction and location of the force, and the material properties of the skull and brain [29]. If the force vector of an impact passes through the head's centre of gravity, it will mainly induce a linear movement of the head. However, if the force vector does not pass through the head's centre of gravity, it will generate an impulsive moment, leading to both a linear and rotational acceleration of the head [29]. Angular accelerations resulting from high-energy projectiles with incidental impact may be the predominant mechanism, rather than linear accelerations, in causing certain focal lesions, particularly subdural hematomas, as a result of the tearing of bridging veins [34,35].

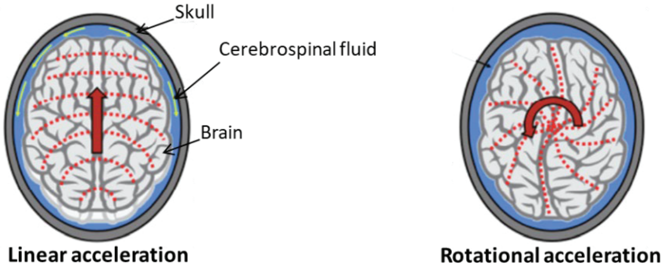

**Figure 1.** Brain's movement when exposed to a linear acceleration (left) or rotational acceleration (right) [31]

The head's mechanical response to loading can vary depending on the phenomenon's frequency content. The brain has a viscoelastic behaviour [36]. The skull can be considered as a rigid shell for an impact frequency content below 150 Hz. However, over 200 Hz, the skull exhibits a vibratory response that can increase or modify the skull flexure patterns produced by the force [37]. Gurdjian et al. stated that head accelerations recorded opposite to the blow do no not correlate with rigid body acceleration in short duration impacts, sometimes differing by a factor greater than 2 [37]. This can explain the differences observed between long duration impacts (low frequency) and short duration impacts (high frequency) [38]. Therefore, it is complicated to compare the long duration impacts studied in the literature, like road accidents which have a duration of around 10 ms, with ballistic impacts, which generally last less than 5 ms [39–42]. Furthermore, impact velocities in ballistic cases are way higher than those in road accidents.

Since brain injuries can result from different types of solicitation, defining a single injury criterion for the head, representative of all the different types of brain injuries, remains challenging [36].

### 4.2 Head surrogates

Currently, various standards attempt to assess the injury risk of BHBT in ballistic testing on helmets. Among those, the AEP 2920(A)(V2) proposes to mount helmets on a rigid headform filled partly with BackFace Signature (BFS) materials such as Roma Plastilina #1, but the tolerated indent depth (residual deformation) shall be set by the National Authority (NA). The French ministry of defence (MoD) requires the use of HERBIN SUEUR 40 plastilina with a specification on the indent depth [43]. This remains under the scope of the AEP 2920(A)(V2) which allows the NA to use other BFS materials. According to the VPAM HVN 2009, a headform made of ballistic soap shall be used and the maximum energy transferred to the head, which is calculated based on the ratio between the indent depth made by the ballistic impact and the indent depth made by the calibration test, shall not exceed 25 joules.

These methods can be used to compare the protection level of different helmets regarding BHBT, but they present some limitations. Indeed, not only the ability of these types of surrogates to represent the injuries that can occur on a human are greatly questioned, but they also don't take into consideration the head's dynamic response. OP't Eynde et al. studied the fidelity of the Roma Plastilina #1 and concluded that the rebound effect of the plastilina causes a diminution of the final deformation of 5 to 25% [44].

To address these limitations, another type of surrogate can be used, which can be instrumented with sensors at the centre of gravity of the surrogate to record the three-dimensional linear and rotational accelerations endured by the head. Indeed, the NIJ 0106.01 proposes to use an instrumented headform made of magnesium to monitor the linear acceleration which must not exceed 200-g. The Hybrid III (HIII) head, developed initially by General Motors (USA) for crash tests, is one of the surrogates that are mainly used [45]. It was designed from biometric data collected on 13 cadavers whose head circumferences were between 56 et 58 cm [46]. The Ballistic Load Sensing Headform (BLSH), designed by Biokinetics (CAN) specifically for ballistic helmet testing, is another type of surrogate that can be used to assess skull fracture risks due to backing effects. Other surrogates exist, but they are mostly used for non-ballistic impacts. The differences between the main surrogates found in the literature for ballistic testing are presented in Table 3.

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Headforms' size, mass and moments of inertia are important to take into consideration and having those similar to the real values is important for the experimental values to be exploitable. Some of them exist in different sizes, but the most commonly used size is the one corresponding to the 50th percentile male, which is available for the HIII and BLSH headforms. The ATLAS headform is modular and the Pk17dynA headform was originally an XXL size (645 mm), but is now also available for helmets in L-XL sizes (425 mm). Regarding the mass, the HIII headform weighs 4.54 kg, the BLSH headform weighs 4.91 kg, the ATLAS headform is rigid, as it is fixed on a solid neck, and the Pk17dynA weighs 7.84 kg (including instrumentation devices).

**Table 3.** Example of different surrogates used to measure the accelerations and / or force

| | Developer | Initial purpose | Material | Instrumentation | Ref. |
|---|---|---|---|---|---|
| **HIII** | General Motors (USA) | Crash tests | Aluminium and vinyl skin | Acceleration / velocity sensors | [45] |
| **BLSH** | Biokinetics (CAN) | Ballistic testing on helmets | Magnesium alloy (and skin pads on force sensors) | Acceleration / velocity sensors, + Force sensors | [47] |
| **ATLAS** | John Hopkins Applied Physics Laboratory | Ballistic testing on helmets | Polymer headform, steel impact cap, neoprene impact pad | Force sensors | [48, 49] |
| **Pk17dynA** | University of German Federal Armed Forces, Bundeswehr Research Institute for Materials, Institute of Legal Medicine of Munich | Ballistic testing on helmets | Polyamide headform. Sensors covered by curved steel plates | Force sensors | [50, 51] |

## 4.3 Injury criteria

### *4.3.1 Main injury criteria*

All of the surrogates discussed previously have the same objective, which is to measure a mechanical phenomenon upon impact on a helmeted surrogate, whether it is the accelerations and / or velocities and / or force. The signals recorded are then used to determine different injury criteria, presented in Table 4, to offer insight on different aspects of head injury risks.

The most basic criteria that can be calculated are peak translational acceleration (PTA), peak translational velocity (PTV), peak rotational acceleration (PRA), and peak rotational velocity (PRV).

The most commonly used criteria are the Head Injury Criterion (HIC) and the Brain Injury Criterion (BrIC), which come from the automotive industry. The HIC, created in 1971, is based on the linear accelerations measured at the head's centre of gravity, and comes from studies on PMHS. It succeeded the Wayne State Tolerance Curve (WSTC), which was created in 1960, as a result of the first studies done on PMHS in car accidents [29]. The WSTC is a graph that shows the tolerance threshold of a linear acceleration depending on the time of exposure. Indeed, an impact with a high acceleration can be endured for a shorter duration compared to an impact with a lower acceleration, before it becomes critical. The HIC was used by the National Highway Traffic Safety Administration (NHTSA) at first in 1972, in order to evaluate the injury risk of automotive crash tests [52]. The BrIC was invented in 2013, in order to study the rotational phenomenon of the impact, by taking into consideration the rotational velocities. Therefore, the HIC represents the linear phenomenon of the impact, while the BrIC represents the rotational phenomenon.

The Head Impact Power (HIP), created in 2000, is a mix between the HIC and the BrIC, as it takes both aspects (linear and rotational) into consideration. It is representative of the power transmitted to the head, given in kW [53].

The Blunt Criterion, developed in the early 2000s as well, is used by the U.S. Department of Defence in order to quantify the vulnerability of ballistic impacts [54]. It takes into consideration the kinetic energy of the projectile and its diameter, as well as the target's capability to tolerate the impact energy, by considering its weight and thickness. This criterion is not relevant here as it only takes into consideration the initial parameters, and not the phenomena observed upon impact, and is therefore independent of the helmet tested.

The impact force is another relevant physical parameter, which could be used to predict the risk of skull fractures, but can only be obtained with a headform that has force sensors to record the force time curves, like the BLSH.

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Other criteria exist, based on brain tissue deformation instead of head kinematics, like the maximum principal strain (MPS), the cumulative strain damage measure (CSDM), the dilatation damage measure (DDM), and the relative motion damage measure (RMDM). The MPS and CSDM are used for the evaluation of diffuse axonal injuries, while the MPS and DDM are used to evaluate contusion, and the RMDM is used to assess subdural hematoma [55]. However, those criteria are computed, which means they come from simulation and cannot be determined with experimental tests.

**Table 4.** Injury criteria for assessing the risk of head injuries

| Criterion | Formula | Unit | Main injury studied | Ref. |
|---|---|---|---|---|
| Peak Translational Acceleration | $PTA = \max\left(\sqrt{{a_x}^2 + {a_y}^2 + {a_z}^2}\right)$ | g *or* m/s$^2$ | Skull fracture | [54] |
| Peak Translational Velocity | $PTV = \max\left(\int \left(\sqrt{{a_x}^2 + {a_y}^2 + {a_z}^2}\right) dt\right)$ | m/s | / | / |
| Peak Rotational Velocity | $PRV = \max\left(\sqrt{{\omega_x}^2 + {\omega_y}^2 + {\omega_z}^2}\right)$ | rad/s | Diffuse axonal injury | [56] |
| Peak Rotational Acceleration | $PRA = \max\left(\frac{d}{dt}\left(\sqrt{{\omega_x}^2 + {\omega_y}^2 + {\omega_z}^2}\right)\right)$ | rad/s$^2$ | Subdural hematoma | [56] |
| Force | $F = \sum F_i$ *where i is the number of sensors* | N | Skull fracture | [57] |
| Blunt Criterion | $BC = \ln\left(\frac{\frac{1}{2} \times m \times v^2}{W^{1/3} \times T \times D}\right)$ | / | Skull fracture | [58] |
| Head Injury Criterion | $HIC = \left[\frac{1}{(t2 - t1)} \int_{t1}^{t2} a_r \, dt\right]^{2,5} (t2 - t1)$<br>$where\ a_r = \sqrt{{a_x}^2 + {a_y}^2 + {a_z}^2}$ | / | Skull fracture, Brain injury, Concussion | [59] |
| Brain Injury Criterion | $BrIC = \sqrt{\left(\frac{\omega_x}{\omega_{xc}}\right)^2 + \left(\frac{\omega_y}{\omega_{yc}}\right)^2 + \left(\frac{\omega_z}{\omega_{zc}}\right)^2}$ | / | Diffuse axonal injury | [60] |
| Head Impact Power | $HIP = \sum m.a.v + \sum I.\alpha.\omega$ | kW | Concussion | [53] |

To obtain the different criteria, the recorded signals need to be filtered before they are used in the equations. The filtering process is a fundamental aspect, which is rarely mentioned according to the bibliographic researches made for this publication. Regarding the HIC for example, the cut-off frequencies have been established from sport and automotive impacts. The duration of these impacts is much longer and the deformation velocities are less important than for ballistic impacts, which implies a much higher frequency content for the latter. The use of filtering values that are based on long duration impacts may potentially underestimate the real effect in ballistic impacts. This would mean that the values of the filter cut-off frequency need to be increased. In addition, the resonance frequency of the system composed of the surrogate head and the helmet also needs to be taken into consideration.

The linear accelerations are filtered with a CFC (Channel Frequency Class) 1000 [36, 62]. The angular velocities are sometimes filtered with a CFC 180 for motorcycle helmets [62], and sometimes with a CFC 600 according to the HFM-271 [36]. Human Factors and Medicine (HFM) are NATO groups working on defence science and technology activities. Regarding the HIP, Newman et al. filtered the signals with a CFC 1000, and later re-filtered digitally with a CFC 180 [53]. According to the Biokinetics software, the force signals obtained with the BLSH seem to be filtered with an equivalent to a CFC 2166. No other publication was found explaining which filters were used and why.

### *4.3.2 Injury criteria thresholds*

In order to be able to use these criteria, a tolerance threshold has to be determined. The helmet can be considered having a sufficient protection level when the value obtained for a criterion is under the threshold.

However, the thresholds used usually come from long duration impacts, and can therefore be questioned as those types of impacts are very different from ballistic ones. This question can also be raised regarding the criteria's formulas. For instance, the HIC formula corresponds to the integral of a linear approximation for a time duration impact between 5 and 50 ms of the WSTC log-log plot [61]. The slope of this approximation might be different under 5 ms.

For the acceleration of the head, apart from the NIJ 0106.01 which specifies a threshold of 200-g, as previously discussed, the European standard for motorcycle safety ECE 2206 specifies a threshold of 275-g tolerance for the head, which corresponds to an AIS of 5, which is quite high [62].

For the HIC and the BrIC, the thresholds are determined as 10% of AIS 2+ by the HFM-271 [36]. Thus, the HIC's threshold used for protection of mounted soldiers is 250 (Figure 2), which is the value recommended as a concussion threshold for football players, and is lower than the one used in crash tests (700) [36,63]. HFM-271 proposes a threshold of 0.27 for the BrIC when the signals are filtered with a CFC 600 [36].

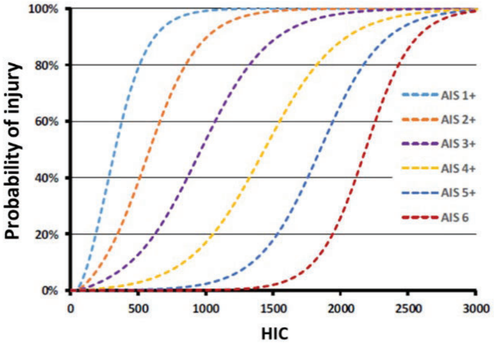

**Figure 2.** Probability of injury depending on the HIC and the AIS [36]

There is no official threshold regarding the HIP. Newman et al. found that there is a 50% probability of concussion for a HIP of 12.8 kW, based on 12 cases involving 24 football players, which have been re-enacted [53]. Marjoux et al. re-enacted 61 accidents, including pedestrian, motorcyclist and footballer accidents. They found that a 50% risk of skull fracture was associated to a HIC of 667 and a HIP of 38 kW, a 50% risk of subdural hematoma was associated to a HIC of 1 429 and a HIP of 55 kW, and that a 50% risk of moderate neurological injury (AIS 3-4) was associated to a HIC of 533 and a HIP of 24 kW [64]. Once again, these values do not come from a ballistic context, and it is therefore questionable whether or not they can be used for the evaluation of combat helmets.

Several thresholds have been proposed for the force applied to the skull, depending mainly on the age of the subject, but on the impact's location on the skull as well. Raymond et al. proposed a force threshold, on the parietal region, where fractures have a 50% probability of appearing, at 5 970 N, based on impact tests performed on PMHS at low velocities (20-35 m/s) [22]. Anctil et al. set this 50% probability of fracture threshold at 4 256 N for the temporal region [65]. Bolduc and Anctil proposed, in a study on the BLSH headform, that 6.0 kN corresponds to a 25% risk of skull fracture, identified as a threshold for head blunt trauma associated with ballistic impact, and a force of 7.2 kN for a 50% risk of skull fracture [66]. Finally, regarding the standards and as summarized by Philippens [67], FprCEN/TR16148:2010 [68] specifies a force of 5 to 8.5 kN for the fracture threshold. EN397 and EN12792 specify respectively 5 kN and 10 kN against the same threat: 5 kg spherical impactor at 1 m/s impact velocity on a helmeted rigid mounted metal headform. In a literature review from Li et al., they reported that the use of a non-lethal projectile generates a loss of consciousness for a force between 2.5 kN and 5 kN, and meningeal injuries for a force between 5 and 7.5 kN [69]. The variability in those thresholds highlights the need for further research to establish robust injury risk models.

### 4.4 Numerical simulation

Simulation can be useful, for different reasons. First, numerical simulation is more affordable than real ballistic tests. Also, a numerical model can represent the several parts of the head more precisely than the physical surrogates. Simulation avoids the use of animals or PMHS, that are usually representative of an older population than soldiers and causes ethical issues. Furthermore, one can have access to metrics that cannot be available experimentally, such as internal skull energy (see Figure 3). Finally, this solution enables parametric studies to better understand the consequences of ballistics impacts on (un)helmeted heads.

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The first ballistic helmet simulation studies, such as the one conducted by Tham et al., aimed to demonstrate the feasibility of reliably modelling the dynamic deformation of the shell impacted by a 9 mm FMJ bullet [70].

Some, like Palta et al., used simulation as an intermediate verification step. If BFD values are consistent with and without a clay headform, the modelling is deemed reliable, allowing for parametric research [71].

Simulation has also enabled the evaluation of configurations incorporating headform simulators (like HIII and BLSH headforms) and digitally biofidelic human head surrogates. For instance, Miranda-Vicarion et al. establish relationships between BHBT and impact velocity [72], while Tan et al. analysed the effects of selecting between impact-absorbing foams and suspension nets on peak skull acceleration [41]. Caçoilo et al. examined the influence of impact angle and projectile energy on the Head Injury Criterion (HIC) and found that a 0° impact is the most severe [73].

The use of biofidelic headforms allows a better understanding of injury mechanisms. Aare and Kleiven analysed the forces transmitted to the skull and brain deformations depending on the shell material and impact angle [42]. Li et al. quantified the influence of standoff, thickness and pad hardness on ICP and HIC, showing that increasing the standoff by 2 mm can reduce intracranial pressure by up to 50% in their case [74]. Deck et al. also demonstrated that the probability of skull fracture significantly decreases with a slight increase in standoff. Their parametric study with a 9 mm bullet found that skull fracture probability drops from 100% at 22.5 mm to 22% at 23 mm, while the risk of DAI decreases from 86% at 19 mm to 6% at 20 mm [40]. Li et al. conducted a review on finite element method (FEM) models of biofidelic heads used for non-ballistic impacts (blunt trauma), ballistic impacts, and blast exposures, highlighting their diversity [75].

However, anatomical models remain complex to develop, limiting their widespread use. In addition, to specify helmets, ballistic testing is still necessary, and simulation cannot be sufficient by itself.

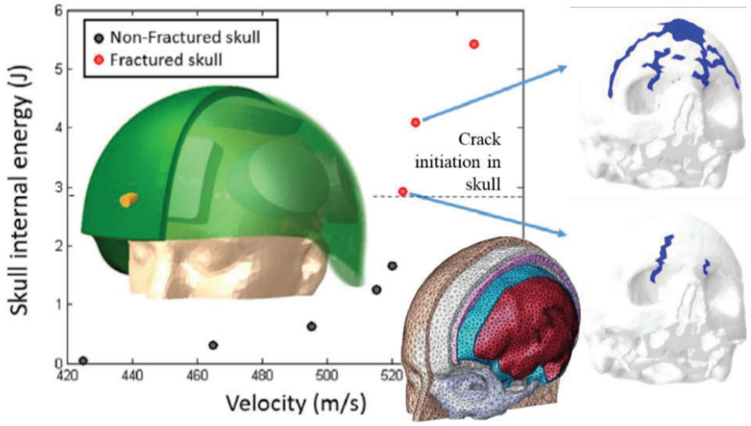

**Figure 3.** Example of skull fractures simulation using a model derived from MRI scans, triggered by a ballistic impact on a helmet [71]

## 5. DISCUSSION

Theoretically, protection from BHBT is important to take into account in helmet specifications, especially with the worldwide integration of UHMWPE as a protective material instead of para-aramid. However, there is not a lot of clinical feedback regarding the current protection, in particular on whether head injuries on the battlefield are mainly due to penetrating bullets or to BHBT.

Studies currently conducted by the French MoD with different types of helmets and headforms highlight the multiplicity of parameters that consistently influence the ability of helmets to protect the head against BHBT. These factors must be thoroughly understood and controlled to effectively minimize the BHBT against different types of projectiles, up to the 7.62 mm bullet. Some of these parameters are the standoff distance [76], the impact location (frontal/occipital/lateral), the presence of a pad at the impact location, the surrogate used, and other parameters directly linked to the helmet tested, such as the material used, the thickness of the helmet, its mass and the friction coefficient.

Clay headforms are convenient for assessing a helmet's ballistic protection due to their affordability and ease of use. However, evaluating the ability of a helmet to minimize BHBT solely by measuring the BFS in clay headforms is insufficient, as it does not account for the dynamic response of the head, which can lead to various types of injuries.

Instrumented headforms offer a more relevant method for determining real injury risks. Dynamic phenomena measured during a ballistic impact test on helmeted instrumented headforms can be used to determine different injury criteria which can be associated to various types of injuries, to a certain extent.

Despite their advantages, instrumented headforms are relatively expensive and require multidisciplinary knowledge for effective use. Moreover, the correlation between the residual measurements in a clay headform, which is yet the only standardized material to assess BHBT, and the dynamic response of instrumented headforms is not straightforward [43]. Additionally, the method for filtering the data before calculating the different criteria significantly impacts the results, and it is crucial to dissociate high-velocity (ballistics) from low-velocity (road accidents) impacts, as they have distinct characteristics. Some filtering values, criteria formulas and associated thresholds currently used are derived from the automotive field and may need reconsideration for ballistic applications. Furthermore, the variety of injury assessment metrics and thresholds poses challenges in establishing definitive criteria, indicating a need for more standardized research approaches to define injury thresholds more accurately. This inconsistency arises from differences in skull material behaviour, mechanical properties across various regions of the skull, biological variations between specimens, and test conditions (the projectile, helmet if there is one, and surrogate used). The lack of identical impact conditions in biomechanical tests further complicates the processing of the results.

Therefore, it seems important to consider the different studies conducted regarding this subject, to have a more complete approach in determining injury criteria and their thresholds, like in the literature review done by Nsiampa and Coghe [77]. In another review on the mechanisms and research methods for blunt ballistic head injury, Li et al. stated that to their knowledge no existing headform is suitable for the study of both skull and brain injuries [69].

From a clinical perspective, the transposition of laboratory tests to current protection from brain injuries appears hazardous. Firstly, the links between different criteria and specific types of superficial injuries (such as soft tissue and skull injuries) and primary brain injuries are difficult to confirm. Under the same loading conditions, consequences can vary significantly, and for the same mechanism, clinical outcomes can be very different. For example, a significant impact on the frontal bone, associated with a depressed fracture, can result in a mild TBI, whereas a lighter impact resulting in a simple closed fracture on the temporal bone can lead to a severe TBI with catastrophic epidural hematoma. Secondly, the role of helmets in preventing secondary brain injuries appears even more challenging to assess, as these injuries result from indirect pathophysiological mechanisms and are not necessarily linked to the primary injuries. A moderate primary injury can lead to severe secondary injuries, depending on associated injuries, particularly in haemorrhagic polytrauma, which may result in ischemia with major secondary brain injuries.

Military personnel are also particularly exposed to other types of threats like the blast overpressure (primary blast) coming from high-explosive ammunitions (IED, Artillery shell, etc.) but also from cumulative effect of low intensity blast overpressure coming from allied weapons systems (artillery canons, mortars, etc.). As the former can cause direct injuries, the latter can cause secondary injuries from repetitive mild TBI, resulting in deficits in cognition, memory, sleep, vision, and hearing, which cannot be studied with the same methods and materials than for BHBT [78]. The need to protect the soldiers against this threat can bring incompatibilities with the specifications regarding the protection against ballistic impacts [79].

## 6. CONCLUSION

Epidemiological data indicates that BHBT is a serious threat to soldiers. Understanding the blunt injury mechanisms caused by the BFD remains crucial to improve their prevention. However, assessing the clinical consequences of BHBT is challenging due to the interactions between various complex pathophysiological mechanisms. While it is possible to describe wounds post-injury, predicting the type of wounds that may occur from a non-penetrating impact remains very difficult.

To predict this risk, standardized plastilina headforms offer affordability and ease of use but do not account for dynamic phenomena. In contrast, instrumented headforms can measure accelerations, velocities, and impact forces to study the dynamic response of the head and determine various injury criteria. However, the lack of correlation between these injury criteria and actual clinical consequences, the numerous factors involved in their calculation, and the reliance on low-velocity impact thresholds make it difficult to use these injury criteria to specify helmet effectively in a ballistic case.

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