Effects of torso and head protection from blast overpressure on intracranial biomechanics

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Blast-related traumatic brain injuries (bTBI) become of major concern among active soldiers and veterans. The mechanisms underlying bTBI have been largely studied, but there are still diverging hypotheses in the literature. So far, these mechanisms have been divided into two categories: direct (effect of the shock wave on the brain) and indirect (remote effects from torso or acceleration of the head itself). The indirect components are the most controversial, even though they are supported by numerous animal studies. They include, among other phenomena, blast wave transfer to the brain through the vascular and cerebrospinal compartments. Since the mechanisms responsible for blast wave transfer from the thorax and abdomen to the brain are still unknown, it is difficult to determine if ballistic protection for the torso may protect soldiers from bTBI, or not. To begin understanding how adding torso ballistic protections would change brain overpressure kinetics during blast wave exposure, a representative large animal model was exposed to a blast overpressure load, important enough to cause a clinical response and generate a moderate to severe pulmonary and intestinal injuries. We used adult female Large-White swine equipped with sensors to collect data without affecting their physiology or increase risks of death. Animals were divided into three groups: 1) no torso or head protection against blast overpressure corresponding to the wearing of soft body armour protection alone, 2) torso protection only and 3) head protection only. Animals in the protected torso groups were fully protected by adding to the soft pack the ceramic plates at the torso and pelvic levels. The helmeted group was defined with the head enclosed in a rigid box whereas wearing the soft body armour only.
The blast threat used was characterized by a 460 ± 60 kPa side-on peak, with a 2.2 ± 0.4 ms peak duration and a 240 ± 40 kPa.ms impulse, which corresponds to a 50% risk of mortality on Bass' injury risk curve. Our results indicate that torso protection decreased the duration of blast overpressure in the oesophagus and reduced intracranial pressure, as compared to both the unprotected and soft armour protected groups. Head protection diminished the duration of the overpressure wave in the oesophagus and the maximal vascular and intracranial pressures and impulses, and increased the duration of vascular and intracranial overpressures. Taken together, these data give the first tendencies observed on large mammals resulting from direct blast exposure in free field which will be helpful for understanding of the mechanisms involved in the bTBI. Several avenues have been identified for future studies such as assessing the main characteristics of the threat involved, in order to update existing criteria or propose new ones and, ultimately, establish a strategy for developing new military armour that would protect soldiers against bTBI.

1. INTRODUCTION

Blast-related traumatic brain injury(bTBI) results from blast exposure during combat or training and are of major concern among the military. The estimated incidence of TBI in soldiers and veterans varies among studies [1-2]. BTBI incidence is likely to be underestimated, due to difficulties in reporting and documenting cases in combat theatre settings [3]. The main cause of military bTBI is the exposure to the blast event and its shock wave [1-2;7]. Sudden and transient increase in intracranial pressure during the travel of the shockwave and brain motion relative to the skull are expected to be the main causes of brain damage [8-9]. Few studies suggest that body protections can exacerbate the effects of the blast wave on the body, and particularly at the head level. Indeed, helmets [4] or ballistic vests [5, 6] can reinforce energy transfer of the blast wave. Therefore, military protections might need to be updated and optimized to be adapt to the blast threat.

Although there is no consensus about the mechanisms underlying bTBI, they have been divided into two main categories: direct and indirect [2-3;8;10-12]. Direct mechanisms include propagation of the blast wave through the cranium and orbital and/or oral openings [2;8;10;13]. The skull provides little protection against blast wave and skull flexure seems to be involved in its transmission to the cranial content [4;13-14]. Once the blast wave has passed the cranium, it may cause high-frequency contraction and relaxation of brain parenchyma and blood vessels, particularly resulting in damage of blood vessel walls and haemodynamic abnormalities [2]. *In silico* experiments suggest that the shockwave can also be transmitted to the brain from flexure of the skull, which creates localized regions of low pressure

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waves [4]. Progression of the shockwave through orbital and/or oral openings has also been evidenced, and is associated with direct damage to ocular neurons. However, the involvement of this mechanism in TBI is controversial [7][8]

Indirect mechanisms are more controversial than direct mechanisms, but they have been supported by studies done in animal models [7;11-12]. Blast overpressure has also been reported to cause macroscopic translational and rotational acceleration of the brain, resulting in TBI similar to those caused by head impact [7]. Cernak et al. [11] proposed a mechanism in which the kinetic energy of the overpressure wave is transferred to the central nervous system and impacts brain tissue. Other proposed another mechanism in which the kinetic energy is transferred through the vascular system and cerebrospinal fluid [7;13;15]. The blast wave on the thorax would suddenly increase the pressure on the walls of large blood vessels, which would accelerate blood. With no valves to regulate blood ascension to brain, it would easily result in increased intracranial blood pressure leading to rupture of the capillaries [7;15].

Although they are controversial, indirect mechanisms are to be considered when developing military protections. Indeed, incidence of TBI has increased with the improvement of body armors and higher efficiency of thoracic protection [1]. It was proposed that soldiers wearing protections may get closer to the center of the blast and thus, may be exposed to higher blast levels. Alternatively, Kevlar vests may facilitate brain damage by increasing intrathoracic pressure [6;13]. The aim of our study was to test this latter hypothesis. For this purpose, a representative biological model (bodyweight, layout of organs, thickness of the chest wall which are as close as possible to the human body) was exposed to an explosive load. Adult female swine were used, with subgroups wearing different body armors conferring different levels of protection. Few swine were equipped with both the lower-level body protection and a helmet. Swine were equipped with sensors to collect holistic data relevant to blast overpressure kinetics, and wave transmission to the body, especially to the brain, without affecting its physiology or increasing risk of death.

2. METHODS

2.1 Threat characteristics and setup

The threat corresponded to a blast overpressure exposure under free-field conditions. A sphere of 4kg of Hexomax B2269B (Eurenco, France), moulded into a polystyrene shell, was placed on a cardboard cylinder at a corresponding height of burst of 39 cm above a concrete slab. Indeed, due to the distance from the charge fixed to 3 m range, the targets experienced the Mach stem regime of the blast, i.e. an ideal Friedlander wave, to simplify the threat profile and consequently our understanding (Figure 1A). The level was strong enough to cause sufficient pulmonary injuries, consistent with clinical and morphological changes. Sizing was chosen referring to the Bowen tolerance curves [16] revisited by Bass et al. [17] to achieve the 50% survival curve. Hence, a 400-kPa peak overpressure and 2.5-ms positive-phase duration were targeted. For comparison purposes, the targets were subjected to blast in pairs (Figure 1B). Each of them was accompanied at equal distance and height of a piezoelectric pencil probe 137A22 (PCB Piezotronics, United States) for recording the incident pressure-time history. The aim was to be able to follow both kinematics of the ribs and pressures which required invasive approach and short monitoring period, with, in the same time, an animal lightly instrumented monitored longer for others clinical and biochemical parameters trends.

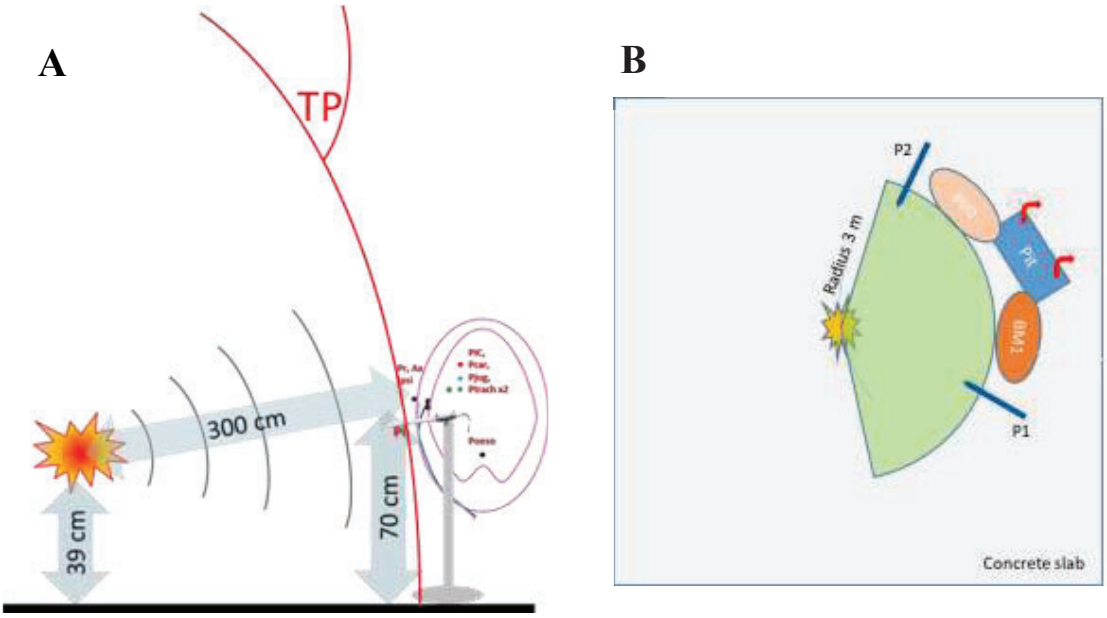

Figure 1. A): charge and targets distances (instrumented model and pencil probe for the reference incident pressure); the red line describes the Mach stem and its triple point passing around 2 m high. B): Overview of the scene near the pit: BM1 and BM2 exposed simultaneously with their reference incident pressure probes P1 and P2 at 3 m range from the charge

2.2 Model and instrumentation

Twenty-eight female Large-White swine (51 ± 4 kg weight; 118±6 cm length) were deeply anesthetized and prepared in accordance with the European directive 2010/63/EU on the protection of animals used for scientific purposes. They were adequately instrumented for cardio-respiratory monitoring with multi-parameter monitors (Propaq CS Monitor, Welch Allyn, United States) and Biopac modules (Biopac Systems, United States). Data were recorded on site from beginning at 15 minutes before blast initiation to 60 minutes after blast. Two distinct instrumentation protocols were used:

- strongly instrumented animals, for which a basic set of clinical parameters allowed to monitor cardiorespiratory functions and arterial blood pressure was used and reinforced by a set of more invasive instrumentation allowed to manage with physical data such as transient pressure surges through: large vessels (jugular vein or carotid artery), brain parenchyma (ICP), and with kinematics of the chest wall (acceleration, velocity, displacement of the rib). The monitoring time was limited to one hour on site after explosion.
- lightly instrumented animal model, for which the basic set of clinical parameters only was essential, allowed us to extent clinical monitoring period up to 6 hours after explosion in an operating room.

At the end of the sequences, animals were sacrificed by exsanguination under anesthetic overdose and autopsies were carried out for scoring cerebral, torso and abdominal injuries.

2.3 Protections and configurations tested

Table 1 below shows the distribution of the models according to their protection and the experiment duration. The level of instrumentation “S” for Strong and very complete instrumentation and “L” for the light and essential one, is also indicated and applies to every group.
In order to amplify the exposition of the torso to the blast overpressure effects, animals in the “unprotected” torso group were equipped with a soft body armour that has been reported to increase injury risks during exposure to blast overpressure compared with naked individual [6]. Targets were wrapped in a specifically designed thoracic protection either limited to the soft pack of the body armour

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defined as P2 and assimilated to the Th- entity, or including thoracic and pelvic ceramic plates in their respective pockets and defined as P3E-CERA and Th+ entity.
The helmeted condition, corresponding to the head encapsulated into a rigid aluminium box to prevent against direct blast loading, is defined as the H+ entity. The unprotected head is defined as H-.
Finally, three scenarios or groups are investigated: unprotected (Th-/H-), helmeted only (Th-/H+), or torso protected only (Th+/H-).

Table 33. Groups and their characteristics

Group	Sub-Group	Thoracic protection	Head protection	Instrumentation: S=strong; L=light	Number of cases	
					n	total
Th-/H-	**Th-/H- L**	P2	No	L	4	10
					2	
	Th-/H- S	P2	No	S	4	
Th-/H+	**Th-/H+ L**	P2	Yes	L	7	10
	Th-/H+ S	P2	Yes	S	3	
Th+/H-	**Th+/H- L**	P3E-CERA	No	L	3	8
					2	
	Th+/H- S	P3E-CERA	No	S	3	

2.4 Physical recordings

In addition with usual clinical instrumentation for hemodynamic measurement, cardio-respiratory and brain functions, other sensors were placed on the biological models to observe how the equipment affects the pressure transfer inside the body. The list of the sensors and their respective uses and filtering are presented in Table 34. Their locations are depicted in Figure 2.

Table 34. List of sensors, data collected and filter used

Sensors	Parameters	IIR Filtering, type Bessel		
		Frequency (kHz)	Advanced parameters	
Uniaxial accelerometer (PCB, 3501A)	Rib acceleration (screwed on #K8 ipsilateral right side)	1.65	4 poles	CFC1000
Hydrophone (Reson, TC4013)	Resultant pressure on the thorax	80	6 poles	Phase0/Begin/end
Pressure sensor (Kulite, XT190)	Reflected pressure on the jaw (ipsilateral right side)	80	6 poles	Phase0/Begin/end
Hydrophone (Reson, TC4013)	Intra-oesophageal pressure	10	6 poles	Phase0/Begin/end
Pressure sensor (Millar, MPR-500 Mikro-Tip®)	Intracranial pressure in the parenchyma	5	6 poles	Phase0/Begin/end
Pressure sensor (Millar, SPR-407)	Intravascular pressure from carotid and/or jugular	3	6 poles	Phase0/Begin/end
Pressure sensor (Millar, SPR-751 Mikro-Tip®)	Proximal and distal tracheal pressures	0.6	6 poles	Phase0/Begin/end

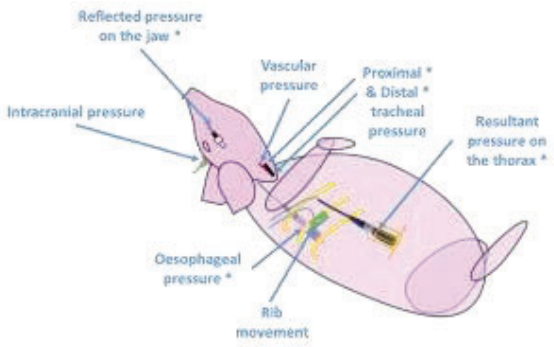

Figure 2. Sensor locations on the strongly instrumented animal model (*: instrumentation for the lightly instrumented animal model).

Signals were sampled at 1MHz using a transient recorder TransCom (MF Instruments GmbH, Germany) from 1 second before explosion to 3 seconds after for all transient events, whereas a continuous basic sampling at 1 kHz was used from -15min to +60 min for other events. Raw data were then post-processed through their respective digital filters to suppress noise but keeping the characteristics of shockwave. The filter characteristics used in DIAdem (National Instruments) are reported in the table 2. The incident pressure was filtered in the same manner as for the thoracic resultant pressure and reflected pressure on the jaw.
The level of threat has been defined as the maximal peak pressure, the duration of overpressure or the impulse. Because of the lack of consensus, the three of them were considered in this study.

2.5 Statistical analyses

Statistical analyses were performed using JMP® (15.2.0). As previouslt described [18], the distribution normality was graphically checked and then the equality of variances were checked using Levene's test. Once the hypothesis of normal distribution and homoscedasticity were validated, statistical analyses was performed using the Tukey-Kramer's HSD (Honestly Significant Difference) test. If the homoscedasticity hypothesis was not validated, Welsh's test was used. Only the duration of threat overpressure, the threat impulse, the maximal distal tracheal pressure and acceleration of the ribs were compared using Welsh's test. The other parameters were compared among groups using the Tukey-Kramer's HSD test.

3. RESULTS

All graphs show results expressed in bars and not in kPa, keeping in mind that 1 bar = 100 kPa.

3.1 Blast parameters

Figure 3 illustrates the pressure-time profile and corresponding impulses during the last experimental trials. The incident pressure profile reached an average maximal value of 460 ± 60 kPa, with an overpressure duration of 2.2 ± 0.4 ms and an impulse of 240 ± 40 kPa.ms. No significant difference was observed in maximal pressure and overpressure duration between groups. Impulse was significantly higher for the Th-/H- group compared to the Th-/H+ group (+40 kPa.ms, p= 0.0281). No other significant impulse difference was observed between groups.

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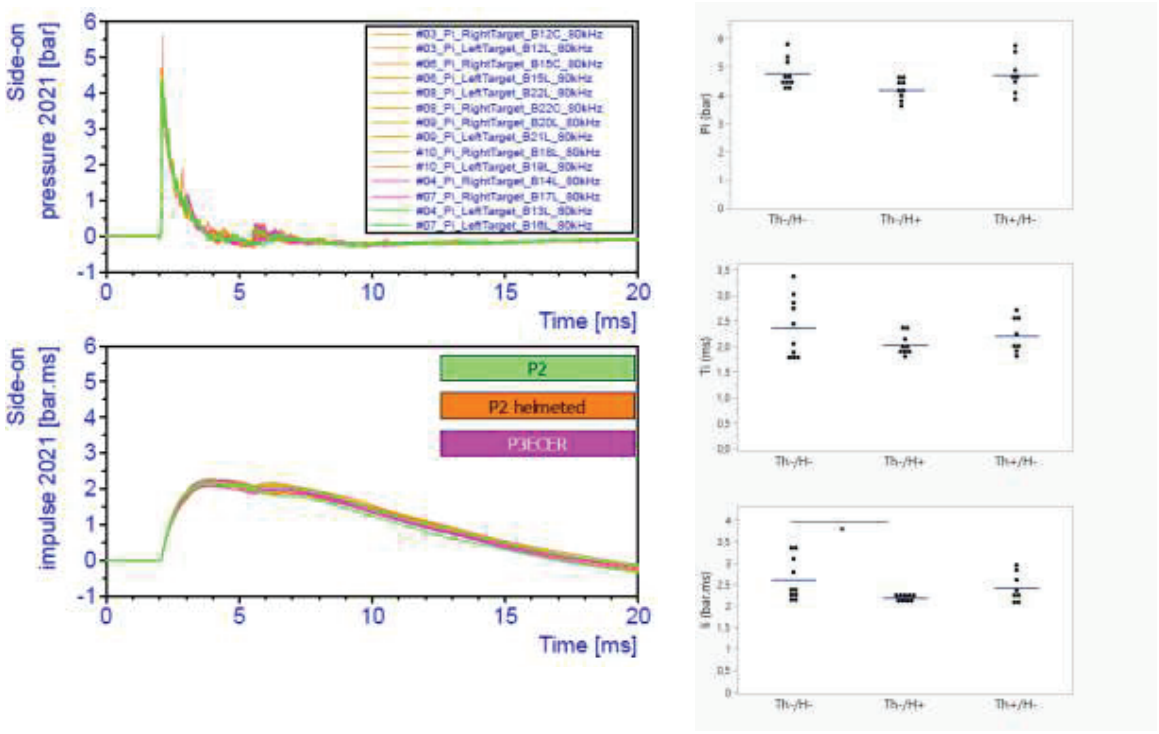

Figure 3. Blast kinetics for the 2021 test series (left); Pi: peak pressure, Ti: positive phase duration, Ii: impulse, *: $p<0.05$, describing the threat experienced by subgroups (right)

3.2 Screening on and through the body

- At the skin surface (exposed right side thorax/abdomen), the resultant pressure signals behind the armour reached a maximal value of 780 ± 360 kPa, with an overpressure duration of 1.7 ± 0.7 ms and an impulse value of 270 ± 120 kPa.ms. No significant difference was observed in maximal pressure, overpressure duration and impulse between groups, as described in Figure 4.

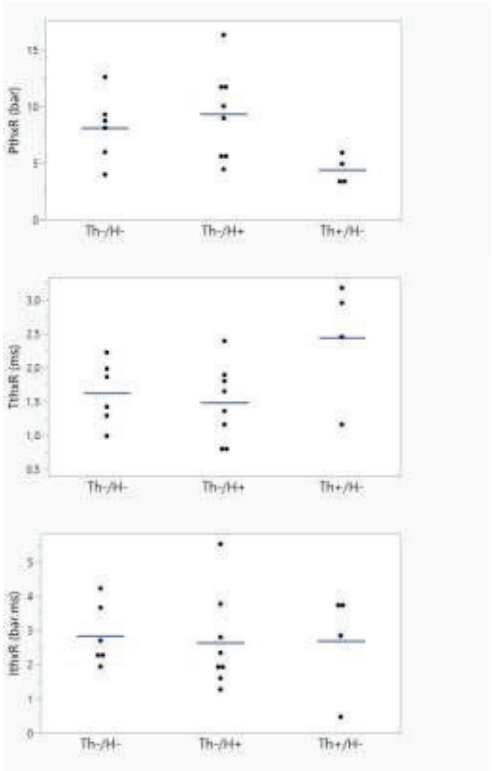

Figure 4. Resultant surge of pressure at the right thorax/abdominal skin surface: PthxR: peak pressure, TthxR: positive phase duration, IthxR corresponding impulse.

- At the jaw level of the exposed (right) side (Figure 5), the resultant pressure signals were significantly higher for Th-/H- and Th+/H- groups compared to the Th-/H+ group (+600 kPa, $p<0.001$ and +546 kPa, $p=0.0002$, respectively). No significant difference was observed between the other groups. The overpressure duration was significantly lower for theTh-/H- and Th+/H- groups compared to the Th-/H+ group (-6.72 ms, $p=0.0010$ and -6.52 ms, $p=0.0012$, respectively). No significant overpressure duration difference was observed between the other groups. Finally, the impulse value was significantly higher for the Th-/H- and Th+/H- groups compared to the Th-/H+ group (+213 kPa.ms, $p=0.00142$ and +266 kPa.ms, $p=0.0041$, respectively). No significant impulse difference was observed between the other groups ($p>0.15$).

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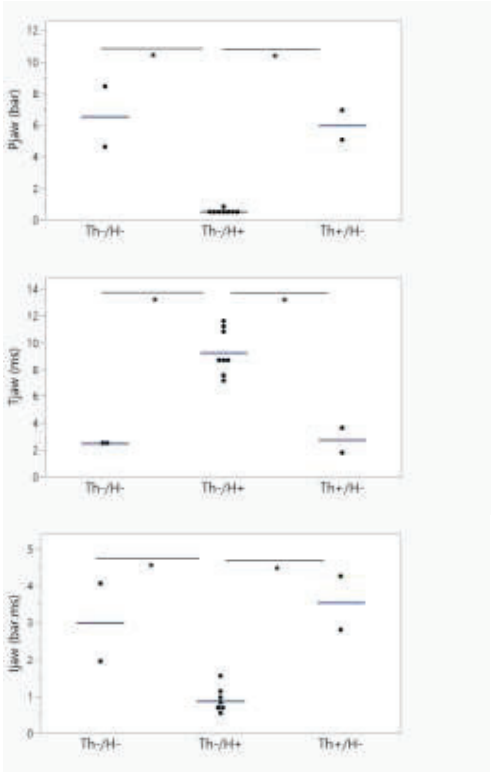

Figure 5. Resultant surge of pressure at the jaw surface: Pjaw: peak pressure, Tjaw: positive phase duration, Ijaw and corresponding impulse, *: $p<0.05$

- In terms of kinematics of the chest wall, the axial acceleration of the rib #K8 reached a maximal value of 25 ± 8 $km.s^{-2}$, with a maximal velocity of 7.3 ± 2.0 $m.s^{-1}$ and a maximal displacement of 6.2 ± 4.3 mm. Values obtained from the double integration of the acceleration are questionable due to the inherent shift during the processing. No significant differences were observed in any of these parameters between groups.

- In the intrathoracic area, oesophageal pressure reached a maximal value of 240 ± 140 kPa, with an overpressure duration of 6.4 ± 1.0 ms and an impulse value of 300 ± 90 kPa.ms. Figure 6 depicts the surge of pressure propagating at the center of the thorax (oesophagus) while the chest wall is suddenly compressed. No significant difference was observed in maximal pressure between groups. The overpressure duration was significantly longer for the Th-/H- group compared to the Th-/H+ (+1.4 ms, p= 0.0021) and Th+/H- (+1.1 ms, p=0.0334) groups. No significant difference was observed between the Th+/H- and Th-/H+ groups (p=0.7070). The impulse value was significantly higher for the Th-/H+ group compared to the Th+/H- group (+130 kPa.ms, p=0.0137). No significant difference was observed between the other groups (p>0.1).

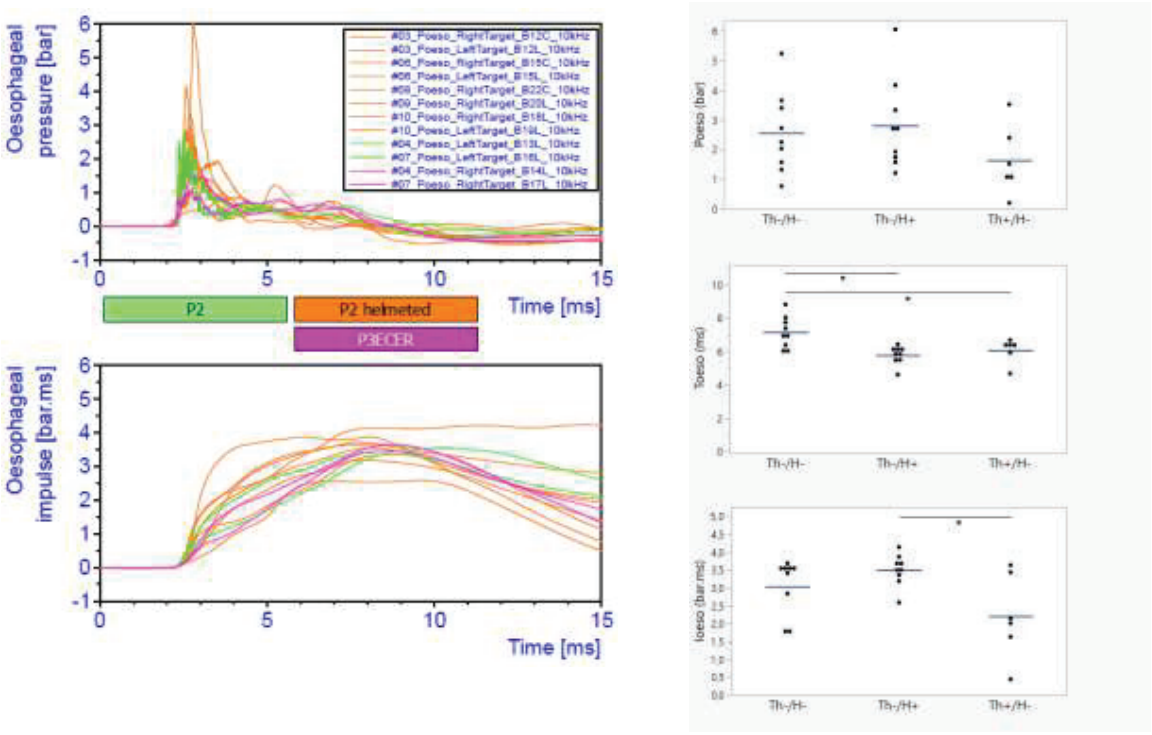

Figure 6. Pressure-time histories of the intrathoracic pressure for the 2021 series (left); Poeso: peak pressure, Toeso: positive phase duration, Ioeso: impulse, *: $p<0.05$, describing the resultant surge of pressure by subgroups (right)

- In the large vessels coming from or going to the head, the vascular pressure was measured at the jugular and/or carotid level (Figure 7). Jugular pressure was measured only for the Th-/H- and Th-/H+ groups. The maximal jugular pressure was significantly higher for the Th-/H- compared to the Th-/H+ group (90 ± 0.4 kPa vs +66 kPa, p= 0.0315). The overpressure duration was more important for Th-/H+ group compared to the Th-/H- group (4.8 ± 1.2 ms vs +2.2 ms, p=0.0004). The impulse value was higher in the Th-/H- group compared to the Th-/H+ group (1.0 ± 0.4 bar.ms vs+70 kPa.ms, p=0.0153). Carotid pressure was measured only for the Th-/H- and Th+/H- groups. Carotid pressure reached a maximal value of 160 ± 40 kPa, with an overpressure duration of 2.8 ± 0.6 ms and an impulse value of 127 ± 3 kPa.ms. No significant difference was observed between both groups.

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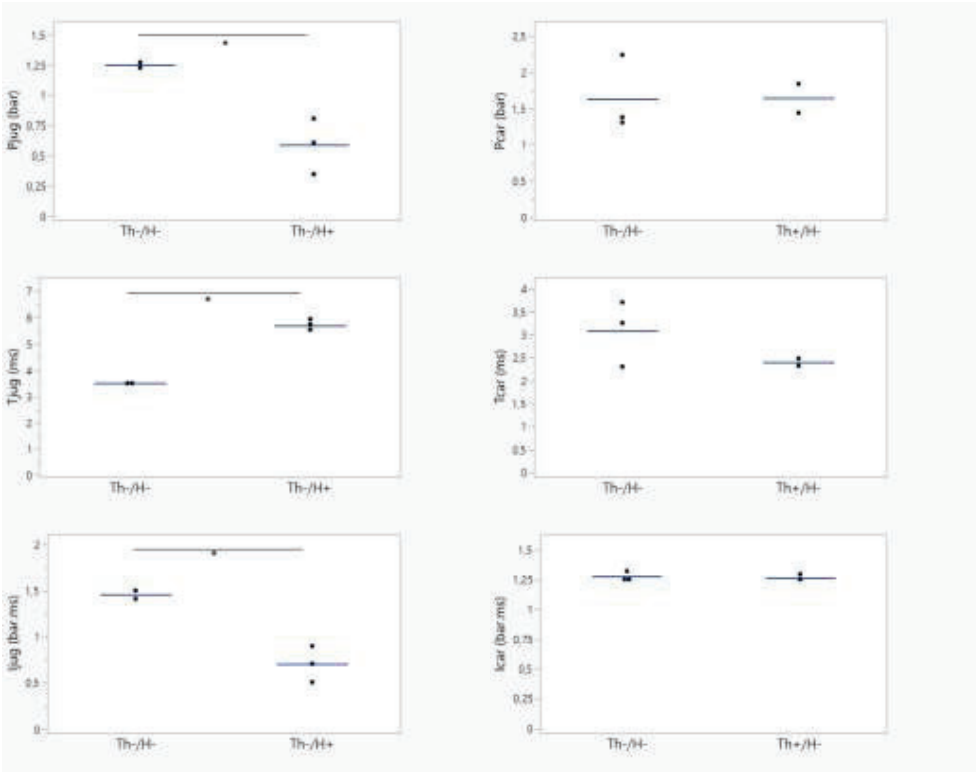

Figure 7. Resultant surge of pressure into the large vessels: jugular (left); carotid (right) in terms of peak pressure (top), positive phase duration (center) and corresponding impulse (bottom).

- Inside the brain parenchyma (Figure 8), the maximal intracranial pressure ICP reached significantly higher levels for the Th-/H- group compared to the Th-/H+ group (+186 kPa, $p<0.0001$) and for Th+/H- (+29 kPa, $p=0.0206$). ICP values were significantly higher for the Th+/H- group than for the Th-/H+ group (+157 kPa, $p<0.0001$). The overpressure duration was significantly longer for the Th-/H+ group than for the Th-/H- group (3.8 ± 2.5 ms +4.4 ms, $p=0.0224$) and for Th+/H- (+4.3 ms, $p=0.0231$). No significant difference was observed in duration of overpressure between Th+/H- and Th-/H- ($p=0.9992$). The impulse value was lower in the Th-/H+ group than in the Th-/H- group (-120 ± 40 kPa.ms vs -80 kPa.ms, $p=0.0140$) and than in the Th-/H- group (-60 kPa.ms, $p=0.0348$). No significant difference was observed in terms of impulse between the Th-/H- and Th+/H- groups ($p=0.5524$).

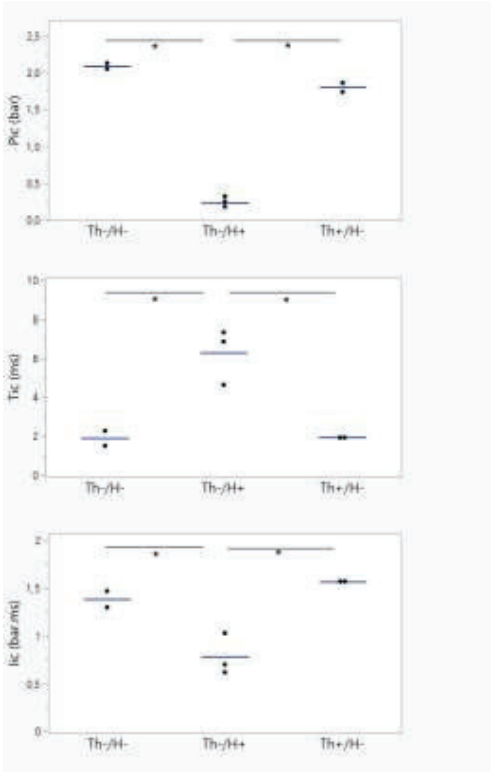

Figure 8. Resultant surge of pressure into the brain: Pic: peak pressure, Tic: positive phase duration, Iic: impulse, *: $p < 0.005$.

- In the trachea, the maximal proximal and distal pressures were not significantly different between groups ($p>0.7$). The delays in detection of the shockwave in the trachea were compared using a simple correlation analysis. The results showed they were related with a factor 1 ($p<0.0001$), but the constant was not significantly different from 0. Thus, it is not possible to determine which of the pressure sensors was hit first to deduce the wave direction. The difference of maximal pressures between both positions in the trachea was computed. Its average was 0 and no significant difference was observed between groups ($p>0.4$). These observations led to suppose there is no propagation of the shockwave in the caudal/cranial direction.

4. DISCUSSION

4.1 Blast threat characteristics

Statistical analysis on the data of the incident pressure showed the only significant difference was a higher impulse for the group Th-/H- than for the group Th-/H+ (+40 kPa, $p=0.0463$). As no other significant difference was observed, the threat was considered similar for all groups.

4.2 Effect of the different protections

None of the protection had a significant effect on the resultant pressure on the thorax and on the axial acceleration of the rib. Adding ceramic plates and head protection had no significant effect on the maximal pressure measured in the oesophagus whereas it significantly shortened the overpressure duration. Both effects seemed to be unrelated as no significant difference was observed between the Th-/H+ and Th+/H- groups. Removing ceramic plates and adding head protection (Th+/H- vs Th-/H+) significantly increased the oesophageal pressure impulse. However, the differences were not significant when removing ceramic plates (Th-/H-vs Th+/H-) or adding head protection (Th-/H+ vs Th-/H-) seemed to increase the impulse value.

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So, there is a combined effect on the torso loading by the blast wave of adding the head protection and removing the ceramic plate.
Significant differences were observed in the pressure profile measured in the oesophagus whereas none were observed in the resultant pressure on the thorax and in the movement of the ribs. That could imply that complex mechanisms of reflection and propagation of shockwave are involved in the resultant pressure measured in the organs. Moreover, the protection equipment may have an effect on those mechanisms and not directly on the threat the body is exposed to.
Adding ceramic plates (Th+/H- vs Th-/H-) did not have any impact on the vascular pressure profile and on the duration and impulse of intracranial overpressure, but it significantly decreased the maximal intracranial pressure. Protecting the head (Th-/H+ vs Th-/H-) significantly decreased the maximal vascular and intracranial pressures and corresponding impulses while both vasculat and intracranial overpressure durations were significantly increased. The effect of protecting the head was stronger than the effect of adding ceramic plates as the differences between Th+/H- and Th-/H+ were similar to the differences between Th-/H- and Th-/H+ and opposite to the differences between Th+/H- and Th-/H-.
Our results showed that he maximal pressure peak was reached at the same time in the distal and proximal parts of the trachea, and had the same amplitude for both. This suggests that there is no propagation of the shockwave in the caudal/cranial direction.
In summary (Table 3), adding ceramic plates (Th+/H- vs Th-/H-) was associated with a shorter duration of overpressure in the oesophagus and a smaller maximal intracranial pressure. Protecting the head (Th-/H+ vs Th-/H-) was associated with a shorter duration of overpressure in the oesophagus, smaller maximal vascular and intracranial pressures and impulses and longer duration of vascular and intracranial overpressures.
Taken together, our results demonstrate that the addition of a protection to the thorax impacts the profile of the post-blast rise in intracranial and oesophageal pressure. This supports the results of previous studies showing brain consequences to chest exposure to blast [7;10-11;15].

Table 35. Summarised significant changes observed at the different sites when torso or head were protected

<table>
<tr><th rowspan="2">Group</th><th rowspan="2">Thoracic protection</th><th rowspan="2">Head protection</th><th colspan="6">Significant influence (Overpressure (P) / Duration (T) / Impulse (I))</th></tr>
<tr><th>Reflected pressure</th><th>Kinematics of the rib</th><th colspan="2">Oesophageal pressure</th><th>Vascular pressure</th><th>Intracranial pressure</th><th>Tracheal pressure</th></tr>
<tr><td>Th-/H-</td><td>No</td><td>No</td><td>Ref</td><td>Ref</td><td colspan="2">Ref</td><td>Ref</td><td>Ref</td><td>Ref</td></tr>
<tr><td>Th-/H+ vs Th-/H-</td><td>No</td><td>Yes</td><td rowspan="2">- / -</td><td rowspan="2">- / T↘ / I ↗</td><td rowspan="2">- / Combined or separate effects</td><td>P ↘ / T↗ / I↘</td><td>P ↘ / T↗ / I↘</td><td>-</td></tr>
<tr><td>Th+/H- vs Th-/H-</td><td>Yes</td><td>No</td><td>- / - / -</td><td>P ↘ / - / -</td><td>-</td></tr>
</table>

4.3 Propagation of the shockwave in organs

The results on the times of arrival and maximal pressures in the trachea suggests that the direction of the shockwave was mainly lateral to medial and not caudal to cranial. However, the consequences of adding thoracic or cranial protection led to think that the shockwave is partially transferred in the caudal/cranial direction. For both to be true, the only solution is for the shockwave to be transferred through fluids, such in blood vessels. Because fluids are less compressible than gas, it can be hypothesized that the shockwave transferred in caudal/cranial direction, based on the observations on the consequences of adding thoracic or cranial protection, is transferred through the vascular system. This is in agreement with mechanisms that have been described in the literature [3;7;13;15].
For two cases, both jugular and carotid pressures were measured, and in both cases, the carotid pressure was about 0.1 bar higher than the jugular pressure. This could be due to the cardiac valves allowing the blood to travel in one direction only, to the tissue nature (venous or arterial) and their mechanical characteristics. Further studies would be required to investigate the effect of blast on the cardiac system.

5. CONCLUSION

As evidenced in the literature [3;7;13;15] and in this study, when developing new military protection, it will be important to take into consideration the repercussions on local and distal body parts.
The first tendencies observed here on large mammals resulting from direct blast exposure in free field which will be helpful for understanding of the mechanisms involved in the bTBI. Indeed, a screen interfaced between threat and body can behave as a protector or a facilitator in relation to the injury. Our study showed a protective effect when hard plates were added in terms of reducing the duration of blast overpressure in the oesophagus and reducing maximum intracranial pressure. In helmeted animals, the chamber acts as a protector by reducing the duration of overpressure in the oesophagus, by reducing maximum vascular and intracranial pressures and impulses and by prolonging the duration of vascular and intracranial overpressures. In addition, it is not easy to protect the head effectively without redirecting blast waves to other parts of the body. Further studies will be needed to clarify this point and determine the strategy to be followed in developing protection, as a number of questions remain unanswered. For example, we need to determine which maximum pressure peaks, the duration of the overpressure and the pulses that present the greatest risk to organs.

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