Mechanical Response and Brain Injury of Swine Subject to Free-Field Blast

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Introduction

The mechanism by which primary blast overpressure produce mild to moderate traumatic brain injury is still unclear. The assessment of the severity of injury following primary blast is still a great challenge clinically. The aim of this study is to explore and quantify relationships between mechanical response and brain injury on live anesthetized swine exposed to open-field blast. The goal of the study is to understand the mechanisms of primary blast-induced injury and to determine the relationship between intracranial pressure in the brain and its effect on animal damage and cellular injury.

Design and methodology

- Instrumented swine tests
  - All the intracranial pressure sensors were calibrated with pencil pressure in a shock tube. Error in peak pressure was less than 10%.

- Biomechanical data
  - Figure on the right shows the distribution of peak IP as a function of standoff distance. With higher, ICP readings at different regions of the brain increased (see Figure A, B, C, D and E). In front, among all regions of the brain, ICP readings at parietal region had highest values at medium and high level of blasts (see Figure A). ICP readings at central regions were much lower than parietal regions. ICP readings at frontal region were higher than at occipital region.

- Brain injury -Histology results
  - A total number of 5 non-instrumented tests were performed at Medium Level IP. Sabins and EDSAs are not completed. The framed stains are frontal sections. The preponderance of swollen axons and axons with vacuolations in blast exposed brain sections.

- Biomechanical data
  - The three figures above showed the typical intracranial pressure readings, linear acceleration and angular velocity, respectively, in live IP exposures.

Experimental setup

Swine specimen
- Adult Male Yucatan pig
- Weight: 50-60 kg
- Intracranial pressure (ICP) measurement
  - Kulite pressure sensor
- Location: Frontal, Parietal, Left Temporal, Right Temporal, Occipital and Center lobe
- Near acceleration (LA) and angular rate (AR) measurement
- 3 linear accelerometers and 3 angular rate sensors were mounted in a block and placed on the occipital bone
- Test arrangement instrumented pig paired with non-instrumented pig
- Each instrumented pig was blasted at 3 different levels (Low, medium and high) at 3 directions (front, side and back)
- Data Acquisition system: DEWETRON system

Biomechanical data
- Figure on the right shows the distribution of peak IP as a function of standoff distance. With higher, ICP readings at different regions of the brain increased (see Figure A, B, C, D and E). In front, among all regions of the brain, ICP readings at parietal region had highest values at medium and high level of blasts (see Figure A). ICP readings at central regions were much lower than parietal regions. ICP readings at frontal region were higher than at occipital region.

Results
- A total number of 19 blasts were performed on 3 instrumented pigs. A summary of the tests is provided below:

| Test arrangement | Instrumented pig paired with non-instrumented pig | Each instrumented pig was blasted at 3 different levels (low, medium and high) at 3 directions (front, side and back) | Data Acquisition system: DEWETRON system |
|------------------|--------------------------------------------------|-------------------------------------------------------------------------------------------------|

<table>
<thead>
<tr>
<th>ICP readings</th>
<th>Medium Level IP</th>
<th>High Level IP</th>
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<tbody>
<tr>
<td>Low IP</td>
<td>2</td>
<td>4</td>
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<tr>
<td>Medium IP</td>
<td>4</td>
<td>4</td>
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<td>High IP</td>
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Conclusions

Overall, in front, rear and side blast exposures, ICP in various brain regions increased with increasing IP. The peak ICP values were by and large lower than the peak IP. We are investigating possible cause of this phenomenon, since it was expected that ICPs would be greater than IP. In non-instrumented swine exposed to Medium Level IP, histological evidence of increased GFAP activity and axonal injury was present. Further quantification of biomarker and histology outcomes is underway.

Acknowledgements

This research was supported by MRMC. Contract No. WB-2013-12-3-008, US Army. The views, opinions and/or findings contained in this paper are those of the author(s) and should not be construed as an official Department of the Army position, policy or decision unless so designated by other documentation.