

# Preliminary Investigation of Skull and Brain Dynamics During Blast

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## Introduction

Blast-induced traumatic brain injury (bTBI) is a growing health concern in military populations. Primary blast injury describes damage that is a direct result of blast wave propagation through tissue; however, the mechanisms involved in these injuries are not well understood due to the complex interactions of the blast wave with the physiological systems.<sup>1</sup> While several mechanisms of primary blast brain injury have been proposed, there is little evidence to support these theories. Many of the proposed mechanisms can be explored with preclinical studies. The rodent model has been frequently used for TBI research, however the minipig model exhibits a closer anatomical representation to humans.<sup>2</sup> Using Göttingen minipigs, this study aims to characterize the interaction between the blast wave and brain by measuring skull strain and intracranial pressure during blast exposure.

## Methodology

A young-adult (25 weeks) male Göttingen minipig was exposed to a low- and high-pressure blast in an advanced blast simulator (ABS) located at Virginia Tech. Four intracranial pressure (ICP) transducers were implanted in the right hemisphere of the brain, and a 45-degree rectangular rosette strain gage was fixed to the apex of the skull. The walls of the ABS were instrumented with five pressure transducers to measure the static overpressure at the location of the specimen, and a two-sensor pencil probe was used to measure stagnation pressure and static overpressure near the specimen's head. The specimen was wrapped with a lead blanket to prevent thoracic injury and secured within a rigid fixture in the ABS.

## Results and Discussion

The peak static overpressures for the low- and high-level blasts were 220.9 and 319.6 kPa, respectively, as measured by a transducer mounted on the wall of the ABS where the specimen was secured. The peak blast stagnation pressure in the ABS was 387.1 kPa for the low-pressure test and 593.1 kPa for the highpressure test. Initially, the transient peak ICP was either equal to or lower than the peak stagnation pressure (Figure 1). For the initial 0.5 milliseconds, the ICP signals followed the stagnation pressure signal and then oscillated about the stagnation pressure. For the two blast pressure levels, the ICP measurements showed repeatable trends. Beyond the initial 0.5 milliseconds, the maximum principal strain exhibited a similar trend as that of the ICP signals, perhaps indicating that skull strain has a role in the blast wave propagation through the brain.

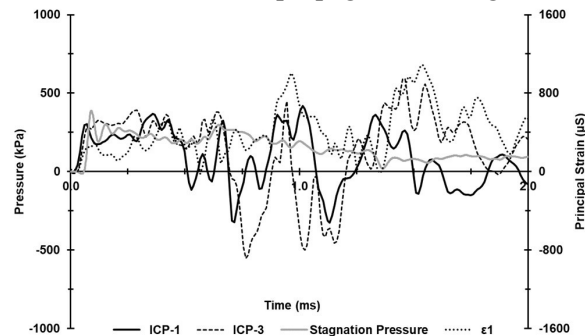


Figure 1. Transient ICP, ABS stagnation pressure, and skull principal strain responses for the low-pressure test.

## Conclusions

Mechanisms of primary blast brain injury are highly disputed, yet there is little support for any of the current proposed theories. The results from this study illustrate trends between ICP and skull strain that, with a greater sample size, could help elucidate the skull flexure dynamics or direct cranial transmission theories. This information could directly lead to a better understanding of the underlying mechanism or set of mechanisms associated with primary blast brain injury and could ultimately result in the improvement of protective equipment as preventative measures.