Development of a murine model of blast-induced growth plate injury

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I. INTRODUCTION

Almost 20% of children worldwide live in conflict zones [1] and a significant prevalence of blast-related extremity injuries can be observed in this vulnerable population during and post-conflict [2]. Blast waves can cause systemic physiological disruption, along with profound local mechanical effects that are especially deleterious at the interfaces of tissues of different densities [3]. Growth plates are cartilaginous tissues, located towards the ends of long bones, which drive longitudinal bone growth in children and are critical to normal bone development. Mechanical insults to the growth plates, or to their adjacent metaphyseal or epiphyseal bone, can lead to the formation of osseous bridges and a subsequent loss of growth potential at the region of injury. This in turn can lead to growth disturbances, such as angular deformities or limb length discrepancies [4], that contribute to functional deficit and potentially secondary musculoskeletal conditions [5]. The precise immediate and long-term impacts of blast exposure on growth plate function is currently unexplored. This work outlines the first steps that have been undertaken to establish a protocol to achieve blast-induced growth plate injury in cadaveric rat specimens using a pneumatically driven shock tube.

II. METHODS

Eighteen juvenile Sprague-Dawley rat cadavers (42-48 d/o) were placed on a protective platform mounted on the outlet flange of a pneumatically driven (compressed air) shock tube (Fig. 1). The protective platform was used in conjunction with a targeting insert to confine blast wave exposure to the proximal tibiae of the left hind limbs in the cadaveric rat specimens. The knee and ankle were physically constrained, and blast waves of varied pressure (2, 3, 4, 5, and 6 bar diaphragm burst pressures) were fired from the shock tube.

X-ray imaging was used to evaluate the presence of unintended bone fractures and intra-abdominal gas formation related to excessive bodily displacement. Micro-CT was utilised to assess damage to the proximal tibia. Contralateral limbs served as controls.

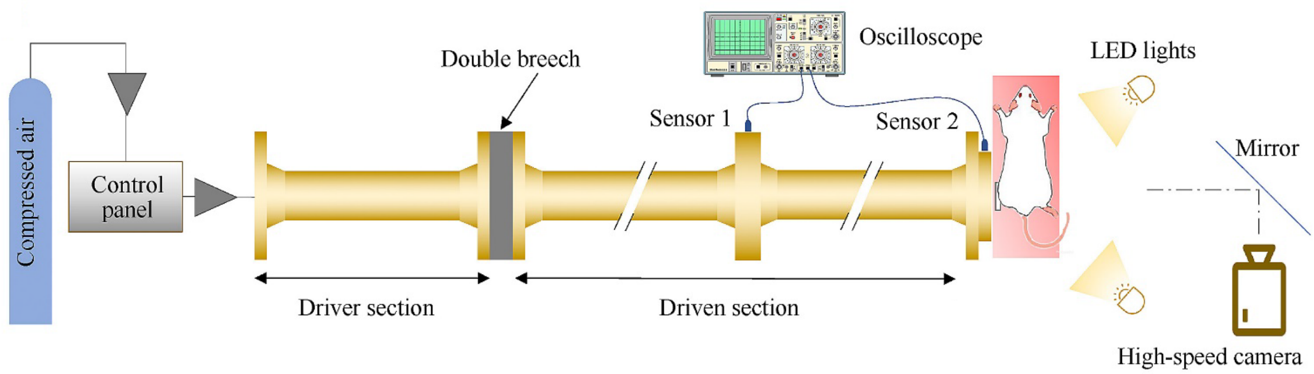

Fig. 1. Experimental set-up used for blast-induced growth plate injury. Figure adapted from [6].

III. INITIAL FINDINGS

Micro-CT images exhibited damage to the proximal tibial metaphysis in all specimens tested at 3 bar or higher (Fig. 2). Intra-abdominal gas or fractures of the tibia or fibula (Fig. 3(a)) were apparent in some specimens tested at 4 bar or higher. No damage was observed in the contralateral limbs.

Metaphyseal bone damage appeared to involve delamination or metaphyseal fragmentation (Fig. 3(b)), with damage severity tending to increase with blast exposure intensity.

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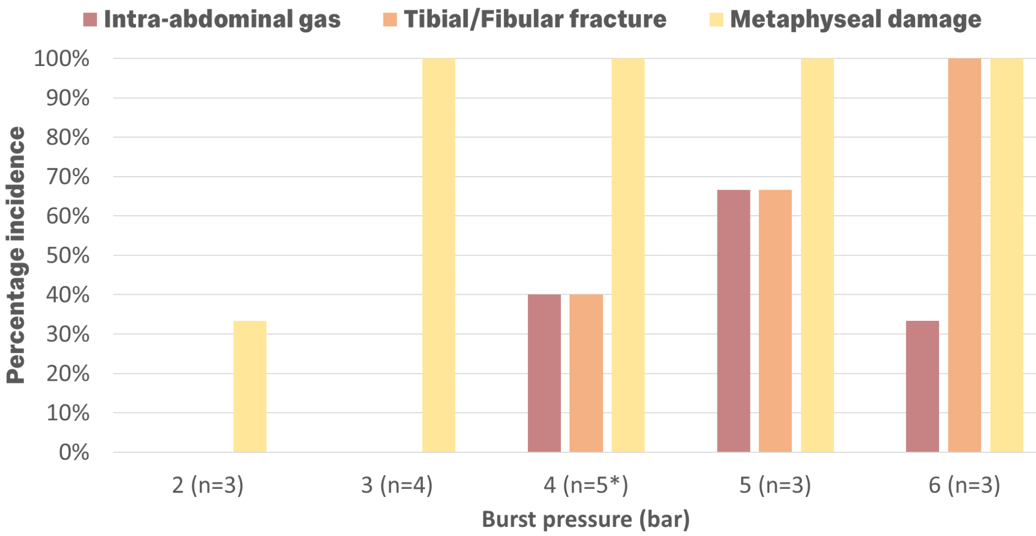

Fig. 2. Injury outcomes in cadaveric rat specimens following proximal tibial blast exposure.
*One sample damaged during dissection and not imaged for metaphyseal damage.

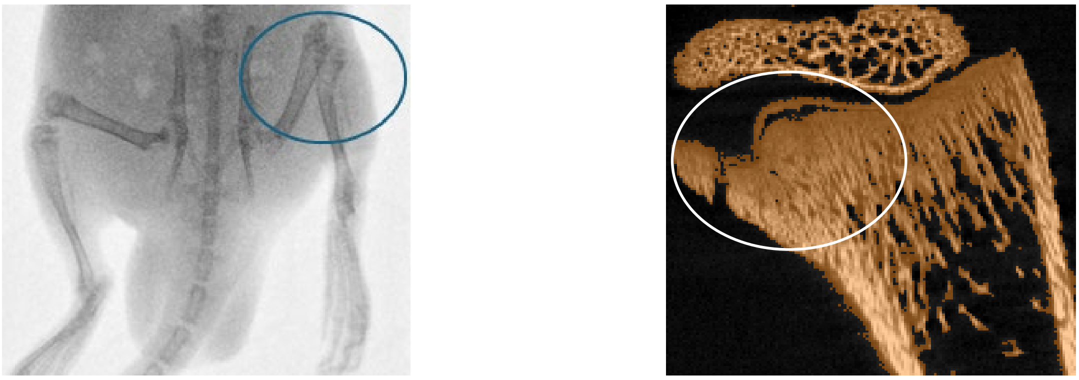

Fig. 3. (a) X-ray image exhibiting tibial fracture following proximal tibial blast exposure (6 bar). (b) Micro-CT scan exhibiting proximal tibial metaphyseal damage following proximal tibial blast exposure (5 bar).

IV. DISCUSSION

This is the first study to have created isolated (and non-isolated) growth plate damage using an isolated blast wave. The metaphyseal damage observed in the blast-exposed proximal tibiae can be associated with damaged growth plate vasculature and disrupted growth plate function. Injuries to the growth plates could not be directly measured using the tested imaging methods. One specimen tested at 4 bar could not be imaged with micro-CT due to separation of the proximal tibial growth plate during sample preparation, potentially due to transverse injury involving the growth plate. Intra-abdominal gas formation may have been related to improper positioning of the protective platform locking mechanism in some specimens. As the protocol for blast-induced growth plate injury is to be translated to an *in vivo* model, the desired growth plate injury outcome must be achieved with the absence of unintended bone fractures or intra-abdominal gas formation (related to organ damage). These preliminary results suggest that blast-exposure intensities of 3–4 bar burst pressure may be suited for this.

Future work will involve further optimisation of the blast exposure and targeting approach used for the blast-induced growth plate injury protocol. Additionally, alternative modalities, such as contrast enhanced micro-CT, or histopathology, may be employed to examine the mechanical effects of blast exposure on the growth plate.

V. REFERENCES

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