The Injury Mechanism of Explosive Blast Trauma, with Protective Strategies for the Pelvis and Lower Limbs

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I. INTRODUCTION

Explosive blast has been the most common cause of wounding and death in recent military conflicts. Where blast resulted in injury to the pelvis and lower limbs of an on-foot casualty, the mortality rate was high. The mechanism of injury by which this occurs is not known. The research presented sought to understand the pattern and mechanism of this devastating injury, in order to develop protective strategies.

An analysis was performed of battlefield data which identified pelvic vascular injury as the cause of death in these casualties. Furthermore, it showed displaced pelvic fractures, perineal wounding, and traumatic amputation to be associated with this lethal injury [1].

Hypothesised mechanisms of injury were investigated using cadaveric animal models of blast. These investigations showed rapid outward movement of the lower limbs (*limb flail*), caused by the blast wave, to be necessary for displaced pelvic fractures with vascular injury to occur [2]. High velocity sand ejecta, as propagated by blast (*sand blast*), showed correlation with increasing velocity and injury patterns of worsening severity across the trauma range. This included the associated injuries of perineal wounding and traumatic amputation [3]. Following this research, lower limb flail and high velocity sand blast were identified as the mechanisms of injury of blast to the pelvis.

Pelvic protective equipment was developed to limit lower limb flail in a cadaveric animal model of blast. This resulted in a reduction of pelvic fractures and elimination of pelvic vascular injury [4]. Following this work, the research direction of our group returned to the injury mechanism of traumatic amputation.

II. METHODS

In the novel research presented, traumatic amputation is reproduced using high-velocity environmental debris utilising a gas-gun system of high velocity sand blast in an animal model. The sandy gravel was housed within a hollow polycarbonate sabot which was loaded into the firing chamber of a double-reservoir gas-gun system. Within this system, a 2-litre reservoir charged with air or helium and a Mylar® diaphragm firing mechanism was used to accelerate the sabot-sand unit down a 3m long, 32mm bore barrel. The output velocity was controlled by the thickness of the Mylar® diaphragm. The reservoir section of the gas gun was charged to a predetermined firing pressure, to accelerate the sabot-sand unit to the desired velocity. The pressure was maintained within the reservoir section by a Mylar® diaphragm of appropriate thickness (ranging from 50 to 150 μm). The system utilises a priming section, which is charged to a pressure below the rupture pressure of the diaphragm. This reduces the pressure gradient across the mylar diaphragm (containing the reservoir system) and prevents it from rupturing early, as the reservoir is filled. The pressure in the prime section is vented at the point of initiating firing of the gas gun, resulting in rupture of the diaphragm, with release of the pressurised gas. The gas-gun system accelerates the sabot-sandy-gravel unit down a barrel to exit into a target chamber, where the sabot is separated from the sandy gravel by a sabot

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stripper. The sabot is halted at this point, while the sandy gravel continues to travel towards the mouse specimen at the intended terminal velocity.

Further novel research presented investigated the extent to which an under-layer of personal protective equipment (PPE) limits the injuries sustained by energised environmental debris following an explosive event. A post-mortem human subject (PMHS)t model of gas-gun mediated high-velocity sand blast was used in order to simulate the effect of energised environmental debris on injury to the thigh and quantify the reduction in wound severity by a PPE silk under-layer.

III. INITIAL FINDINGS

Fifty-nine cadaveric mice were used across experiments, comprising of a total of 117 lower limbs impacted by high-velocity sandy gravel soil, and one lower limb control specimen. No injuries were seen in the control specimen. A gas-gun system was used to accelerate the sandy gravel; the average sand blast velocity at the exit of the gun's barrel ranged from 20 ± 5 m/s to 136 ± 5 m/s. The injury curves presented show a link between increasing sandy gravel velocity and likelihood of injury. Ideally distributed dry sandy gravel, scaled to the animal model, showed a 25%, 50% and 75% risk of traumatic amputation at sand blast velocities of 62 m/s, 71 m/s, and 79 m/s, respectively.

Twelve PMHS thigh samples were divided into two groups: those wearing trousers with PPE and those wearing trousers without. A gas-gun experimental setup was utilised to simulate the effects of high-velocity environmental debris on injury to the thigh of both groups and post-test injury patterns were recorded. Impact with high velocity sand resulted in soft tissue injuries to all samples, representative of those seen clinically following blast, with injuries ranging from superficial skin avulsion to penetrative wounds deep to the subcutaneous tissues. Mean sand velocity across experiments was 519 ± 23 ms^{-1}. Within the PPE group, a significant reduction was seen in the total surface area (143 mm^2 *vs.* 658 mm^2, $p = 0.004$) and depth (0 *vs.* 23, $p < 0.001$) of injuries; no samples in the PPE group sustained a penetrating injury deep to the subcutaneous tissues of the thigh (odds ratio = 0.0074, 95% confidence intervals 0.0004 – 0.1379).

IV. DISCUSSION

This novel work is the first to recreate traumatic amputation from propagated high velocity sand in an animal cadaveric model and the first to recreate wounds from high velocity sand in a PMHS model. These findings show high velocity environmental debris, as ejected from some blast events, as a critical mechanism of injury in the blast casualty. This injury mechanism should be a key focus of future research and mitigation strategies. A silk under-layer of PPE was shown to markedly reduce the severity of injury, wounds deep to the subcutaneous tissues were eliminated and a reduction in the total surface area of injuries was seen.

V. REFERENCES

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