

## **Novel Fracture-Based Porcine Model of Acute Compartment Syndrome Creates Elevated Intracompartmental Pressure Using Only a Simulated Blast Injury**

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**Purpose:** No existing animal model of acute compartment syndrome (ACS) creates a musculoskeletal injury that spontaneously elevates intracompartmental pressure (ICP). The purpose of the current study was to evaluate the ability of a blast injury model of ACS to spontaneously elevate ICP.

**Methods:** Right juvenile swine hindlimbs were fractured at the tibial diaphysis using a 3-point bending mechanism. The fractured limb was then subjected to 7 blasts of compressed air at 100-110 psi. Both the injured and the contralateral, uninjured control leg were subject to continuous intracompartmental pressure monitoring of the anterior compartment. In the continued compartment syndrome group, the tibial shaft fracture was stabilized using plate and screws followed by imbrication of the anterior compartment fascia at 4 hours post-injury. Select pigs were harvested between 48-72 hours post-injury and a spot pressure measurement was made at the time of harvest. In the fasciotomy group, animals underwent operative fixation followed by immediate fasciotomy. ICP data were compared between control, fasciotomy, preoperative experimental, postoperative experimental, and 48-72 hours post-injury experimental groups.

**Results:** At 4 hours post-injury, the novel model of compartment syndrome created significantly elevated ICP ( $45.9 \pm 12.6$  mmHg; N = 14) compared to control limbs ( $16.9 \pm 7.1$  mm Hg; N = 12;  $P < 0.001$ ). Operative fixation further increased ICP ( $85.0 \pm 42.2$  mm Hg, N = 8) relative to the preoperative state ( $P = 0.042$ ). Fasciotomy resulted in marked decreases in anterior compartment pressure following operative fixation ( $14.8 \pm 2.8$  mm Hg, n = 4) which resulted in pressures equivalent to control limbs ( $P = 0.686$ ). Five animals from the continued compartment syndrome group were maintained for 2-3 days post-injury. Pressure measurements at the time of harvest demonstrated that elevated ICP persisted following injury, although the response between individuals was less predictable ( $87.2 \pm 41.8$  mm Hg, N = 5).

**Conclusion:** This model of ACS elevates ICP comparable to existing models of compartment syndrome using a musculoskeletal injury without exogenous ICP manipulation. ICP elevation is relieved with fasciotomy. ICP remained elevated at 48-72 hours but with wide variability.

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