THE ROLE OF DECOMPRESSION WITH EITHER DUROTOMY OR DURAPLASTY FOLLOWING CERVICAL SPINAL CORD INJURY

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INTRODUCTION: Clinical outcomes following spinal cord injury (SCI) in the cervical spinal cord have historically been rather poor with the deficits from SCI occurring both from the traumatic event and the ensuing biologic response, i.e. secondary insult. Regaining even modest control of upper extremity function can have a dramatic effect on the quality of life of spinal cord injured patients. Traditionally, decompression of the bony and soft tissue elements has been the focus of early surgical intervention although the timing and method of surgery remain controversial. While there is no current intervention that can prevent or repair damage that results from the primary insult, novel methods to reduce secondary damage following SCI are of great interest. These secondary changes include ischemia, hemorrhage, alterations in cerebrospinal fluid, and macrophage accumulation.(1) The role of decompressing the subarachnoid space through a durotomy as a treatment option for acute traumatic spinal cord injury (SCI) in the cervical spinal cord has not been explored. Following compressive SCI, spinal cord edema and hematoma formation may augment the secondary inflammatory response and contribute to increased scarring and cystic cavitation. In addition, the potential for accommodating this expanding volume is rather limited by a relatively non-compliant dura. Currently, there is no standard treatment algorithm to decompress the subarachnoid space following spinal cord injury. We sought to determine the role of decompression with either durotomy or duraplasty in acute cervical SCI and evaluate its effects on inflammation, scar information, and functional recovery.

METHODS: Seventy-two adult female Sprague-Dawley rats were randomly assigned to 3 groups: contusion injury alone (n=24), contusion injury with durotomy and decompression (n=24), and contusion injury with durotomy and decompression followed by placement of a dural allograft (n=24). Dural allografts were harvested from additional donor Sprague-Dawley rats (280-300g) and stored overnight in Hanks Balanced Salt Solution (HBSS). Following a posterior approach to the cervical vertebral segments, a laminectomy was performed at C5 to expose the spinal cord. The cervical spine was rigidly immobilized using a stereotactic device. A moderate (200 kilodyne) contusive injury was delivered to the center of the exposed spinal cord segment using a force directed impactor device (Infinite Horizon Impactor) with recordings of the actual delivered force and displacement. In the decompression groups, the injured segment was re-exposed 4 hours following the initial injury and a durotomy was performed. A durotomy with a diameter of approximately 4mm was created using microsurgical instruments and was centered over the contused spinal cord. This decompression extended approximately 1mm beyond the area of hemorrhage and edema that was visualized microscopically. For the duroplasty group, the dural grafts were affixed to the surrounding intact dura using a fibrin sealant (Tisseel- Baxter Health Corporation) after the decompression was performed.

The Grip Strength Meter (GSM- designed by TSE-Systems and distributed by SciPro, Inc.) was used to assess forelimb function in the pre and postoperative period.(2) Animals were sacrificed at 2 and 4 weeks post intervention. Scar formation and inflammatory infiltration were assessed with immunohistochemistry using antibodies to glial fibrillary acidic protein (GFAP), chondroitin sulfate proteoglycan (CSPG), OX-42, and ED-1 (markers for astrocytes, protein inhibitors of CNS regeneration, activated microglia and macrophages, and macrophages, respectively).

RESULTS: Animals receiving dural allograft had significantly improved GSM scores in the recovery period relative to animals receiving contusion injury alone. (p<0.05). Those animals receiving a decompressive durotomy alone without dural allograft showed significantly decreased GSM scores relative to animals receiving contusion injury alone (p<0.05).

Immunohistochemical analysis revealed increased scar formation, cavitation and inflammatory response in the animals treated only with a decompressive durotomy. Relative to the group receiving a contusion injury alone, animals receiving decompressive durotomy followed by dural allograft displayed decreased cavitation and scar formation.

DISCUSSION: Functional recovery after acute cervical SCI was improved with decompression of the subarachnoid space and placement of a dural allograft. This behavior data correlated with the histologic evidence of decreased spinal cord cavitation and scar formation. In contrast, animals treated with decompressive durotomy without dural allograft placement showed decreased functional recovery and a corresponding increase in inflammation, cavitation and scar formation relative to contusion injury alone animals. The data supports the rationale of acutely decompressing the subarachnoid space following a compressive spinal cord injury. It appears that sustaining this benefit and reducing the secondary inflammatory response requires the continuity of an intact overlying dura with an expanded subarachnoid space. Decompression of the subarachnoid space after an acute traumatic cervical SCI may be an important new approach to reducing the deficits from the secondary insult after spinal cord injury and warrants further clinical investigation.

REFERENCES
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