Controlled cortical impact injury

Controlled cortical impact injury“ (CCII) was introduced to model traumatic brain injury.

This method uses a rigid impactor to deliver mechanical energy to an intact dura exposed following a craniectomy. Impact is made under precise parameters at a set velocity to achieve a pre-determined deformation depth. Although other TBI models, such as weight drop and fluid percussion, exist, CCI is more accurate, easier to control, and most importantly, produces traumatic brain injuries similar to those seen in humans. However, no TBI model is currently able to reproduce pathological changes identical to those seen in human patients. The CCI model allows investigation into the short-term and long-term effects of TBI, such as neuronal death, memory deficits, and cerebral edema, as well as potential therapeutic treatments for TBI [1)].

Purpose of this study was to investigate the development and nature of brain edema following CCII.

Traumatic brain injury was applied to the intact dura of the left hemisphere in Sprague-Dawley rats (n = 52, 250-350 g b.w.). Ketamine/xylazine-anesthesia or inhalation-anesthesia were used. A pneumatic impactor with a diameter of 5 mm contused the temporo-parietal cortex with a velocity of 7 m/s and an impact depth of 2 mm. 24 hours post injury the brains were removed. Posttraumatic hemispheric swelling and water content were determined gravimetrically, Evans blue extravasation spectrophotometrically, area and volume of ischemia by staining with TTC. MRI studies were performed with T1-,T2- and diffusion-weighted sequences. Posttraumatic swelling following CCII was 14.3 +/- 3.1%. Brain water content increased to 82.5 +/- 0.5% in lesioned hemisphere compared to 79.9 +/- 0.2% in control hemisphere. Following TTC staining, the average ischemic tissue volume was 56.7 +/- 19.2 mm3. There was a moderate uptake of Evans blue into the lesioned hemisphere. MRI studies demonstrated edema in 35.4 +/- 9.5 mm3 of the lesioned hemisphere. Gd-DTPA was taken up early after trauma only. A significantly decreased ADC (apparent diffusion coefficient) indicates the cytotoxic (ischemic) component of edema in this model. In conclusion, CCII produces significant post traumatic brain swelling and edema which is both, of vasogenic and cytotoxic nature. Thus, the CCII models the human cortical contusion more appropriately and opens new avenues for therapeutical studies focussing on cortical contusions [2)].

1)
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2)
Unterberg AW, Stroop R, Thomale UW, Kiening KL, Päuser S, Vollmann W. Characterisation of brain edema following “controlled cortical impact injury” in rats. Acta Neurochir Suppl. 1997;70:106-8. PubMed PMID: 9416293.