**Introduction**

Central Post Stroke Pain (CPSP) was first described 100 years ago as thalamic pain by Dejerine and Roussy (1906). Taiker (2001) described this as "among the most spectacular, distressing, and intractable of pain syndromes". This is a central neuropathic pain characterized by constant or intermittent pain and is associated with sensory abnormalities, particularly thermal sensation, in the painful body part (Andersen et al. 1995). While the pain is frequently described as burning, stinging, or freezing, symptoms are usually vague, making early diagnosis difficult. Diagnosis is further complicated by cognitive abnormalities that occur as a result of the stroke as well as other functional disturbances including depression, anxiety, and sleep disorders. Most patients also experience both stimulus-evoked and spontaneous dysesthesia, allodynia, and hyperalgesia (Shive et al. 1989). The plethora of symptoms makes the diagnosis difficult and there is currently no medical treatment. A lack of understanding of the mechanisms underlying CPSP impedes the development of targeted treatment strategies.

An animal model depicting CPSP was developed in order to identify the mechanisms underlying the changes in nociceptive mechanisms associated with stroke in the region of the thalamus and the spino-thalamic pathway. This haemorrhagic model exhibits sensory abnormalities including tactile hypersensitivity and aversion to cold stimuli. The present poster demonstrates behavioural, structural and circulatory changes associated with this model at different times following model induction.

**Materials and Methods**

- Male Sprague-Dawley rats weighing 250-300 g
- Intracerebral hemorrhage induced in anaesthetized rats by administration of bacterial collagenase type IV - 0.1 U/L over a ten minute period
- Intracerebral hemorrhage induces site-dependent tactile and cold hypersensitivity in this animal model of CPSP
- **Histology**
- **Mechanical & Thermal sensitivities**, Histology, Apoptosis, Necrosis

For details, please see the handouts on the board.

**Results**

**Plasma Extravasation**

- Fig 1. Histograms showing plasma extravasation in brain slices before and one hour after collagenase injection. Control histogram represents slices taken one hour prior to injection. The black and check histograms represent the ipsi and contralateral side of the brain tissue respectively. Spectrophotometric readings were taken for each sample at 620nm, which were then divided by the dry weight of the tissue. **P<0.05, *** P<0.001 (one group).

**Cold Sensitivity in CPSP Rats**

- Fig 6. The histograms show that compared to the pre-surgical latency, the sensitivity to cold is increased significantly during the 15 of period and returns to normal value by 21 days. **P<0.05, *** P<0.001.

**Histology**

- Figs 7 & 8. At 21 days there was strong Necrosis Signal [3H]PK-11195 binding to reactive astrocytes. Apoptosis peaked at 7 days and by 21 days no Apoptosis signal was detected.

**Conclusions**

- focal haemorrhagic stroke in the area of the thalamus induces site-dependent tactile and cold hypersensitivity in this animal model of CPSP
- these functional changes are accompanied by time-dependent changes in cerebral vasculature
- these functional changes are also accompanied by time-dependent changes in neuronal cells, as indicated by apoptosis as well as by necrosis.

We suggest that this model may be useful for research into mechanisms of central post-stroke pain

**References**


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