Seminar Summary: "Targeting collateral and microcirculatory failure to improve outcome after stroke."

Central hypothesis/hypotheses

The dynamics of collateral flow and microvasculature patency post stroke were hypothesized to predict stroke outcome and constitute a potential therapeutic target.

Key results

1. Collateral flow post middle cerebral artery (MCA) occlusion is obviously diminished in aged versus young animals, appears mediated by faster vasoconstriction of collaterals. Larger infarct volume followed in the aged group.
2. Remote ischemic perconditioning preserved vessel diameter in aged rats and appeared to improve penumbral perfusion.
3. Endothelin 1 (ET-1) progressively elevates in blood plasma with MCA occlusion, but not when remote ischemic perconditioning is applied.
4. Neutrophil stall density is higher in tissue which fails to reperfuse post recanalization.

Conclusions

Rate of collateral vasoconstriction appears to associate with rate of stroke infarct spread. Neutrophil stalls occluding microvasculature appear to create zones of non-reperfusion post recanalization. ET-1 is a potential upstream therapeutic target for the vasoconstriction and neutrophil adhesion signaling cascades.

Questions on assigned journal club paper – Sensory related neural activity regulates the structure of vascular networks in the cerebral cortex:

1. How are normal mechanisms of neurovascular coupling chronically impacted by sensory deprivation mediated reductions in vascular network complexity?
2. How did genetically-mediated reductions in thalamocortical signaling preserve cortical cytoarchitecture while lesioning afferents did not?