Abstract: 8S

Harnessing non-invasive ultrasound to understand brain blood flow regulation following stroke

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Stroke is an acute cerebrovascular disorder and a leading cause of death and long-term disability. Although stroke can be ischaemic or haemorrhagic, the large majority of strokes are caused by ischaemic events interrupting blood flow to a region of the brain. Acute ischaemic stroke therapy aims to remove the occluding factor (usually a blood clot) through either pharmacological (tissue plasminogen activator) or physical means (endovascular thrombectomy). The overall goal is to re-open the occluded vessels to enable blood flow to return to the ischaemic region. Importantly, even after clot removal, brain damage continues to spread resulting in expansion of the ischaemic core into the surrounding brain regions known as the penumbra – this ultimately increases brain damage and resulting disability. While clot removal effectively restores large artery flow, the extent to which blood flow returns to the microvasculature (which supplies neurons with nutrients) and how microvascular blood flow changes over time after a stroke remain to be fully determined.

We have recently pioneered the application of non-invasive transcranial contrast enhanced ultrasound (tCEU) for real-time assessment of brain blood flow in rodents. This method uses an intravenous, tracer infusion of phospholipid microbubbles to visualise and quantify real-time changes in blood flow across an entire hemisphere or within discrete regions of the brain. We have applied tCEU to directly quantify cerebral blood flow before, during and after ischaemic stroke (intraluminal filament middle cerebral artery occlusion [MCAO] model) in rats. Surprisingly, we found that the ischaemic regions of the brain have substantially increased blood flow above baseline levels immediately following stroke. Animals that have co-morbidities such as type 2 diabetes, where vascular function is already compromised, exhibit further increases in cerebral blood flow immediately after stroke that predict brain damage two weeks later. This talk will provide an overview of our current understanding of cerebral blood flow post-stroke and propose that normalizing blood flow may be a viable therapeutic strategy for reducing brain damage after stroke.