EDUCATIONAL OBJECTIVES

1. Understand the importance of the blood brain barrier and capillary endothelial permeability in the pathophysiology of brain tumors, stroke and other pathology
2. Understand the definition of $K_{\text{trans}}$ and how it is measured in DCE-MRI including the effect of flow and surface area
3. As a blessing, understand some of the theory of how DCE-MRI can be used to assess brain capillary endothelial permeability
4. As a curse, understand how endothelial leakage can cause underestimation or overestimation of perfusion metrics such as rCBV from DSC-MRI
5. Review the clinical applications of DCE-MRI and DSC MRI in clinical management of patients

PRESENTATION SUMMARY:
The blood brain barrier is an important physical barrier which can be compromised by various pathologies such as tumor and stroke, where it is felt to be mediated by agents such as vascular endothelial growth factor, VEGF (also called vascular permeability factor, VPF) or matrix metalloproteinase, MMP-9 respectively. $K_{\text{trans}}$ is a measure of the capillary endothelial permeability, however although it is permeability weighted, it is also affected by flow rate and the surface area for leakage. Various pharmacokinetic modeling methods can be used to estimate $K_{\text{trans}}$ or vascular permeability from T2 based DCE MRI (Dynamic Contrast Enhanced MRI) technique. However, the endothelial leakiness can also be a curse in causing underestimation or overestimation of rCBV when doing T2* DSC MRI (Dynamic Susceptibility Contrast MRI). These pitfalls will be reviewed along with some of the potential clinical applications of perfusion and permeability imaging (1-3). We will also review the metrics which can be derived from a 2 compartment model such as the extravascular, extracellular space (EES) plasma volume ($V_p$). Determine how automation and standardization is required to move these tools to the clinic. Finally demonstrate how histogram techniques can be used in an automated fashion to determine glioma biology, differentiate tumor progression from therapeutic necrosis and characterize pseudoprogression and pseudoresponse.

REFERENCES: