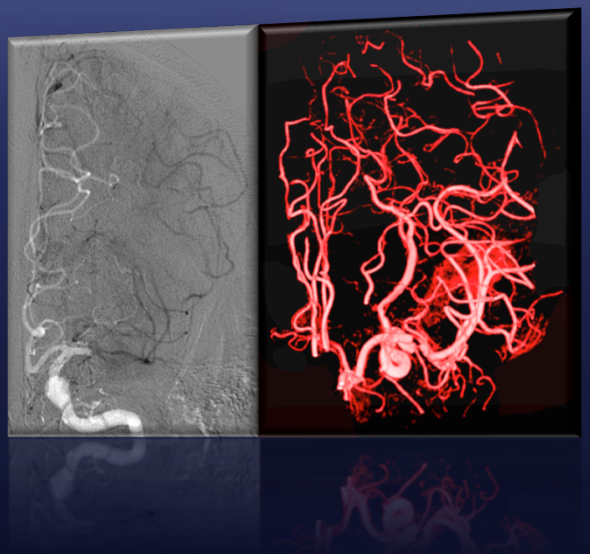
Novel Imaging of ICAD



David S Liebeskind, MD

Professor of Neurology & Director of Stroke Imaging

Associate Neurology Director, UCLA Stroke Center

October 26, 2013

Consultant to Stryker, Covidien

NIH/NINDS P50NS044378, K24NS072272, R01NS077706, R13NS082049

UCLA

SVIN Society of Vascular and Interventional Neurology 6th Annual Meeting

imaging of ICAD

- plaque may cause deformation of the arterial lumen (stenosis), yet many other features and mechanisms
- imaging evaluation of ICAD beyond stenosis:
  - characterization of plaque architecture
  - detailed anatomy of luminal contour
  - flow measures across diseased arterial segments
  - downstream microemboli
  - territorial perfusion abnormalities and dynamic response
- noninvasive modalities – TCD and multimodal CT or MRI for both anatomic and functional detail of such lesions

imaging of ICAD

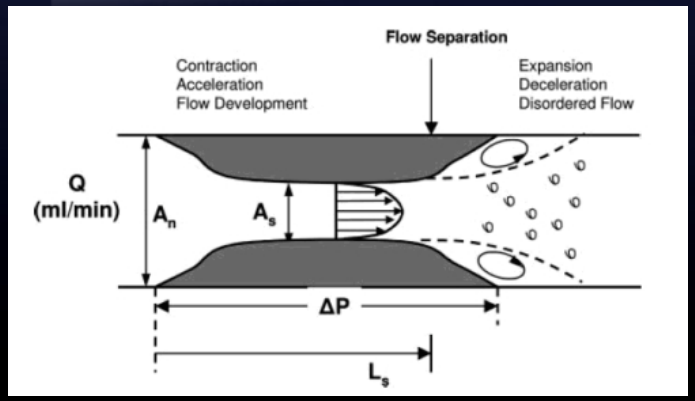
- novel approaches to measure flow across these lesions with fractional flow techniques and computational fluid dynamics (CFD) may be used to select only the highest risk patients that require invasive angiography

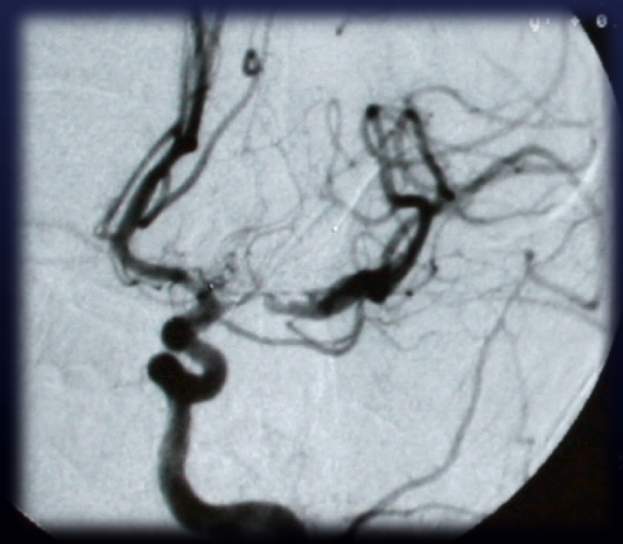
- perfusion angiography and CFD post-processing techniques may also be used on conventional angiography to ascertain risk of subsequent ischemia

atherosclerosis, not just stenosis

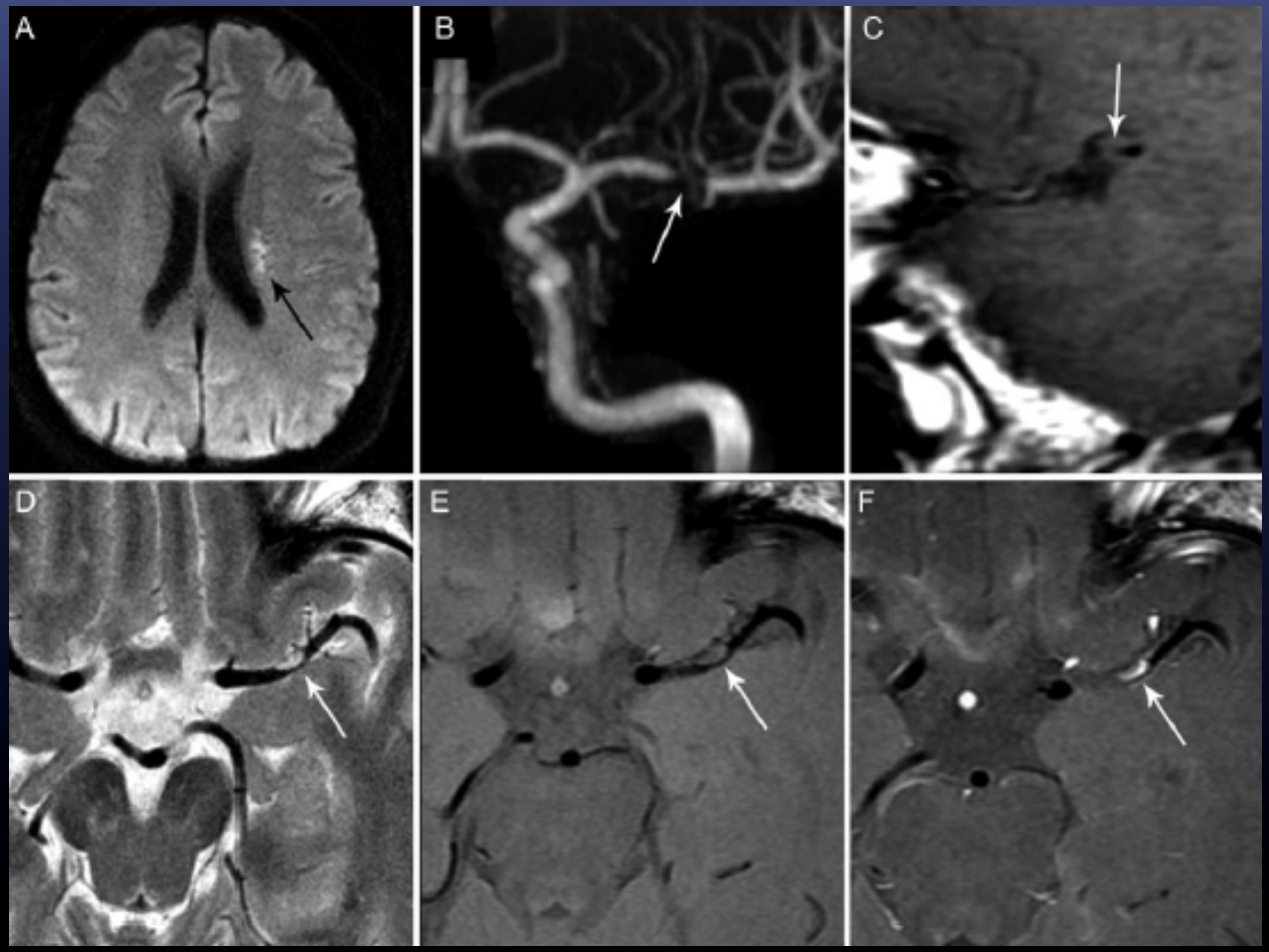
- physical properties, length, architecture, surface irregularity, diffuse disease, tandem disease
- % stenosis is a poor descriptor of intracranial atherosclerosis, not accounting for complexity of lesions and diffuse disease

$$Q = \pi \Delta P r^4 / 8 L \eta$$



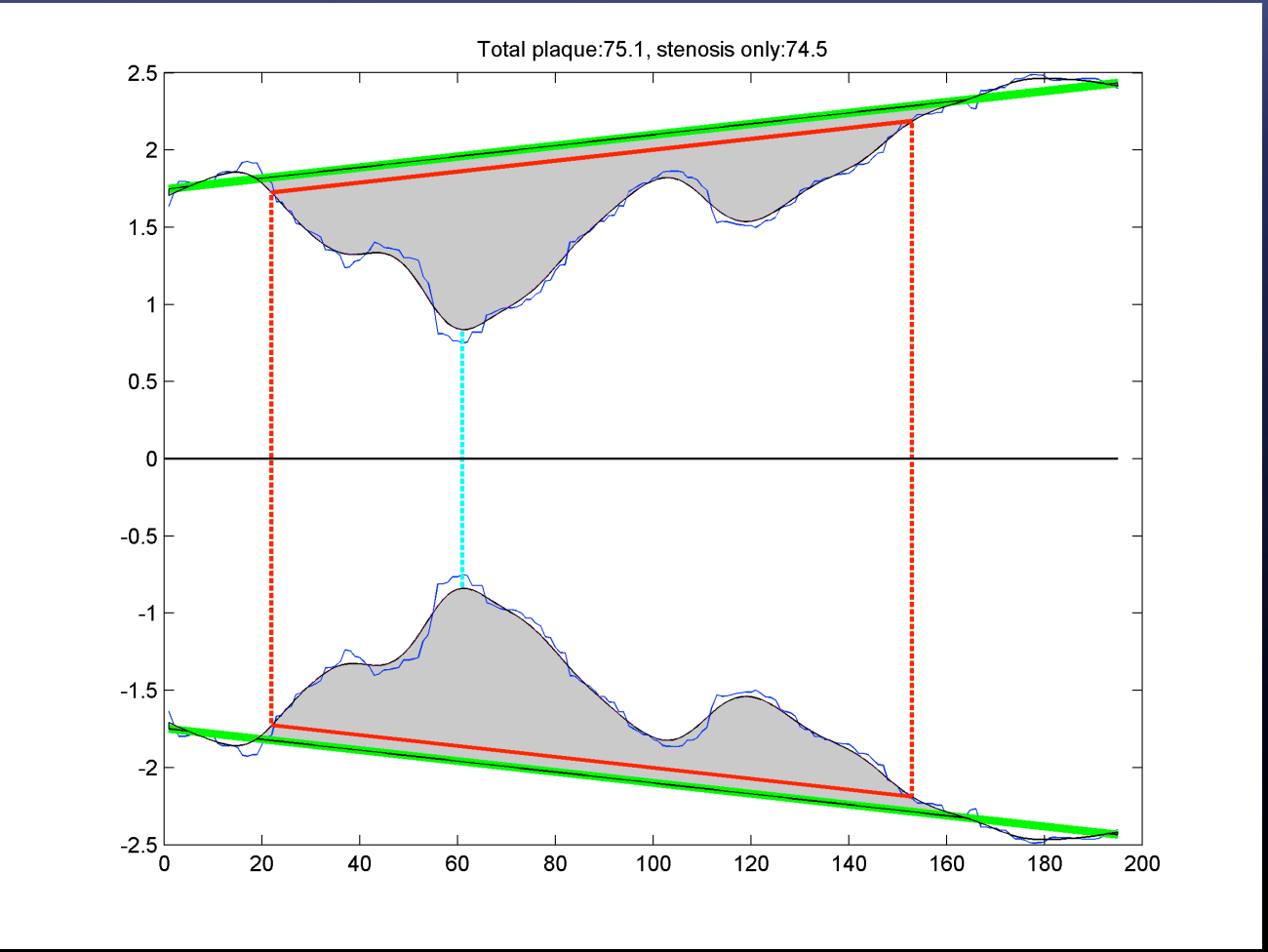


MRI of ICAD



Intracranial arterial wall imaging using high-resolution 3-tesla contrast-enhanced MRI
R. H. Swartz, S. S. Bhuta, R. I. Farb, et al.
Neurology 2009;72;627

luminal contour

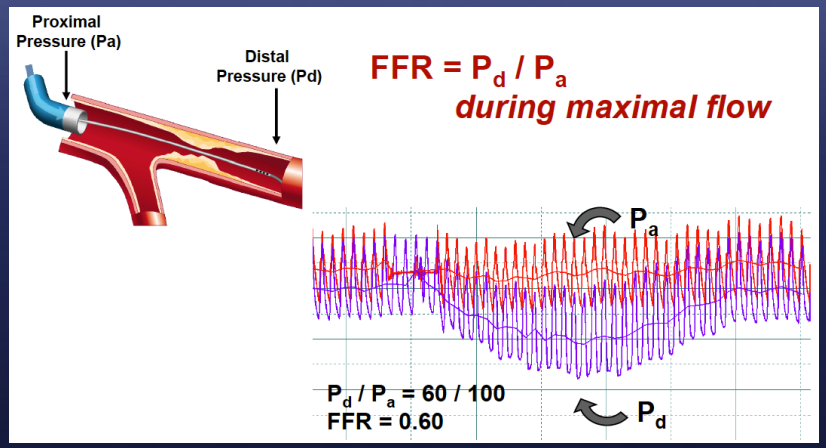


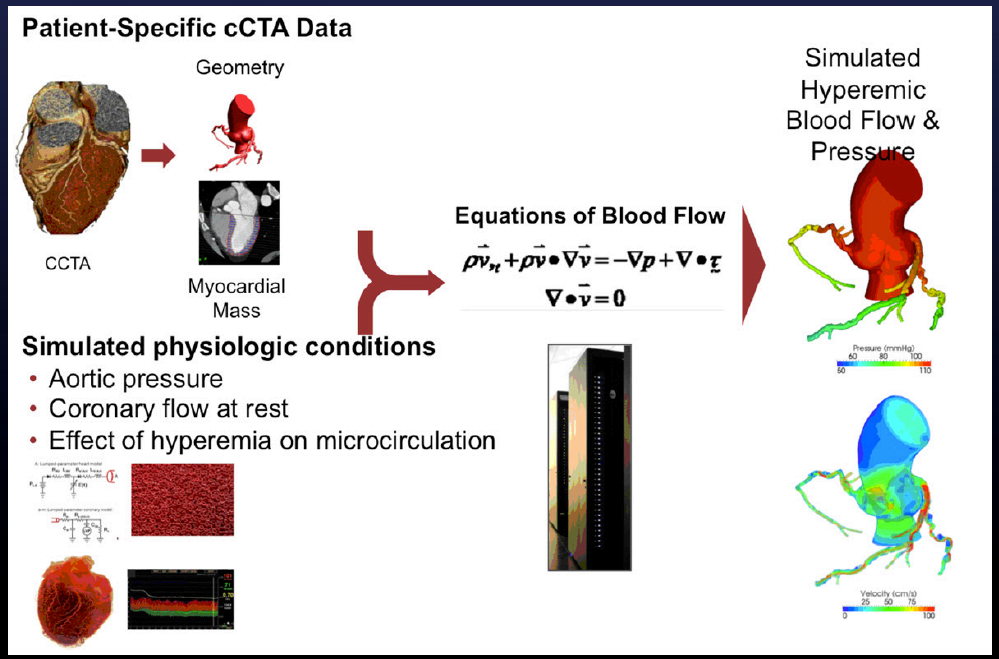
myriad mechanisms

- recurrent stroke risk predicated on mechanisms
  - decreased flow due to arterial narrowing
  - impaired tissue perfusion
  - plaque instability with perforator occlusion and/or distal embolism

Patient A (left) has low QMRA volumetric flow rate (28 mL/min) with impaired angiographic collaterals (ASITN grade 2), and partial angiographic perfusion (TICI grade 2B). Patient B (right) has normal QMRA flow (75 mL/min), complete angiographic perfusion (TICI grade 3), normal vasomotor reactivity (BHI 1.0), but 2 embolic signals were detected distal to the stenosis (1 ES noted by arrow).

fractional flow





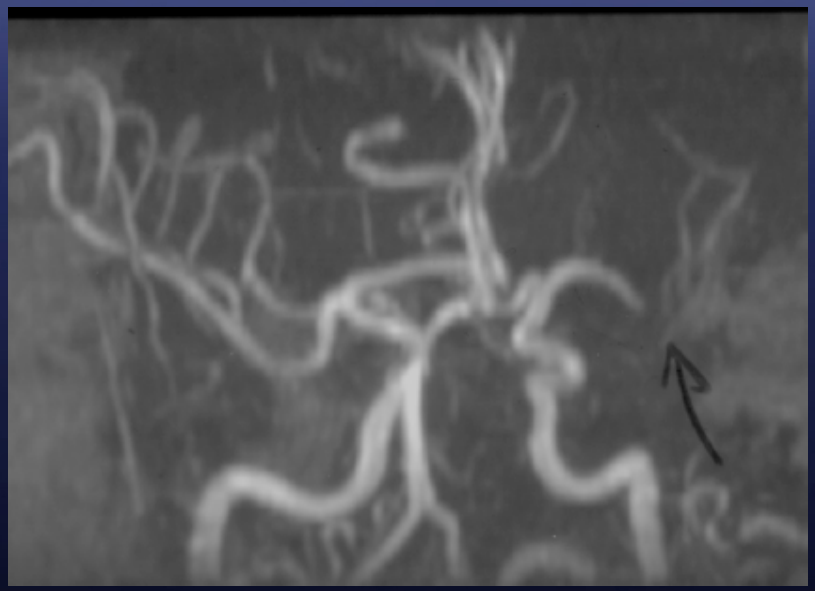
fractional flow

- large, randomized trials established FFR for lesion-specific ischemia
- fractional flow measures proven:
  - enhance clinical decision-making
  - improve event-free survival
  - reduce unnecessary revascularization
  - lowers costs
- FAME, FAME II, DeFACTO

- anatomical luminal stenosis replaced by hemodynamics

TOF-MRA SIR predicts stroke

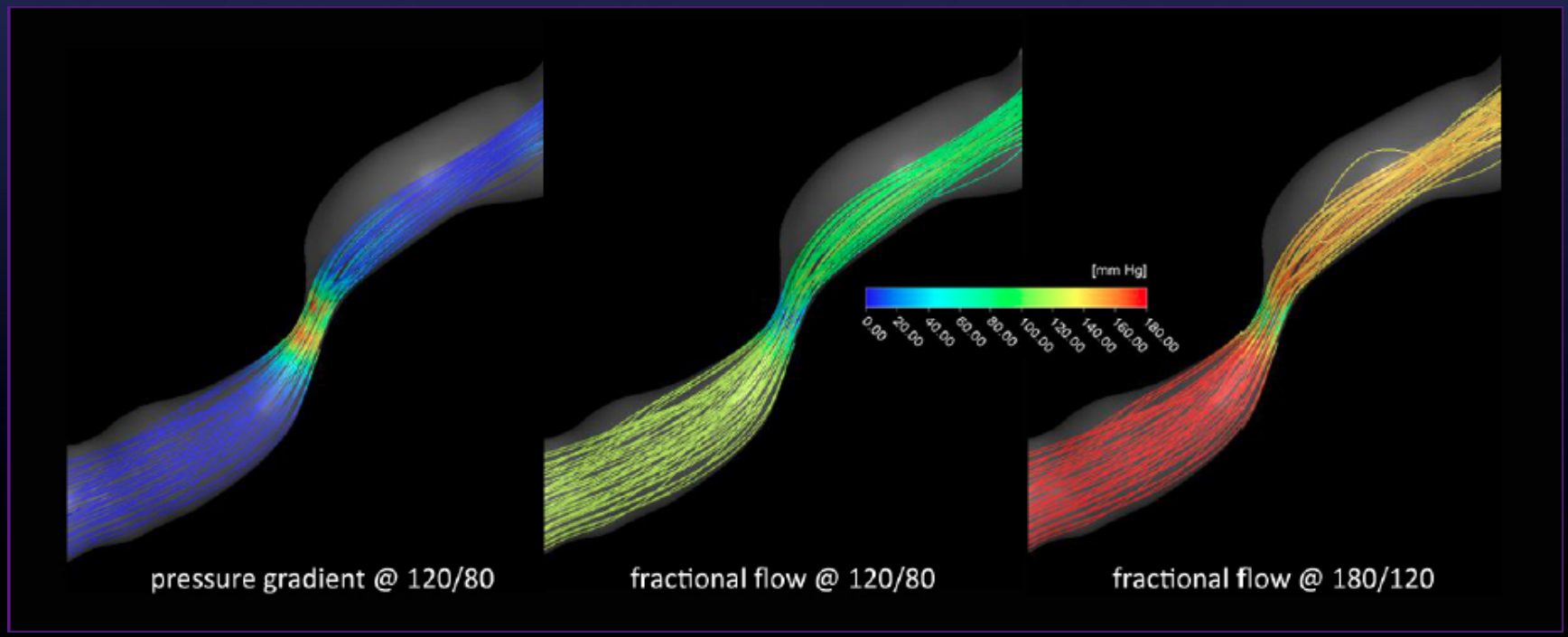
- SONIA-WASID analyses
- analyses included clinical variables, SIR, and invasive angiography measures (luminal stenosis, TICI score of antegrade flow, collateral grade) to identify predictors of stroke in the territory
- 189 patients with 50-99% symptomatic IAD had TOF-MRA available
- univariate analysis, the hazard ratio (HR) for SIT of the symptomatic artery with SIR<0.9 (SIR below median) was 5.2 (1.8, 15.3; p=0.001) as compared to SIR≥0.9
- multivariate analysis correcting for baseline blood pressure, LDL, percent stenosis, recency of symptoms, TICI and downstream collaterals, the HR for SIR<0.9 was 10.9 (2.0, 58.9; p=0.001)
- only collaterals also had a significant independent association with stroke risk, HR 13.8 (3.4, 55.5; p<0.001)
- in patients with <70% stenosis, a SIR<0.9 maintained a significant association with recurrent SIT (p=0.006), with a 2-year event rate of 17.3%, showing that even moderate stenoses can pose substantial ischemic risk



UCLA

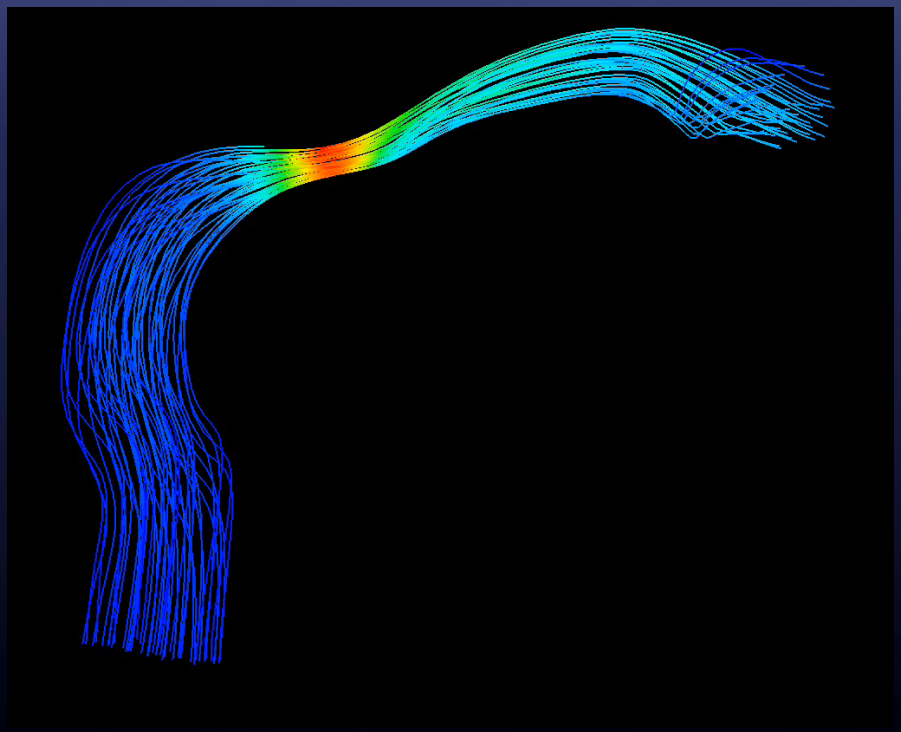
SAMMPRIS CFD

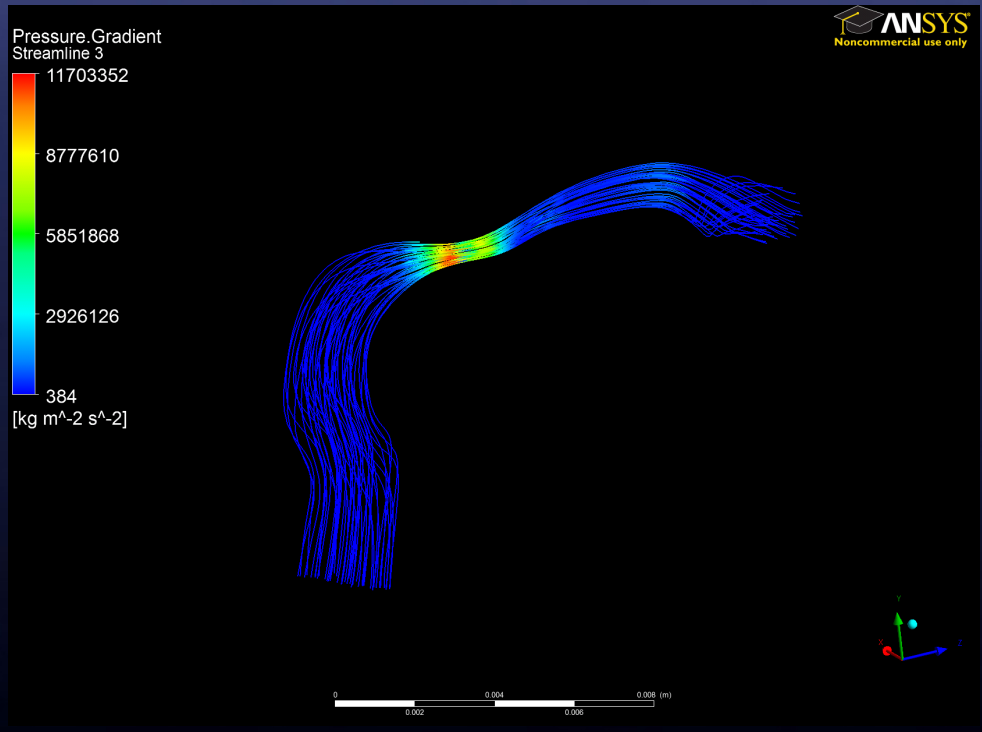
- 407 patients with 70-99% symptomatic stenosis DSA, biplanar views available for 3D reconstruction in 249, and CFD simulations in 188 (25 VA, 45 BA, 32 ICA, 86 MCA)
- under simulated normal inflow conditions (120/80 mm Hg), only 76/188 (40%) cases had low FF
- during simulated hypertension, FF improved to normal in 10/188 (5%) cases
- simulated hypotension caused FF to worsen from normal in 12/188 (6%) cases
- other hemodynamic parameters including shear stress calculated and visually depicted



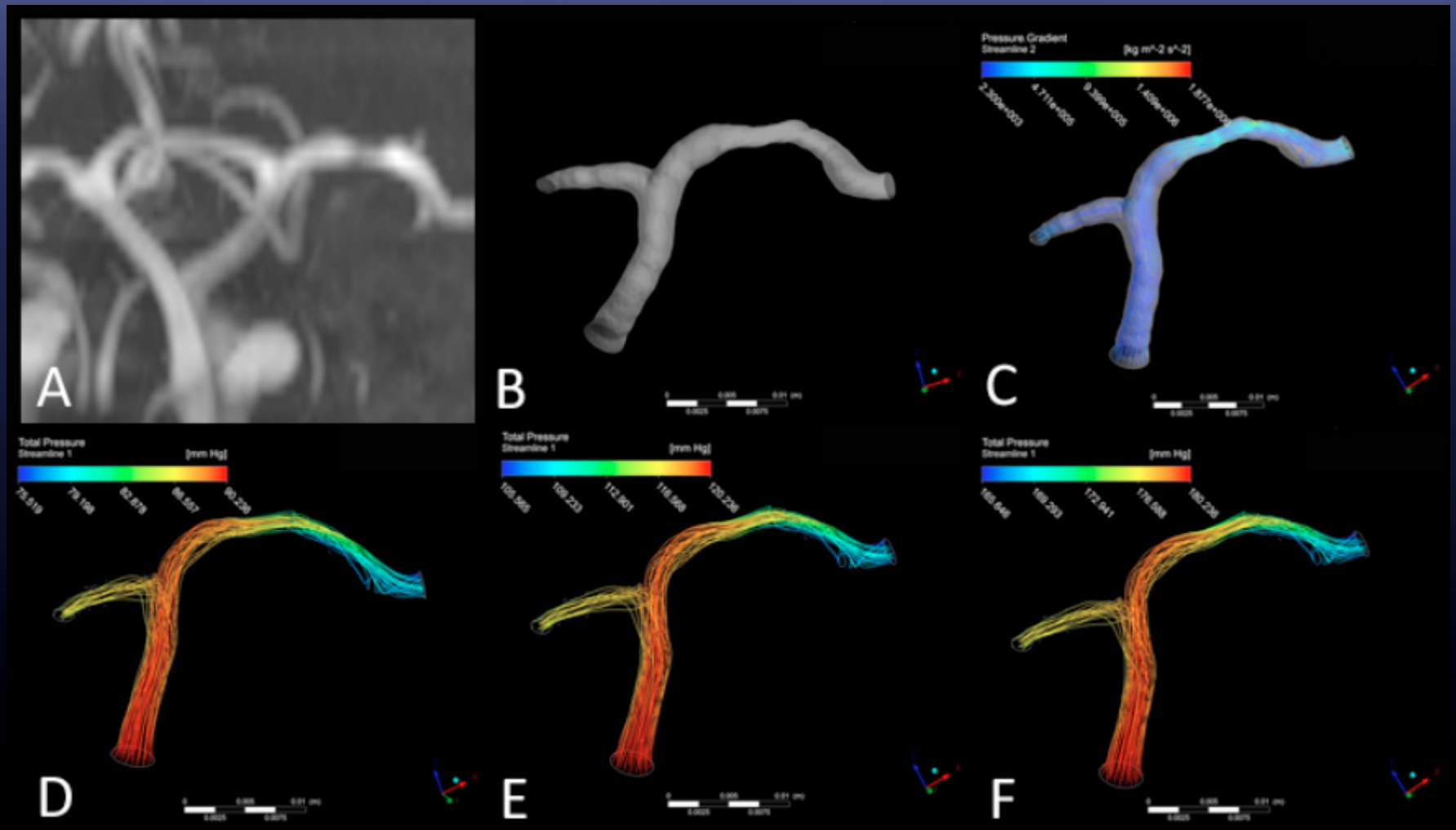
UCLA

CFD – velocity & pressure



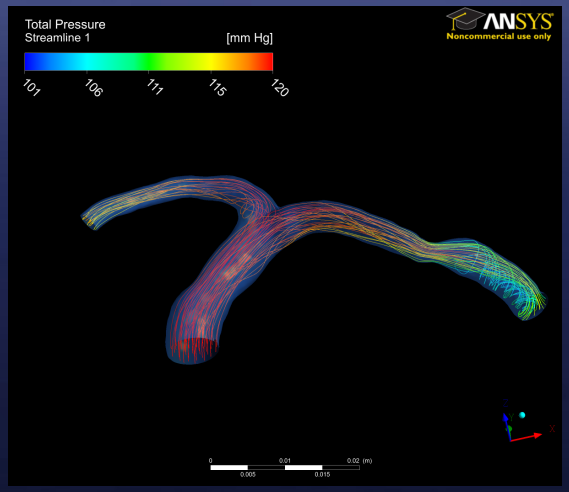


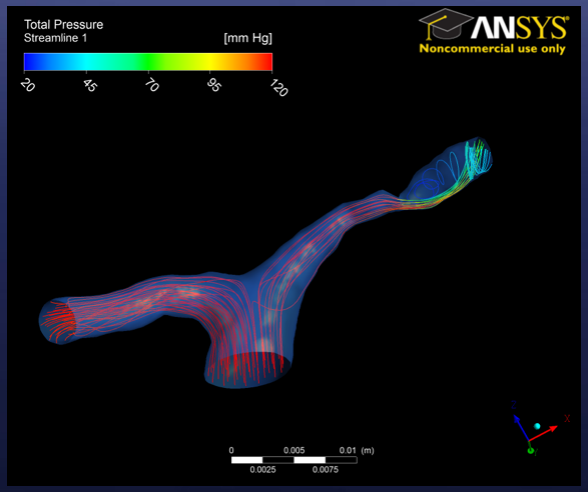
CFD of MRA

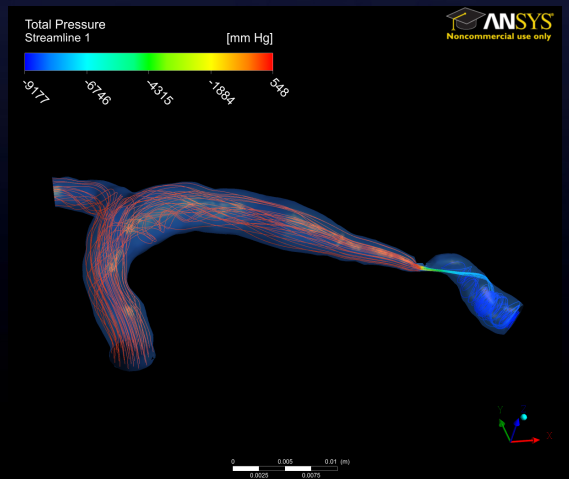


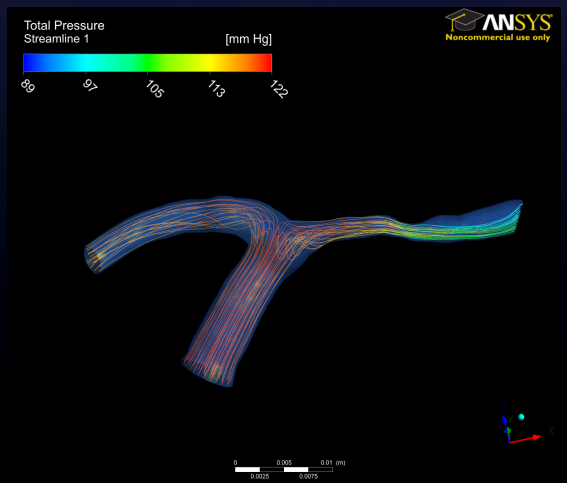
UCLA

CFD of CTA



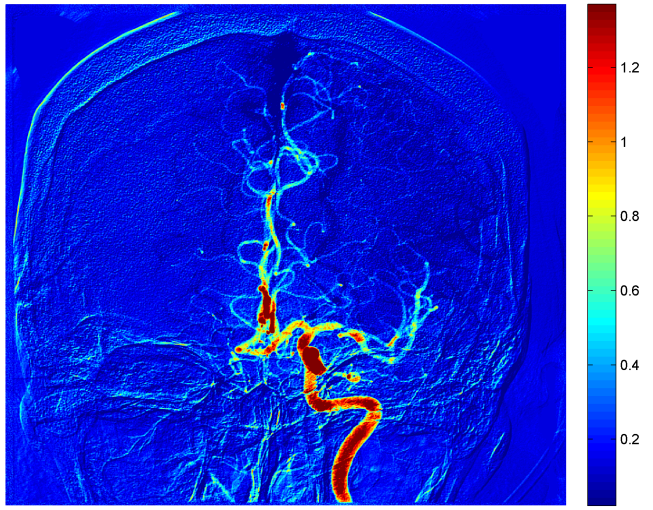


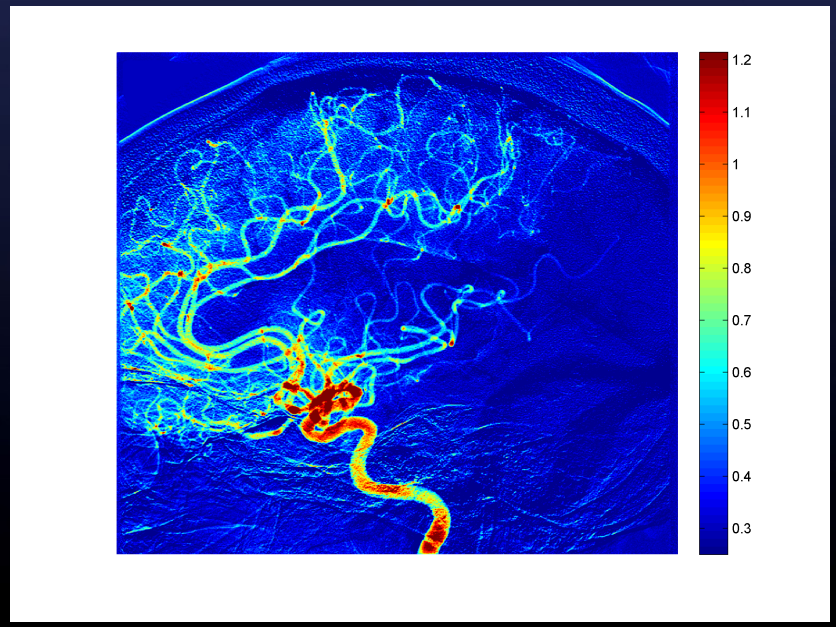




UCLA

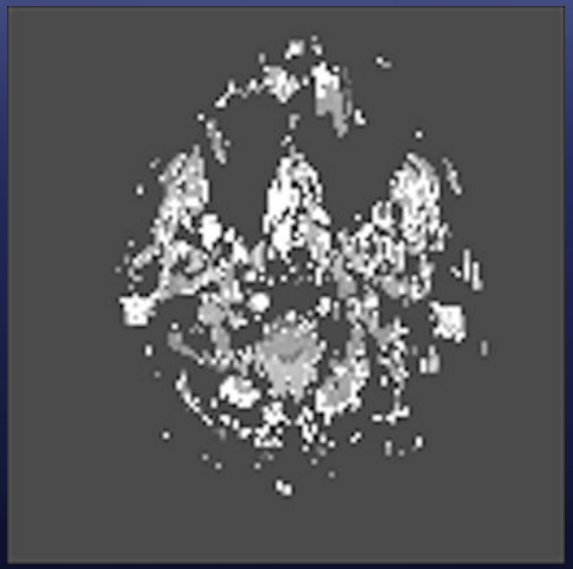
perfusion angiography

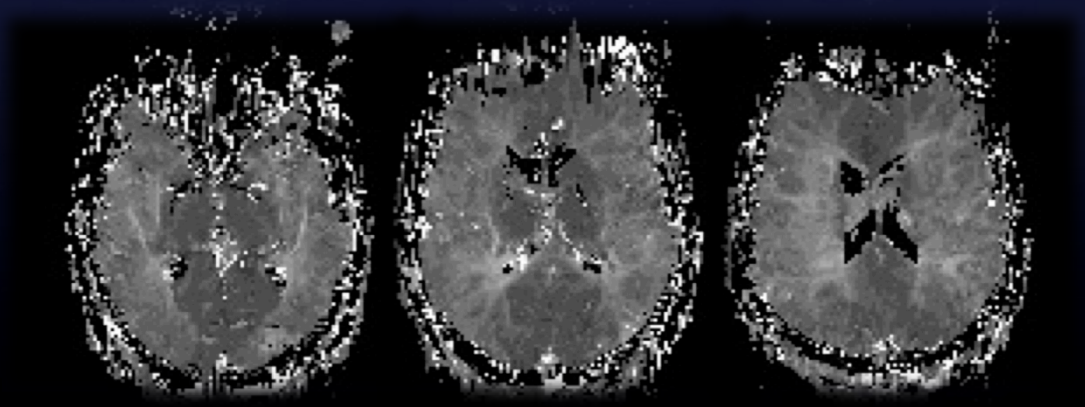




UCLA

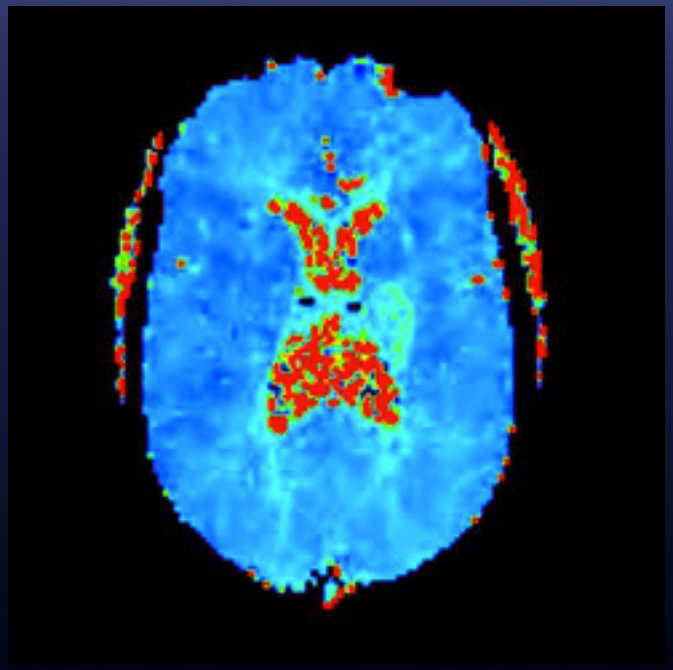
subtle features of ICAD

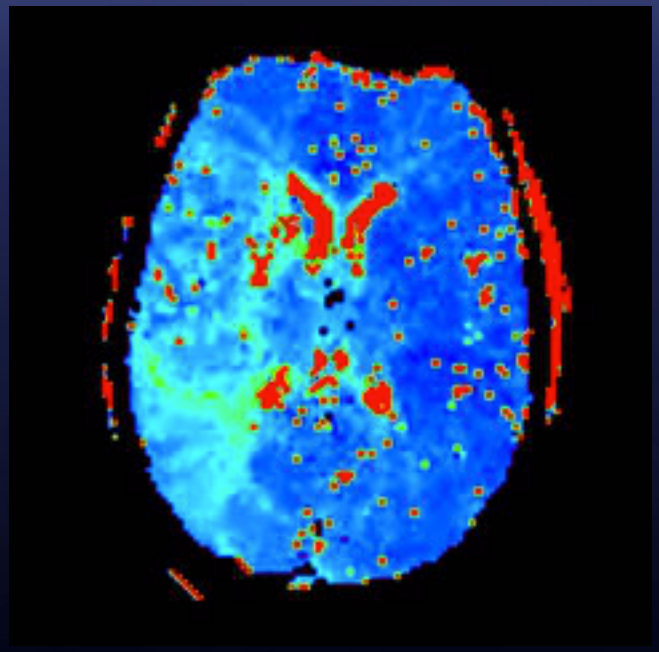




UCLA

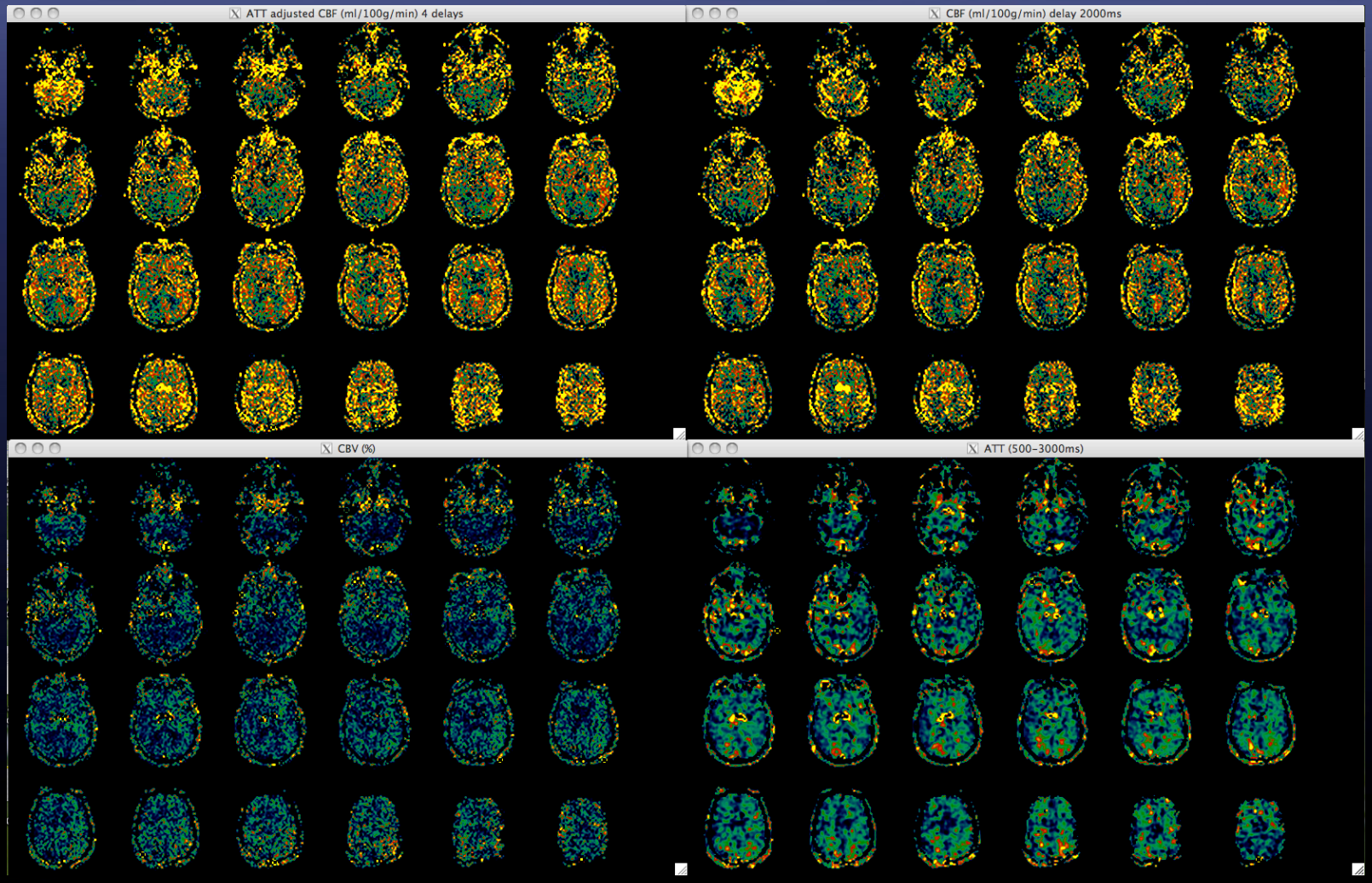
Tmax delays (dynamic CBV) of ICAD





UCLA

ASL of ICAD



UCLA

next steps

- from profusion of imaging features in ICAD extending beyond stenosis to perfusion of brain at-risk – potential to refocus therapeutic efforts on causal mechanisms rather than markers of disease such as degree of luminal stenosis

UCLA