Abstract E-172 Figure 1

and undergoing CTP (between 0–24 hours since last known well) and treated with EVT between March to September 2019 were retrospectively reviewed. Patients with preexisting infarcts were excluded. CTP was processed using software (RAPID, Ischemaview) with HIR automatically calculated. ASPECTS scores were obtained and infarct volumes calculated using the ABC/2 method on the initial noncontrast CT and CT performed 24–72 hours post-EVT. Spearman correlation test were performed between HIR and initial CT ASPECTS, initial CT infarct volume. After excluding 5 patients without successful reperfusion, HIR Spearman correlation with post-EVT CT ASPECTS, final infarct size, and infarct growth was assessed.

Results Thirty-two patients were included in the study (20 males and 12 females), with a mean age of 63.0 years (SD=17.3). Initial ASPECTS scores ranged from 2 to 10 (median 7). Initial infarct volume (IIV) ranged from 0 to 113 ml (median 19). Successful reperfusion (mTICI 2B, 2C, or 3) was obtained in 28/32 (88%). Final ASPECTS scores at discharge ranged from 0 to 10 (median 5.5), and final infarct volume (FIV) ranged from 0 to 367 ml (median 72 ml). There was a significant correlation between HIR and initial ASPECTS (-0.37 , $p=0.03$), and a trend to significance with IIV (0.32 , $p=0.07$) (figure 1A and 1B). With non-recanalizers excluded, a significant correlation was observed between HIR and final ASPECTS (-0.51 , $p=0.006$), FIV (0.50 , $p=0.009$), and infarct growth pre- and post-EVT (0.41 , $p=0.03$) (figure 1B and 1C).

Conclusions In the time-sensitive environment of LVO AIS, HIR allows rapid assessment of collateral status, showing similar correlations with infarct size as CTA collateral scoring. Further investigation is warranted to determine whether HIR is predictive of clinical outcomes.

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E-173 ASSESSING AGE RELATED DISCREPANCIES OF TOTAL CEREBRAL BLOOD FLOW AND CEREBRAL VENOUS OUTFLOW USING 4D FLOW MRI

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Purpose The cerebral arterial inflow, venous outflow, and Cerebrospinal fluid volume and flow are highly regulated in an equilibrium to maintain adequate cerebral perfusion as governed by the Monro-Kellie doctrine. Multiple neurological conditions had been associated with abnormalities in the cerebral venous system including multiple sclerosis and idiopathic intracranial hypertension, among others. We aim to quantitatively study the relationship and discrepancy between total cerebral blood flow (TCBF) and cerebral venous outflow (CVO), as well as the TCBF as a proportion of the cardiac output in adults and how it compares to the pediatric patients, and highlight the discrepancies.

Materials and Methods 31 healthy volunteers (24 adults and 7 children) underwent 4D flow MR imaging for arterial evaluation and 2D CINE PC-MRI for venous evaluation in an IRB approved study. 3D volumetric ECG-gated PC-MRI data with 3-directional velocity encoding (4D flow MRI) was acquired for the arterial system. 2D CINE PC MRI was acquired for the venous system at both transverse sinuses. 4D flow MRI data was processed using in-house software tools in Matlab. We calculated the cumulative TCBF in ml/sec as the sum of inflow in the bilateral ICAs and basilar artery. The CVO was calculated as the sum of the flow in the bilateral transverse sinuses. The ratio of CVO to TCBF was calculated. Also, the TCBF as a percentage of cardiac output was calculated.

Results A significant positive correlation between TCBF and CVO ($r=0.81$ and $P<0.001$) was seen. The TCBF (20.21 ± 4.58 ml/s versus 11.78 ± 2.03 ml/s; $P<0.001$) and CVO (12.80 ± 3.82 ml/s versus 9.03 ± 2.31 ml/s; $P=0.010$) were significantly higher in children compared to adult volunteers. The CVO/TCBF ratio was significantly lower in children versus adult volunteers (0.63 ± 0.01 versus 0.76 ± 0.02 , $P=0.025$). In adults, the correlation of TCBF with age remains strong ($\rho = -0.69$, $t\text{-stat} = -4.5$, $P=0.00018$). However, CVO ($\rho = -0.29$, $t\text{-stat} = -1.42$, $P=0.171$) and CVO/TCBF ratio ($r=0.16$, $P=0.446$) were not significantly associated with age in the adult cohort. The ratio of cerebral arterial inflow to systemic aortic outflow was significantly higher in children compared to adults (0.45 ± 0.08 versus 0.15 ± 0.02 , $P<0.001$).

Conclusions Both TCBF and CVO decrease with age, however unlike TCBF, there is no correlation between the decrease in CVO through the Transverse sinuses and age, which could suggest the early development of alternative venous drainage pathways through the emissary and extracranial veins. This could also explain the differential ratio of CVO to TCBF, which suggests that more than 20% of cerebral venous outflow in adults and more than 35% of outflow in Children are not through the Transverse sinuses in the supine position. Understanding the quantitative differences between TCBF and CVO in healthy volunteers could help identify and manage changes related to venous outflow abnormalities.

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E-174 RECOGNITION OF SAH PATIENTS AT HIGH RISK FOR SYMPTOMATIC VASOSPASM: RESPONSES TO ACETAZOLAMIDE CHALLENGE

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Introduction Cerebral vasospasm can be a devastating sequela of aneurysmal SAH. There is neither a satisfactory predictor nor a standardized protocol for its early detection. We evaluated cone beam CT perfusion (CBCTP) with acetazolamide challenge as a potential tool to detect deficiencies in autoregulation prior to development of symptomatic vasospasm.

Methods Ten patients presenting with aneurysmal SAH within 24 hours of symptom onset were enrolled in this pilot study. Just prior to the initial DSA study, a baseline CBCTP acquisition was performed (60 cc contrast, 60 cc saline). Acetazolamide (1 g IV) was administered and, after a 20-minute delay, a second CBCTP acquisition was done. Data was then reconstructed into perfusion maps (CBF, CBV, and MTT). Delay insensitive deconvolution was used and AIFs were manually selected. Motion correction was applied if possible. Data was analyzed for percent change in perfusion maps in response to acetazolamide, variation between cerebral hemispheres, and

study related complications. Percent change in CBF with a p value <0.05 was defined as significant.

Results Perfusion maps were successfully acquired for all 10 patients. Our analysis was performed on 20 cerebral hemispheres (right and left). Nineteen hemispheres demonstrated a significant change in CBF in response to acetazolamide ($p<0.05$). Six hemispheres demonstrated a 30% change in CBF and 12 had a 15% change. In 9 hemispheres there was an appropriate increase in CBF in response to acetazolamide. In 10 hemispheres there was a decrease in CBF. There was no statistical difference in the percent change between left and right hemispheres which would correlate with the global vasodilatory effects of acetazolamide. Symptomatic vasospasm developed in 2 patients. One vasospasm patient had a pathological decrease in CBF in response to acetazolamide while the other had an appropriate increase in CBF. There were no study related complications.

Abstract E-174 Figure 1 Relative percent change in CBF calculated for 10 patients (20 hemispheres). Positive percent change correlates with increase in CBF and negative with decrease in CBF. Perfusion maps pre and post acetazolamide demonstrate qualitative increase in CBF for case 4

Conclusion Accurate early identification of patients at high risk for symptomatic vasospasm would be a valuable clinical tool in the management of aneurysmal SAH. This small pilot study shows the feasibility of CBCTP with acetazolamide challenge to generate reproducible, diagnostic quality perfusion maps with a response to acetazolamide. These provide the opportunity to evaluate the vasodilatory capacity or state of autoregulation at a time before vasospasm is angiographically or symptomatically evident. A larger study is ongoing to provide data which will help to better understand the significance of changes observed in our study.

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