SUPPLEMENTAL MATERIAL

Data S1.

Supplemental Methods

To assess if the relationship between CBF pulsatility and rCVR is driven by overall CBF we compared rCBF to CBF pulsatility and rCVR, respectively. rCBF was extracted over a one-minute interval during hyperemia (~10min post-ROSC), early hypoperfusion (~20min post-ROSC), and late hypoperfusion (~60min post-ROSC). To determine the association between overall CBF and CBF pulsatility, a Spearman ranked correlation was used during each phase. To determine the association between overall CBF and rCVR, a Spearman ranked correlation was used during each phase.

We performed chronic cranial window experiments to assess if an open or closed cerebral system affects CBF pulsatility. The following procedures were performed. First, the skull was removed, similar to our open craniectomy experiments; second, the dura was removed; third, agarose was placed over the exposed brain; lastly, a coverslip was placed on top and glued to the skull. After surgery, rats were given approximately three weeks before induction of ACA. 5min ACA was used for chronic cranial window experiments. We compared CBF pulsatility from the open craniectomy experiments to the chronic cranial window preparation.

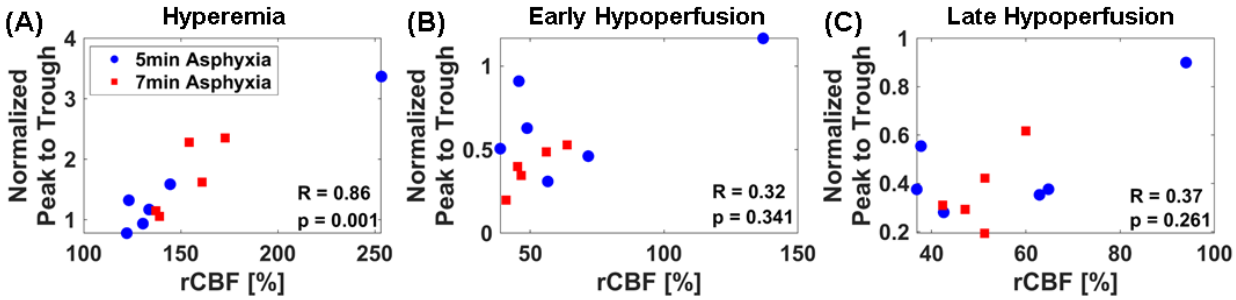
Supplemental Results

Our data shows that rCBF and CBF pulsatility were significantly correlated during hyperemia ($R = 0.86$, $p = 0.001$) (Figure S1A). However, there was no significant correlation during early ($R = 0.32$, $p = 0.341$) (Figure S1B) and late hypoperfusion ($R = 0.37$, $p = 0.261$) (Figure S1C).

There was no significant association between rCVR and rCBF during hyperemia ($R = -0.52$, $p = 0.107$) (Figure S2A), early ($R = -0.35$, $p = 0.286$) (Figure S2B) and late hypoperfusion ($R = -0.58$, $p = 0.066$) (Figure S2C) Although there was a trend towards significance during late hypoperfusion.

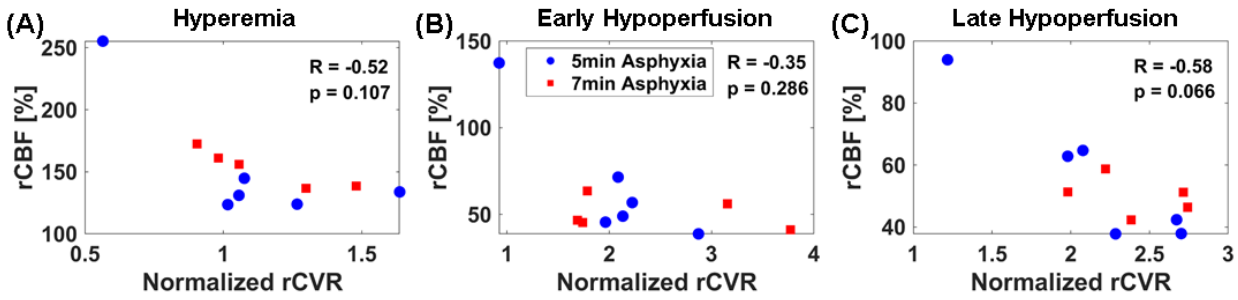
Our data show good agreement between the open craniectomy and chronic cranial window preparations (Figure S3). Furthermore, CBF pulsatility from chronic cranial window preparation is on the lower end of the pulsatility values measured with the open craniectomy preparations. This observation suggests that brain pulsations may potentially be causing a mild artifact in our open craniectomy data. Thus, the open craniectomy experiments may show an artificial increase in CBF pulsatility due to brain pulsations. Nevertheless, this control experiment supports overall corroborating trends between the open and closed craniectomy models during the different phases of cardiac arrest and post-cardiac arrest time points and supports our main conclusions.

Figure S1. Cerebral blood flow (CBF) pulsatility and overall CBF are associated during hyperemia, but not early and late hypoperfusion.



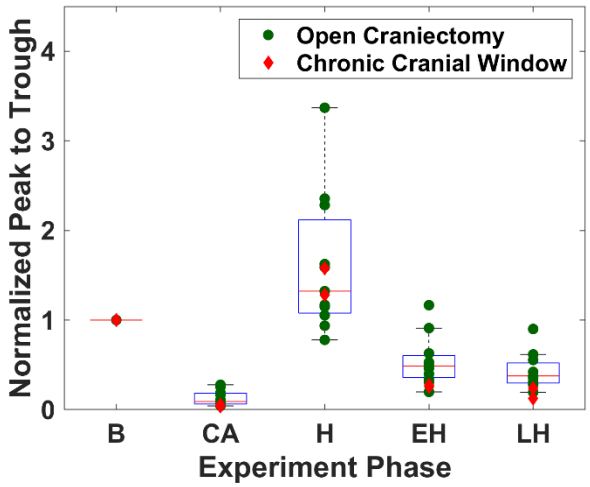
(A) Comparison of CBF pulsatility and overall CBF during hyperemia (R = 0.86, p = 0.001). (B) Comparison of CBF pulsatility and overall CBF during early hypoperfusion (R = 0.32, p = 0.341). (C) Comparison of CBF pulsatility and overall CBF during late hypoperfusion (R = 0.37, p = 0.261).

Figure S2. Overall cerebral blood flow (CBF) and relative cerebrovascular resistance (rCVR) are not significantly associated post-cardiac arrest (CA).



(A) Comparison of overall CBF and normalized rCVR during hyperemia (R = -0.52, p = 0.107). (B) Comparison of overall CBF and normalized rCVR during early hypoperfusion (R = -0.35, p = 0.286). (C) Comparison of overall CBF and normalized rCVR during late hypoperfusion (R = -0.58, p = 0.066).

Figure S3. Chronic cranial window cerebral blood flow (CBF) pulsatility agrees with open craniectomy CBF pulsatility.



The open craniectomy data (n=11) is shown in green and the chronic cranial window data (n=2) is shown in red. Experimental phases are baseline (B), cardiac arrest (CA), hyperemia (H), early hypoperfusion (EH), and late hypoperfusion (LH).