

Ultralow frequency vaso-oscillations in human cerebral arteries occur independently from Mayer waves

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Running headline "Robust brain vasomotion during cardiac bypass"

24 **Abstract**

25 **This study tests the hypothesis that ~0.1 Hz cerebral vaso-oscillations are generated**
26 **intrinsically by the arterioles that perfuse the brain, and not by external drive from**
27 **systemic blood pressure oscillations (Mayer waves). During cardiopulmonary bypass**
28 **that transiently eliminated systemic blood pressure oscillations in 14 patients, we**
29 **observed that vasomotor oscillations persist with normal amplitudes and frequencies**
30 **over the one- to three-hour time course of surgery. In contrast, ~ 0.1 Hz oscillations in**
31 **peripheral blood pressure were predominantly absent. This implies that cerebral**
32 **arterioles generate their own rhythmic vaso-dynamics, although we cannot discount**
33 **that vasomotion can phase-lock with ~ 0.1 Hz systemic physiological rhythms in the**
34 **awake, healthy state. We discuss the impact of this finding on the role of vasomotion**
35 **in modulating the perfusion of blood and the transport of interstitial fluid in the brain.**

36 **Keywords:** cardiopulmonary bypass; cerebral arterioles; intramural periarterial drainage,
37 resting-state; vasomotion

Introduction

Perfusion to the brain is maintained during variations in peripheral blood pressure through autoregulatory changes in the diameter of major and minor feeding arteries to the organ¹. Dynamic cerebral autoregulation acts within seconds on vascular smooth muscle cells (vSMCs), and consequently, slow peripheral blood pressure oscillations can entrain oscillations in cerebral artery diameter. This occurs during both experimentally forced blood pressure changes^{2,3} as well as during spontaneous Mayer wave events, which are observed during resting conditions at ~ 0.1 Hz in humans^{4,5}. Arteries throughout the body also undergo ~ 0.1 Hz vasomotion driven by intrinsic vSMC activity^{6,7}. Vasomotion has been observed in various tissues and species, including intraoperatively in pial arterioles of awake humans⁸. However, dissociating intrinsic vasomotion from sources of external drive is notoriously difficult because of the various central and peripheral influences that can entrain cerebral arterial activity⁹⁻¹³. Here we evaluate the hypothesis that cerebral arterial oscillations can occur independently from Mayer waves by utilizing cerebral blood flow data acquired from human patients undergoing cardiopulmonary bypass surgery.

Vasomotion has recently gained attention for its potential contribution in intramural periarterial drainage (IPAD)¹⁴, which facilitates the removal of toxic metabolites, including amyloid- β (A β) peptides, that are implicated in Alzheimer's disease. IPAD relies on ~ 0.1 Hz vasomotion for the efficient clearance of A β and other metabolic waste^{14,15}. The mechanism for net flow in the face of oscillatory fluidic motion is experimentally unresolved¹⁶, although vasomotion is predicted to induce a net volume flow rate¹⁴ owing to the asymmetry of the arteriolar oscillations during functional hyperemia^{17,18}, the potential for peristalsis^{14,19}, and asymmetric flow through poroelastic tissue²⁰. Lastly, in aging and Alzheimer's disease, cerebrovascular function is often disrupted, leading to reduced waste clearance, the accumulation of A β plaques, and exacerbated neurodegeneration and cognitive decline²¹.

For both reasons of fundamental physiology and biomedicine, it is crucial to identify the driving force for cerebral vasomotion²². Oscillations in cerebral perfusion pressure, termed Mayer waves, are driven by feedback between sympathetic nerve activity and changes in

peripheral vascular bed resistance⁴. In rodents, the Mayer wave frequency is approximately 0.4 Hz²³, whereas in humans the frequency is near 0.1 Hz and thus overlaps spectrally with vasomotion and obfuscates the origin of ~0.1 Hz hemodynamic fluctuations. To disambiguate vasomotion from Mayer waves, we sought to measure cerebral blood flow in the absence of Mayer waves by artificially stabilizing systemic blood pressure²⁴.

One of the most significant advances in the management of heart diseases came from the introduction of cardiopulmonary bypass (CPB)²⁵. A machine is connected to the heart to divert blood away from it and the lungs while operating on an arrested “motionless” heart, typically to replace diseased valves or correct congenital malformations. The CPB machine supports the systemic circulation and gas exchange for the whole body, including the brain. The flow, speed, pressure, volume, temperature, pH, and blood gas concentrations can be manipulated to maintain “normal” physiological homeostasis as much as possible. CPB also allows us to create abnormal states, such as cooling the blood to reduce brain metabolic activity, reducing the cerebral oxygen requirements and allowing reduced blood flow to the body for short spells while facilitating a complex cardiovascular reconstructive procedure²⁶. Since CPB removes cardiac variability and artificially stabilizes aortic pressure, it provides the ideal condition to test if cerebral blood flow oscillations depend on systemic Mayer waves.

Material and Methods

Patients and procedures

A protocol-driven observational study was set up under ethical approval - Integrated Research Application System (IRAS) (Number 264726); complete statements on ethical considerations are given at the end matter. Participants were recruited from patients who were admitted for cardiothoracic surgical procedures. All patients had general anesthesia with routine monitoring of radial arterial blood pressure (ABP), central venous pressure (CVP), arterial blood gases, central and peripheral temperatures, heart rate, pulse oximetry and ECG. These were connected to a monitor for continuous data display and recording

(MetaVision®, iMDsoft, Tel Aviv, Israel). The CPB system was LivaNova Sorin Stockert S5, widely used in cardiopulmonary bypass. This system contains a peristaltic pump that was set to deliver a constant flow, as opposed to a pulsatile flow or a constant pressure. Nonetheless, the frequency of the pump in the CPB machine is BPM/60 Hz, where we used a BPM of 120/minute, so the pump head generated pulsatile oscillations at 2 Hz. There is no internal mechanism in the pump that generates a 0.1 Hz signal. A final point is that the ventilator, which runs at 0.2 Hz, is only on before and after, but never during, CPB. Thus the ventilator cannot be a source of 0.1 Hz during CPB.

Ethical considerations

This observational study received ethical approval from the Integrated Research Application System (approval #264726) on 04/01/2019. All patient information was de-identified and patient data will not be shared with third parties. The protocol has been agreed and accepted and the Chief Investigator agreed to conduct the study in compliance with the approved protocol and will adhere to the principles outlined in the Declaration of Helsinki.

TCD recording

There was additional monitoring of the middle cerebral artery using a transcranial Doppler ultrasound (TCD) Dopplerbox (DWL GmbH, Singen, Germany). This was connected via an analogue to digital converter, to a PC which was streaming digital outputs from the clinical monitors. The TCD and CPB signals were simultaneously recorded at 240 Hz using ICM+ software (ICM+, Cambridge Enterprise, Cambridge, UK, (<http://www.neurosurg.cam.ac.uk/icmplus>).

After the patient had been transferred to theatre, and the anesthetic team were satisfied with their lines and monitoring, surgery began, and the transcranial Doppler probe was adjusted to obtain an optimal signal from either left or right middle cerebral artery. Recording started as soon as a good signal had been obtained. It was maintained throughout the operation until wound closure. The arterial blood pressure varied around a mean of 65 mmHg. From the full length of TCD recording we manually discarded times when the signal was either completely absent or corrupted by too much motion or diathermy artifact

(**Figure 1**). We considered a clear TCD signal of 5 minutes in length as “good” data for analysis. All good-quality data sections of 5 minutes duration were used in the analysis. One trial of 3.6 minutes and one of 4 minutes were also included. Data sections were identified according to the stage of the operation as before cardiac bypass (pre-CPB), on bypass (on-CPB) or post-bypass (post-CPB).

Data analysis

This applies to the data of **Figures 2** and **3**. Outlier removal was performed on each time series by detecting and replacing discontinuities of more than two standard deviations from the mean and lasting less than 200 ms, with a linear interpolation of the surrounding data. The best fit linear trend was then removed before spectral analyses. Multitaper spectral analysis was performed in MATLAB using Slepian tapers²⁷. Power spectral density (PSD) and coherence estimates were calculated using a half-bandwidth, denoted Δf , of $\Delta f = 0.02$ Hz and $2 \times T \times \Delta f - 1$ orthonormal tapers, where T is the trial length in seconds.

Low frequency oscillations were identified using a blind, manual peak-selection procedure. Plots of PSD were presented, in random order and without labels as to subject or experimental period, from a list of all pressure and velocity recordings. Further, PSD values were omitted to ensure that peak selection was blind to the identity of each spectrum. Spectra with peaks between 0.02 and 0.2 Hz and prominence above the spectral background were identified, and the frequency-endpoints of these peaks were selected. Vasomotor power was calculated by integrating the PSD between the selected frequency-endpoints and the vasomotor peak frequency was calculated as the midpoint of the frequency-endpoints. Recordings that did not have a definable vasomotor peak were excluded from statistical analyses and plotted as open circles.

Statistics

Statistical analyses were performed in R (version 4.5.2, 2025); results with $p < 0.05$ were considered statistically significant unless otherwise noted. Linear mixed effects models were used to test the effect of surgical period on the vasomotor integrated power, vasomotor peak frequency, and ABP integrated power (**Figures 2A,B** and **3A**). In each case,

we used R's "lme4" and "lmerTest" packages and treated patient number as a random effect variable and bypass state as a fixed effect variable, i.e. dependent variable ~ bypass state + (1|patient number). The no bypass group was excluded from these analyses. We report p values, coefficient estimates and 95 % confidence intervals for the pre-CPB and post-CPB fixed effect coefficients, with on-CPB defined as the reference group; here we define significance if $p < 0.025$, where we divide the significance level by two to account for multiple comparisons. [A second model was used to test for differences between pre-and post-CPB groups, in which pre-CPB was defined as the reference.](#) Spearman's rank correlation coefficients (r_s) were calculated for both vasomotor power versus temperature and vasomotor frequency versus temperature joint distributions, after Henze-Zirkler tests²⁸ rejected the null hypothesis that the distributions were multivariate normal ($p < 0.001$ in both cases). For these datasets, 50th percentile estimates were found using quantile regression with second order Epanechnikov kernels and 1.25°Celsius x-bandwidths²⁹. Estimates were fit with splines using MATLAB's "spap2" function before plotting. For each trial with a peak in the PSD of the blood velocity, the magnitude and phase of the spectral coherence at the vasomotor peak frequency was displayed on a polar plot (**Figure 2F**). To determine which of these trials exhibited significant coherence, we calculated the significance level under the assumption of Gaussian statistics, i.e., $|C|_p = \sqrt{1 - p^{1/(K-1)}}$ for $p = 0.001$ and K tapers. This assumption was validated by calculating $|C|$ for randomized surrogate data drawn from the time series under investigation. Under the null hypothesis of zero coherence, $|C|$ exceeds $|C|_p$ in $p \times 100\%$ of trials. Permutation tests were used to quantify differences in the fraction of trials that exhibited a vasomotor peak between bypass states. Comparisons were performed pairwise, i.e. pre-CPB versus on-CPB or on-CPB versus post-CPB. For each patient, binary peak appearance values were randomly permuted between bypass states. Fractions for each bypass state were calculated after each permutation, these fractions were averaged across patients, and test statistics were calculated as the difference in fraction between bypass states. These statistics were compared to the test statistic value without any permutations to yield a p-value. Random permutations were performed 100,000 times.

179

180 **Results**

181 In this study we used transcranial Doppler ultrasound (TCD), a non-invasive technique that
182 measures the velocity of blood flow in the major intracranial arteries, during CPB surgery. Its
183 sampling frequency, i.e., greater than 100 Hz, makes it suitable for assessing dynamic blood
184 flow changes in real time¹. TCD has been increasingly used during CPB to monitor cerebral
185 blood flow dynamics in real time with the middle cerebral artery (MCA) as a common target,
186 based on accessibility and clinical relevance (**Figure 1A**). Arterial blood pressure (ABP)
187 measured at the radial artery served as a control for residual Mayer wave-like blood pressure
188 oscillations that may persist during CPB.

189 Twenty-two patients were approached between 2020 and 2023. Suitable TCD data were
190 retrieved from 16 patients (11 males and 5 females) with an average age of 66 (36-85) years.
191 Data on-CPB was retrieved from 14 patients while the other two only had traces off-CPB
192 (**Table 1**). Simultaneous ABP and TCD recordings were collected during all surgical
193 timepoints and were included in the analysis if the signals appeared to be stable and of good
194 quality, following visual inspection (**Figure 1B-E, Table S1**).

195 Each of the 16 subjects in the study displayed low frequency MCA blood velocity
196 oscillations near 0.1 Hz during all or some of the periods. Oscillations were present during
197 pre-CPB, on-CPB, and post-CPB periods and produced a characteristic peak in the blood
198 velocity PSD (**Figure 2A-C**). Across subjects, 54 % (15 of 28) of trials exhibited a low-
199 frequency peak during pre-CPB. During normal conditions, beat-to-beat cardiac variability
200 could obscure intrinsic VSMC oscillations. This phenomenon is well documented at
201 frequencies near 0.1 Hz³⁰ and is evidenced here by the broad-band nature of the spectral
202 peaks at heartbeat frequencies (**Figure 2A,C**). Indeed, when the heart was isolated from the
203 circulation, the occurrence of vasomotor-frequency peaks in the PSD increased to 86 %
204 (104 of 121 trials; $p = 0.01$, permutation test) (**Figure 3A,B**). After patients were brought off
205 CPB, the peak occurrence decreased to 58 %, although this decrease was not statistically
206 significant (19 of 33 trials; $p = 0.25$, permutation test).

The observed oscillations were statistically unchanged from pre-CPB to on-CPB in terms of both vasomotor power (**Figure 3A**; $p = 0.23$; fixed-effect coefficient $0.11 [-0.06, 0.28]$, estimate and 95 % CI) and peak frequency (**Figure 3B**; $p = 0.07$; fixed-effect coefficient $9.6 \times 10^{-3} [-5.5 \times 10^{-4}, 2.0 \times 10^{-2}]$, estimate and 95 % CI). Vasomotor power and peak frequency differed significantly between on-CPB and post-CPB periods ($p = 3 \times 10^{-6}$, fixed-effect coefficient $0.37 [0.22, 0.51]$ and $p = 0.02$, fixed-effect coefficient $1.1 \times 10^{-2} [1.5 \times 10^{-3}, 2.1 \times 10^{-2}]$ for power and frequency respectively). The difference in median values in each case were small ($0.19 \text{ cm}^2/\text{s}^2$ and 0.01 Hz respectively).

As expected, cardiopulmonary bypass reduced low frequency ABP power compared to before-CPB and after-CPB periods (**Figure S1A**; $p = 2 \times 10^{-5}$, fixed-effect coefficient $0.7 [0.4, 1.0]$ and $p = 2 \times 10^{-4}$ fixed-effect coefficient $0.5 [0.3, 0.8]$ respectively). The PSD for ABP were generally featureless in the $\sim 0.1 \text{ Hz}$ range (**Figure 2A-C**), and definable peaks were present in only 23 of 202 trials across subjects and surgical periods. However, it is still possible that the observed $\sim 0.1 \text{ Hz}$ oscillations in velocity were influenced by ABP activity in the same frequency band. For this reason, we calculated the coherence between the two signals as a measure of their mutual linear influence (**Figure 2A-C**). We found that 92 % (134 of 145) of trials with blood velocity vasomotor peaks had insignificant magnitude coherence between blood velocity and ABP at the vasomotor peak frequency (**Figure 3C**). We conclude that the observed persistent MCA blood velocity oscillations are not dependent on oscillations in blood pressure and are caused by intrinsic vasomotion.

As a complimentary line of evidence for the role of intrinsic vasomotion, we consider how the spectral power and frequency of blood velocity oscillations vary with temperature and blood pressure, as assessed in pioneering *ex vivo* and animal studies^{6, 31-33}. These relationships also have standalone value, as knowledge of the physiological variance of human cerebral vasomotion may aid future efforts to identify and manipulate vaso-oscillations in humans, as has been proposed to potentially increase clearance in cerebral amyloid angiopathy (CAA) patients¹⁵. We found that vasomotor power varied with temperature measured at the nasopharynx, but that the two were not significantly correlated over a range of hypothermic to normal body temperatures (**Figure 3A**; Spearman's rank correlation

$r_s = 0.08$, $p = 0.34$). Peak frequency was significantly correlated with temperature over the same range (**Figure 3B**; $r_s = 0.51$, $p = 6 \times 10^{-10}$), consistent with animal studies which show that vasomotor frequencies increase with temperature^{31, 32}. Vasomotor power and peak frequency were not significantly correlated with average ABP, although systemic ABP measured here may not reflect the relevant cerebral transmural pressures (**Figure S1B,C**; $r_s = 0.02$ and $p = 0.84$ for both metrics). Across subjects and trials, the peak vasomotor frequency was 0.07 ± 0.02 Hz (mean $\pm$ SD) which is lower than the peak frequency of 0.09 ± 0.04 Hz (mean $\pm$ SD) measured via VSMC Ca^{2+} in mice¹⁹. This difference suggests that intrinsic arteriole dynamics may contribute to species-wise hemodynamic response differences³⁴.

Discussion

Oscillations near ~ 0.1 Hz are a common feature of TCD recordings at rest^{5, 35}. Interventions such as periodically inflated thigh-cuffs, lower-body negative pressure oscillations, and periodic sit-stand maneuvers have been used to enhance systemic blood pressure oscillations near 0.1 Hz and characterize dynamic cerebral autoregulation (reviewed by Claassen et al.¹²). Here we employed the opposite intervention, i.e., elimination of ~ 0.1 Hz blood pressure oscillations that are subscribed to cardiac Mayer waves, to assess the nature of cerebral artery dynamics in the absence of systemic input. We eliminated coherent blood velocity-ABP oscillations in all but a small subset of trials (**Figure 3C**). When ABP oscillations were eliminated, velocity oscillations remained present in TCD recordings from the MCA (**Figures 1A,C and 2B**), indicating that ~ 0.1 Hz cerebral vasomotion is not driven exclusively by Mayer waves. In total, our findings indicate that cerebral arteries undergo intrinsic vasomotion.

There were a relatively small number of trials with significant coherence between blood velocity and ABP, i.e., 11 of 145, that exhibited a consistent temporal relationship between blood velocity and ABP. In all of these, MCA blood velocity preceded ABP, with an ensemble average of 1.1 ± 0.8 s (mean $\pm$ SD, **Figure 3B**). This temporal relationship is consistent with

cerebral autoregulation as characterized previously^{5, 36, 37}. These trials occurred primarily during pre-CPB, i.e., 8 of 15 pre-CPB trials with vasomotor peaks had significant magnitude coherence, indicating that this autoregulatory process remained intact, at least during the initial surgery portion.

Limitations of this study are twofold. *First*, measurements were performed on subjects during anaesthesia and in some cases during periods of hypothermia. While Mayer waves have been classified during various non-surgical conditions⁴, further work is needed to delineate the intrinsic contribution of vasomotion to blood flow dynamics during awake conditions. To this end, recent work identified significant correlation between ~ 0.1 Hz oscillations in peripheral organs, including the heart, and a spatially uniform component of the blood oxygen level-dependent (BOLD) signal^{13, 38}. Our data does not dispute this claim, which is based on a correlative connection. Brain vasomotor oscillations can be independent of systemic oscillations in the body, yet the two can phase-lock if there is a sufficiently strong interaction³⁹. *Second*, the precise vascular origin of ABP-independent MCA blood velocity oscillations in our study is unknown. The MCA diameter is generally accepted to be constant within normal blood gas and blood pressure ranges, supporting our assumption that TCD oscillations reflect pial and parenchymal vasomotion⁴⁰. In rodents, arteriole vasomotion is synchronized across large areas of the brain¹⁹; the consequent impedance modulation to incoming flow from the MCA would presumably result in oscillations in MCA flow velocity. However, the degree of vasomotion synchronization in humans is unknown. While 0.1 Hz oscillations in the input impedance of the brain arteriole vasculature is the most parsimonious explanation for concurrent oscillation in the velocity of blood flow in the MCA, the possibility that TCD oscillations are the consequence of local MCA vasomotion cannot be ruled out⁴¹.

Our study demonstrates vasomotion in humans when blood pressure is tightly controlled. The confounding impact of slow blood pressure oscillations - Mayer waves - on blood flow was thus eliminated. This provides unprecedented confirmation for the presence of intrinsic, rhythmic cerebrovascular activity, which is likely to contribute to perivascular clearance of amyloid and other deleterious peptides^{14, 15}. This setup can be a potential

platform for further observations and subsequent development of a non-invasive TCD based imaging tool that could lead to early detection of failure of perivascular clearance before the onset of dementia.

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Author contributions

AA and ROC conceived the study and obtained Integrated Research Application System approval for the use of human participants; AA performed surgery; AAB acquired physiological data; JD, SI, DK and DMS analyzed the data; AA, JD, DK and ROC wrote the manuscript; and DK and ROC obtained funding.

Statements and Declarations

Consent to participate

Written informed consent for inclusion in this research was obtained from the patients prior to surgery.

Competing interests

The authors declare no competing interests.

Data and code availability

Raw data in NWB format will be deposited on DANDI upon acceptance. Code used for analysis is publicly available at <https://github.com/JDuckworth1/HumanCBF>.

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419

Figure Captions

Figure 1: Full time traces of a patient undergoing CPB.

A, Sketch of transtemporal positioning of a transcranial Doppler (TCD) probe to detect flow from the middle cerebral artery (MCA).

B, MCA Blood Velocity trace in cm/s (Black), radial artery blood pressure trace in mmHg (Blue).

C-E, Three segments of “good data” for analysis are marked and expanded: before (**C**), during (**D**), and after (**E**) cardiopulmonary bypass (CPB), with two-second moving mean traces overlaid (Red).

Figure 2: Spectral and cross-spectral analysis of MCA blood velocity and ABP signals at different stages of CPB.

A-C, Example 5-minute recordings and PSD from a single patient pre-CPB (**A**), on-CPB (**B**), and post-CPB (**C**), with two-second moving mean traces overlaid in red. Magnitude and phase of the coherence between MCA blood velocity and ABP are plotted below the corresponding PSD. Dashed lines indicate the $p = 0.001$ significance threshold for the magnitude coherence. Insets: MCA blood velocity PSD, with expanded frequency range showing heart-beat rhythms.

Figure 3: Assessment of MCA blood velocity oscillation power, frequency, and coherence with ABP.

A, MCA blood velocity integrated spectral power, by surgical period and versus temperature. Median, 25th, and 75th percentiles are overlaid on violin plots and significant differences are indicated by *** $p < 0.001$. Trials with no vasomotor peak are plotted as open circles. The kernel-based 50th percentile estimate is overlaid in black. Horizontal gray lines indicate the preCPB median value, for comparison.

B, Vasomotor peak frequency by surgical period and versus temperature. Significant differences are indicated by * $p < 0.025$.

446 **C**, Coherence between MCA blood velocity and concurrently measured ABP. Two trials with
447 $|C| = 0.44$ and $|C| = 0.14$ and respective null hypothesis thresholds 0.79 and 0.76 were not
448 plotted. Bandpass filtered signals (0.02 – 0.18 Hz) are shown for three example trials.
449

450 **Table 1: Participant demographics and procedures**

Gender	Age (years)	Procedure	On-CPB
M	78	Deep hypothermia aortic arch surgery	Yes
F	55	Deep hypothermia aortic arch surgery	Yes
F	80	Deep hypothermia aortic arch surgery	Yes
M	36	Coronary artery bypass surgery	Yes
F	68	Trans-aortic valve implantation and coronary artery bypass surgery	Off
F	56	Coronary artery bypass surgery	Yes
M	66	Coronary artery bypass surgery	Yes
F	77	Aortic valve replacement	Yes
M	73	Coronary artery bypass surgery	Yes
M	74	Coronary artery bypass surgery	Yes
M	52	Coronary artery bypass surgery	Yes
M	69	Coronary artery bypass surgery	Yes
M	85	Lung resection	Off
M	51	Aortic valve replacement	Yes
M	70	Coronary artery bypass surgery	Yes
M	72	Coronary artery bypass surgery and mitral valve repair	Yes

451

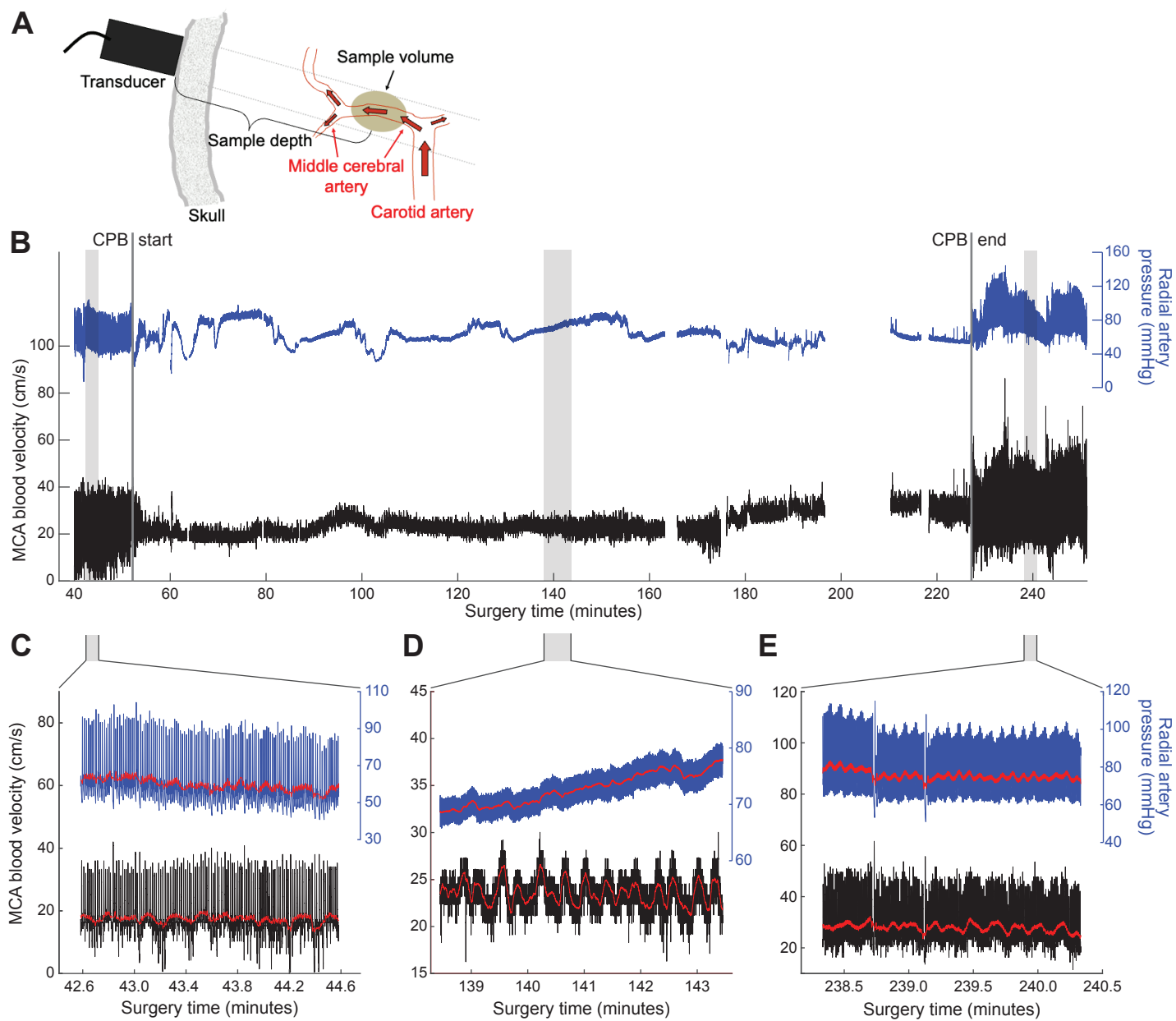


Figure 1. Alzetani, Duckworth, Birch, Iyengar, Simpson, Kleinfeld and Carare

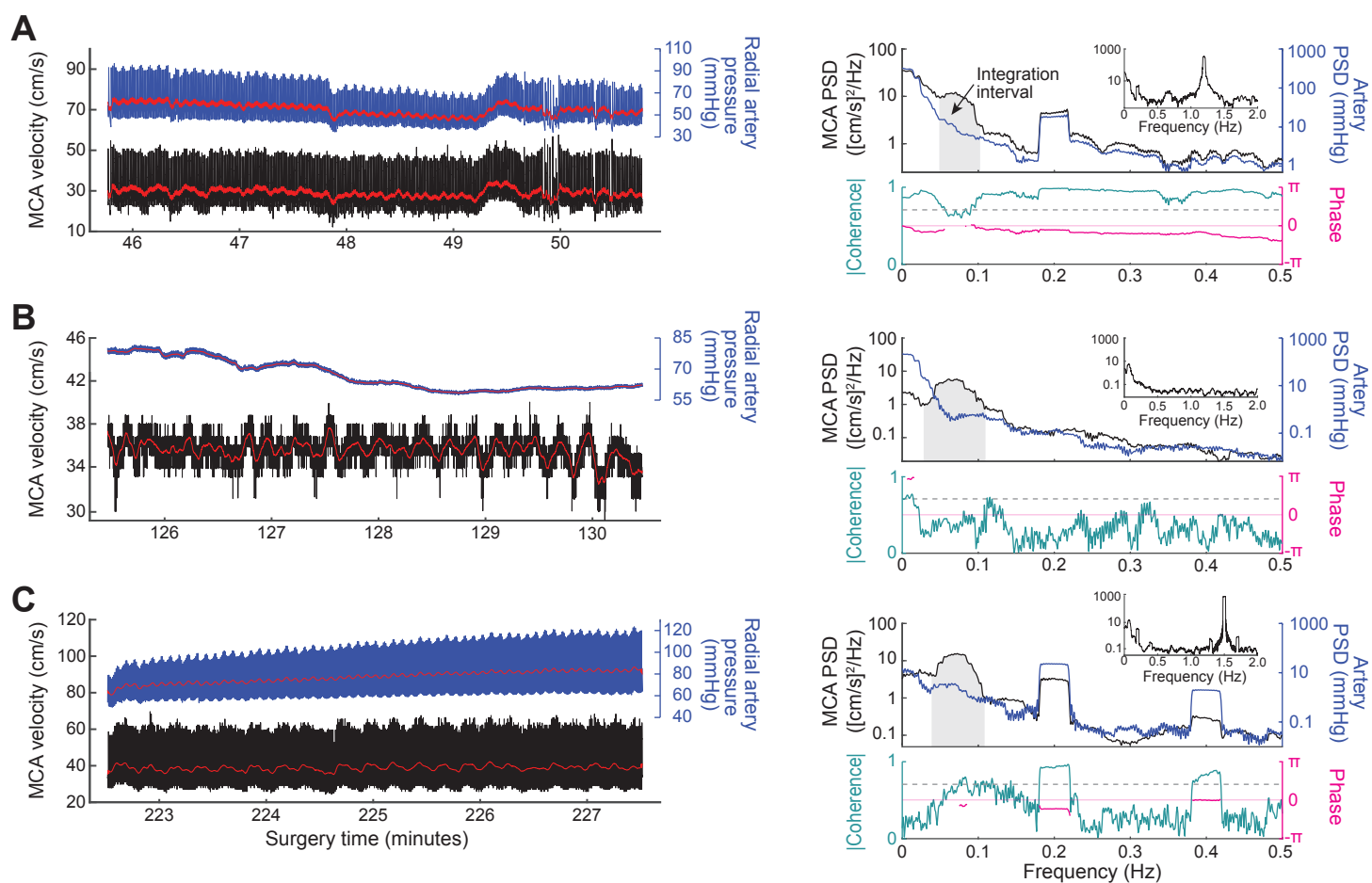


Figure 2. Alzetani, Duckworth, Birch, Iyengar, Simpson, Kleinfeld and Carare

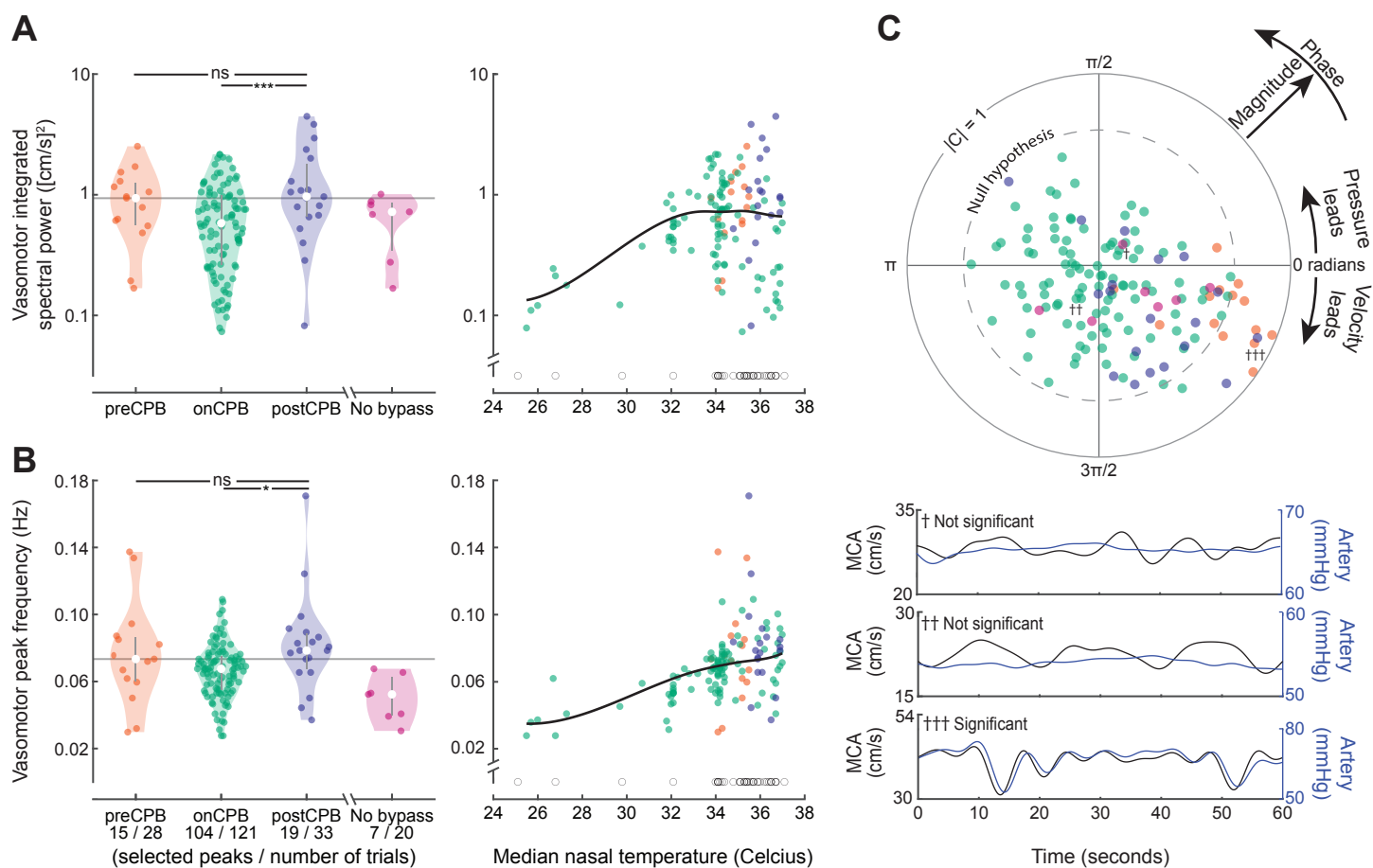


Figure 3. Alzetani, Duckworth, Birch, Iyengar, Simpson, Kleinfeld and Carare

Table S1. Number of data segments by bypass period

	Number of Data Segments			
Patient Number	Pre Bypass	On Bypass	Post Bypass	No Bypass
1	2	17	9	0
2	0	6	0	0
3	10	6	9	0
4	1	10	5	0
5	3	0	0	0
6	1	12	1	0
7	0	3	3	0
8	0	7	0	0
9	3	13	0	0
10	2	12	0	0
11	1	2	2	0
12	4	4	1	0
13	0	0	0	6
14	0	0	0	14
15	0	6	0	0
16	1	23	3	0

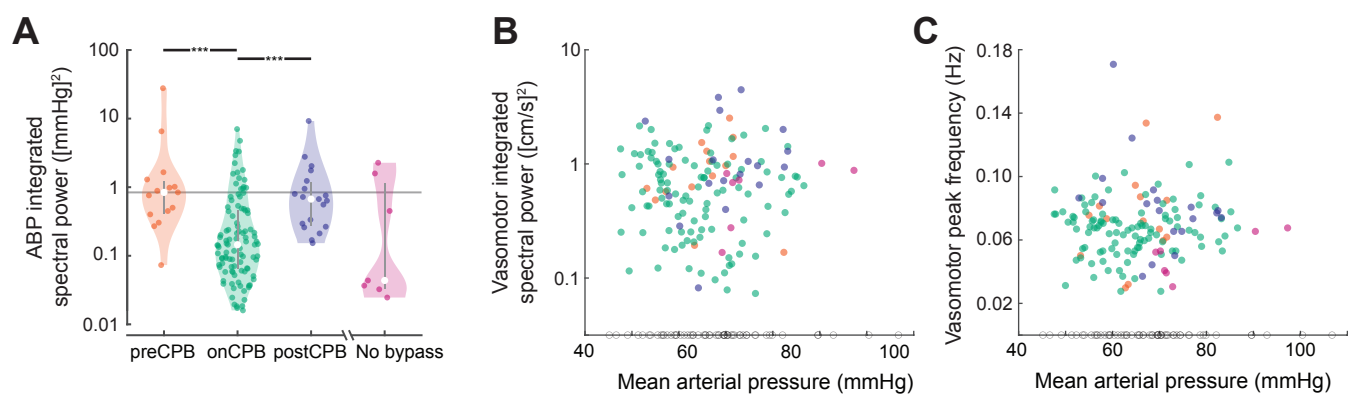


Figure S1. Alzetani, Duckworth, Birch, Iyengar, Simpson, Kleinfeld and Carare

Figure S1: Trial-averaged ABP and ABP oscillation power

A, Arterial blood pressure power versus bypass state, integrated using the same ~0.1 Hz frequency bounds that were used for the MCA blood velocity spectra. Significant differences are indicated by *** ($p < 0.001$).

B, Integrated vasomotor power versus trial-average ABP. Open circles denote trials with no selected vasomotor peak.

C, Vasomotor peak frequency versus trial-averaged ABP.