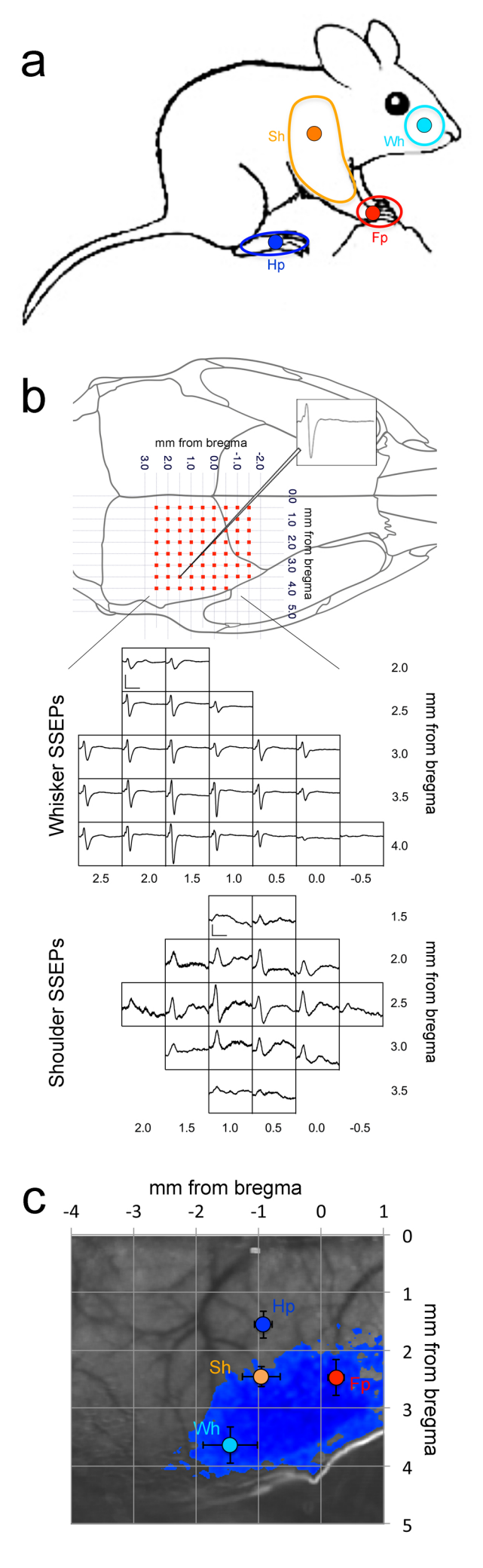
a
Sh
Wh
Fp
Hp
b
mm from bregma
3.0 2.0 1.0 0.0 -1.0 -2.0
0.0 1.0 2.0 3.0 4.0 5.0
mm from bregma
Whisker SSEPs
2.0 2.5 3.0 3.5 4.0
mm from bregma
2.5 2.0 1.5 1.0 0.5 0.0 -0.5
Shoulder SSEPs
1.5 2.0 2.5 3.0 3.5
mm from bregma
2.0 1.5 1.0 0.5 0.0 -0.5
c
mm from bregma
-4 -3 -2 -1 0 1
0 1 2 3 4 5
mm from bregma
Hp
Sh
Fp
Wh

**Figure S1. Tactile stimulation points in anesthetized mice (related to Figures 1-5, and 7).**

a. Diagram showing the regions of tactile stimulation (outlines) delivered manually using a cotton tip applicator at a rate of 3-5Hz. Care was taken not to induce movement in the stimulated part or the rest of the body. Circles show the focal electrical stimulation sites used to map the S1.

b. SSEPs were recorded over a grid with 0.5 mm site separation (red dots) referenced to bregma during electrical stimulation of the 4 stimulus locations. For all stimulated areas, reproducible evoked potentials were obtained. Representative maps from one mouse each during electrical whisker and shoulder stimulation are also shown. These maps were use to determine the site of peak SSEPs in each animal. Whisker scale bars show 1 mV and 20 msec, shoulder scale bars show 0.2 mV and 20 msec.

c. A speckle contrast image of the dorsal surface of the right hemisphere showing the coordinates (± standard deviations in mediolateral and anteroposterior axes) for peak forepaw (Fp, N=7), shoulder (Sh, N=5), whisker (Wh, N=3) and hindpaw (Hp, N=6) stimulation-induced SSEPs referenced to bregma (stimulation locations shown in a, circles). A representative laser speckle perfusion defect (blue background) with less than 30% residual CBF after dMCAO is also shown on the background. These coordinates were used to determine the spatial relationship of stimulation-induced PID origins to respective S1 cortices as well as to quantify the relative CBF in each S1 during stimulation. Specific procedures are detailed in the Methods.

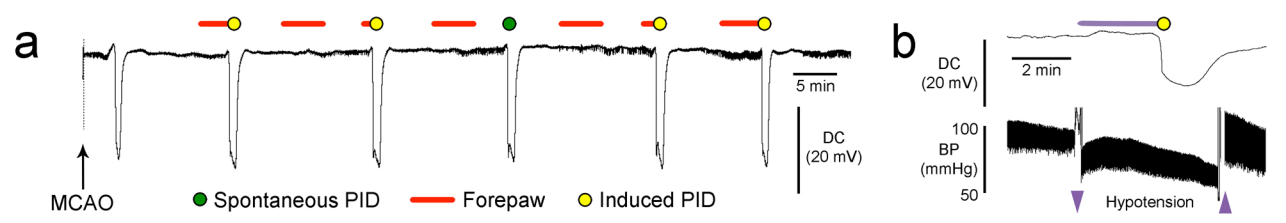
a
5 min
DC
(20 mV)
MCAO
Spontaneous PID
Forepaw
Induced PID
b
DC
(20 mV)
2 min
100
BP
(mmHg)
50
Hypotension

**Figure S2. Electrophysiological confirmation of PID occurrence (related to Figures 1-6).**

a. Electrophysiological tracing of extracellular DC potential showing PIDs (negative DC deflections marked by yellow circle) triggered during forepaw stimulation (red lines) in a representative mouse. Four out of 7 attempts triggered a PID in this experiment (57%). This rate was similar to experiments where PIDs were monitored using laser speckle flowmetry (Fig. 1). One spontaneous PID also occurred in between stimulations (green circle). A total of 10 PIDs were detected electrophysiologically during 19 forepaw stimulations in 3 mice (53%), adhering to the protocol described in Figure 1. Vertical dotted line shows the onset of MCAO. Recordings were obtained from non-ischemic cortex ipsilateral to MCAO. The first PID after MCAO is triggered by the anoxic depolarization in the core spreading onto non-ischemic cortex, and thus not counted as a spontaneous or induced PID.

b. Extracellular DC potential (DC, upper tracing) and blood pressure (BP, lower tracing) showing a PID (yellow circle) during a hypotensive transient (purple line) induced by controlled blood withdrawal (arrowhead pointing down). After confirmation of PID, blood was reinfused (arrowhead pointing up) and BP restored.

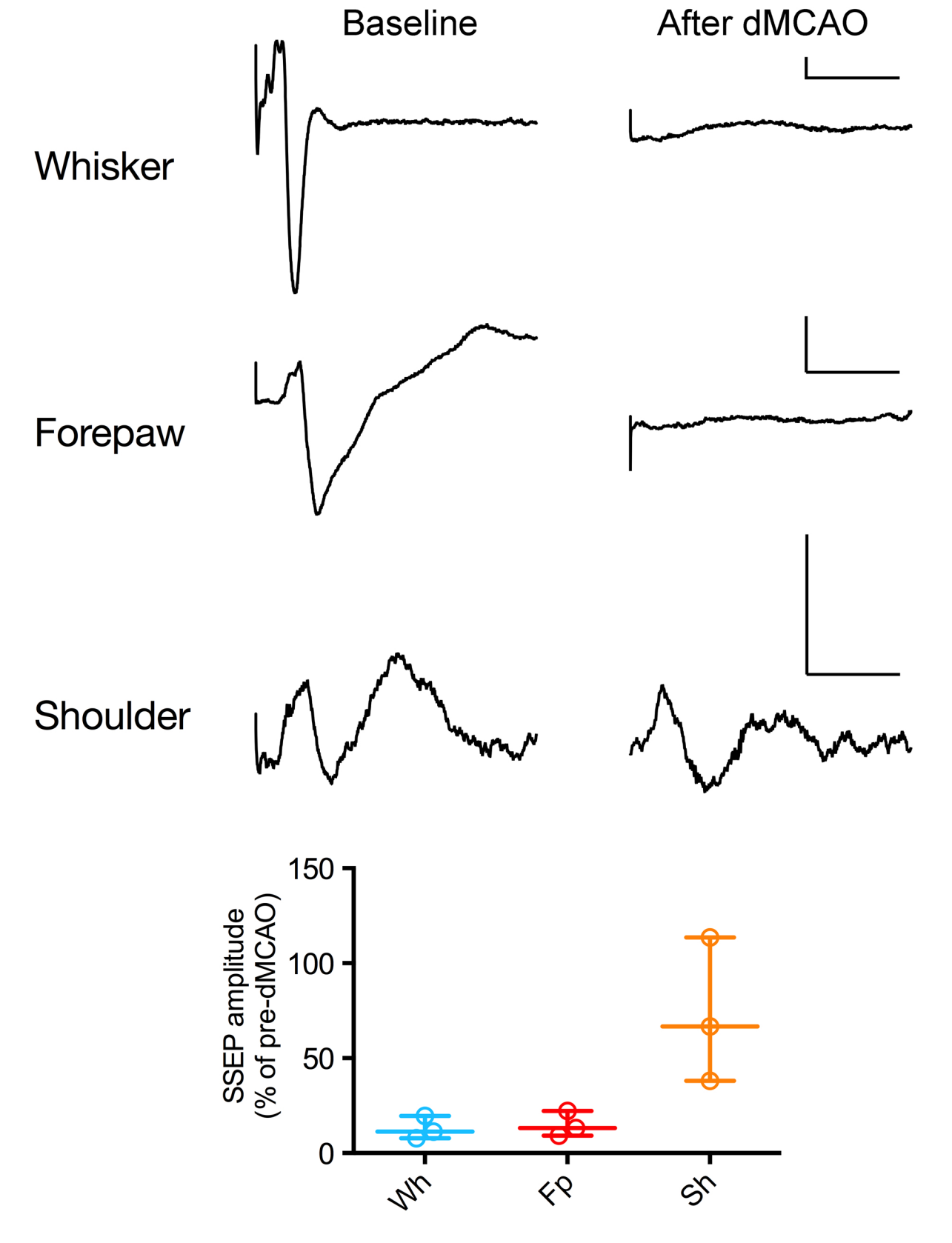
Baseline
After dMCAO
Whisker
Forepaw
Shoulder
SSEP amplitude
(% of pre-dMCAO)
150
100
50
0
Wh
Fp
Sh

**Figure S3. Shoulder but not whisker or forepaw SSEPS are preserved after dMCAO (related to Figures 2 and 3).**

Representative SSEPs in response to electrical whisker, forepaw or shoulder stimulation before and after dMCAO, and averaged data (n=3 each) showing that dMCAO abolishes whisker and forepaw SSEPs, but not shoulder SSEPs. Scale bars show 0.1 mV and 20 msec. Spontaneously breathing mice were anesthetized with1.5% isoflurane in 70% $N_2$ and 30% $O_2$, and lidocaine topically applied onto the skull. A reference electrode was placed on the skull on the contralesional hemisphere caudally and a ground electrode placed on the tail. The recording electrode was placed over the ipsilesional S1. Whisker, forepaw and shoulder were electrically stimulated and SSEPs recorded at the predetermined coordinates before and 10-60 minutes after dMCAO.

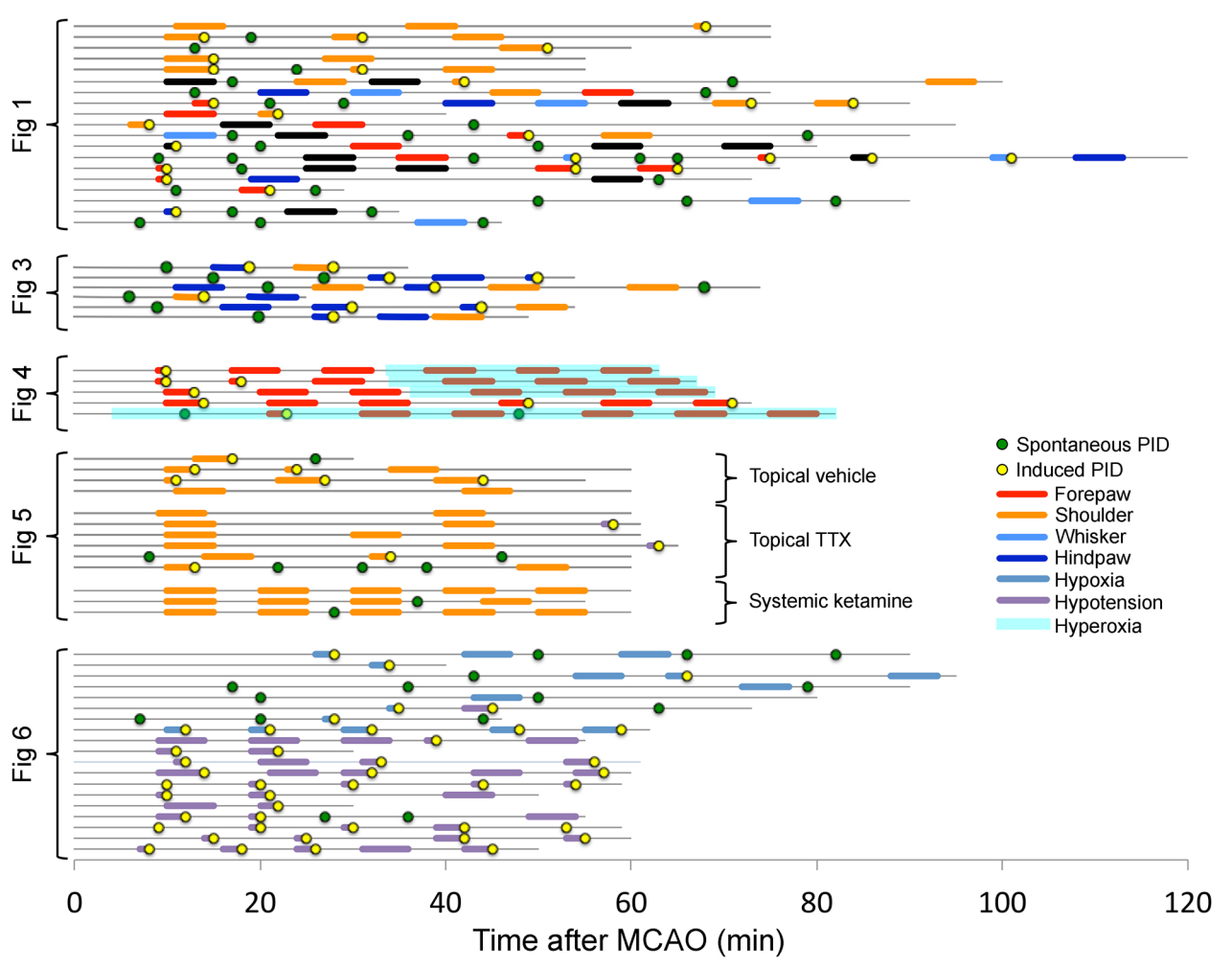
Fig 1
Fig 3
Fig 4
Fig 5
Fig 6
Topical vehicle
Topical TTX
Systemic ketamine
Spontaneous PID
Induced PID
Forepaw
Shoulder
Whisker
Hindpaw
Hypoxia
Hypotension
Hyperoxia
0
20
40
60
80
100
120
Time after MCAO (min)

**Figure S4. Complete experimental timelines (related to Experimental Procedures).**

Entire experimental timelines from Figures 1, 3, 4, 5 and 6 showing spontaneous as well as induced PIDs (green and yellow circles, respectively), and stimulation of different body parts on the same timeline (see Fig. 1 legend for details of representation). Up to 4 body parts were stimulated sequentially in any given animal, but never less than 5 minutes apart. In Figure 1, each body part was shown separately on a different panel for clarity. In only one experiment, hypoxia and hypotension were studied in the same animal.

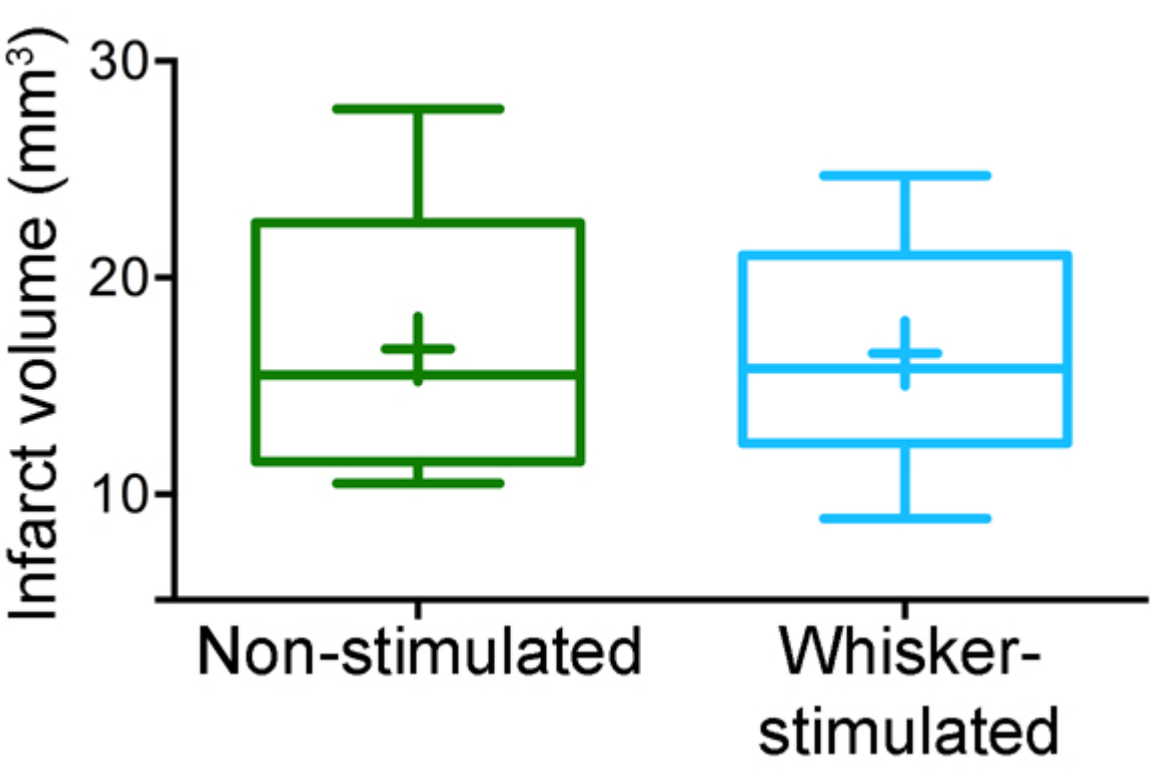
Infarct volume (mm3)
30
20
10
Non-stimulated
Whisker-stimulated

**Figure S5. Whisker stimulation does not alter infarct volume after permanent dMCAO in mice (related to Figure 7).**

Permanent dMCAO was induced as described in the Methods. Infarct volumes were measured 24 hours after dMCAO using TTC staining. Whisker stimulation was delivered according to previously reported protocols as described in the Methods.

**Movie S1. Spontaneous PID (related to Figures 1 and 2).**

Laser speckle contrast imaging shows the perfusion defect in the left hemisphere after dMCAO (blue pixels). Movie shows a spontaneous PID wave emerging into the imaging field from its anterior aspect as a spreading hypoperfusion wave characteristic of PIDs.

**Movie S2. Spontaneous PID (related to Figures 1 and 2).**

Laser speckle contrast imaging shows the perfusion defect in the left hemisphere after dMCAO (blue pixels). Movie shows a spontaneous PID wave emerging into the imaging field anteriorly and after a short latency posteriorly; these mirror waves then collide posteromedially.

**Movie S3. Shoulder stimulation-induced PID originating from stimulated S1 (related to Figures 1 and 2).**

Laser speckle contrast imaging shows the perfusion defect in the left hemisphere after dMCAO (blue pixels). The origin of PID (yellow oval) is also shown. Two separate shoulder stimulations were delivered at 11 and 20 minutes as indicated by the red dot on upper left. Each is followed by a spreading hypoperfusion wave characteristic of PID.

**Movie S4. Hindpaw stimulation-induced PID originating from stimulated S1 in a hypotensive animal (related to Figure 3).**

Laser speckle contrast imaging shows the perfusion defect in the left hemisphere after dMCAO (blue pixels). The origin of PID (yellow oval) is also shown. Hindpaw stimulation was delivered at 44 minutes as indicated by the red dot on upper left, and triggered a spreading hypoperfusion wave characteristic of PID that originated from hindpaw S1. Hindpaw stimulation never triggered a PID from hindpaw S1 in normotensive mice.

**Table S1. Systemic physiological parameters (related to Experimental Procedures).**

| Cohort | BW | BP | pH | pCO2 | pO2 |
|---|---|---|---|---|---|
| Control | 24.9±0.4 | 76±2 | 7.36±0.01 | 28.5±0.9 | 150±6 |
| Somatosensory stimulation (normotensive cohort) | 25.0±0.3 | 75±2 | 7.36±0.01 | 28.9±0.5 | 156±4 |
| Somatosensory stimulation (hypotensive cohort) | 24.8±1.5 | 59±5 | 7.38±0.05 | 37±5 | 115±13 |
| Transient hypoxia and hypotension (baseline values) | 26.2±0.3 | 81±3 | 7.36±0.01 | 29.6±0.6 | 146±5 |
| TTX | 25.0±0.4 | 81±2 | 7.38±0.02 | 30.6±1.4 | 148±8 |
| Ketamine | 25.0±0.2 | 84±5 | 7.35±0.02 | 30.5±4.0 | 157±8 |
| Normobaric hyperoxia | 23.9±0.5 | 76±3 | 7.36±0.01 | 29.2±1.6 | 468±39 |

BW, body weight in grams; BP, blood pressure in mmHg. Blood gas values are in mmHg. One-way ANOVA followed by Tukey's multiple comparisons test.