

A software tool for the simultaneous analysis of cortical slow waves in electrophysiological and laser-speckle hemodynamic recordings of the murine lesion model

Author: Abel Vázquez Santisteban, vazabel02@alumnes.ub.edu
Facultat de Física, Universitat de Barcelona, Barcelona, Spain.

UB Advisor: Roser Sala-Llonch
Universitat de Barcelona, Barcelona, Spain.

External Advisor: Maria V. Sánchez-Vives
IDIBAPS, Institut d'Investigacions Biomèdiques August Pi i Sunyer, Barcelona, Spain.

Abstract: Cortical slow waves (< 2 Hz) can be detected from the firing of cortical neurons, and they also produce a slower change in cerebral blood flow. These two readings are usually done apart, and almost never on the same animal. We present a software tool that handles both at the same time, for the murine focal-lesion model. The tool reads a 32-channel micro-electrode-array (MEA) recording of local field potentials (LFP, Open Ephys, 5 kHz) and wide-field laser speckle contrast images of regional cerebral blood flow, pre-processed by the ICFO Medical Optics group. We describe its four signal-processing blocks: (i) zero-phase Butterworth band-pass filtering of the LFP; (ii) Up/Down state detection on a score that fuses a log-MUA estimate and a high-band variance estimate by principal-component analysis, with an Otsu threshold and a minimum-duration state cleaner; (iii) a wave-propagation block that groups channels by cross-correlation coherence with hierarchical clustering and fits a two-dimensional plane wave over the MEA geometry to get the propagation speed and direction; and (iv) a viewer that re-aligns the channels by the lags from the propagation block. The blocks are run on real LFP recordings of an anesthetized mouse, and all figures are produced by the same code used by the interface. The tool also implements a fifth block that links the electrical and the hemodynamic signals to map neurovascular coupling; it is implemented but not yet tested on real data, since we do not have a recording with both signals from the same animal yet. **Keywords:** Slow waves, Up/Down states, neurovascular coupling, laser speckle, wave propagation, signal processing.

SDGs: 3 (Good health and well-being), 4 (Quality education), 9 (Industry, innovation and infrastructure)

I. INTRODUCTION

The slow oscillation (SO, below 2 Hz) is the spontaneous activity of the cerebral cortex under deep anesthesia and during slow-wave sleep [1, 2]. The cortex alternates between *Up* states, where many neurons fire together, and *Down* states where almost all of them go silent. The same Up/Down regime also emerges around focal cortical lesions: the perilesional cortex falls into a slow-wave state similar to sleep, and this pathological pattern can propagate from the lesion to other regions, dragging them out of their normal activity [2]. Understanding how the SO spreads from a focal brain lesion across the cortex motivates this work, because of its translational relevance to brain injury, stroke and epilepsy.

The SO is not only an electrical phenomenon. The intense firing of an Up state raises the local demand for oxygen and glucose, and the microvasculature answers with a delayed change in blood flow and in the oxy- and deoxy-haemoglobin concentration (HbO and HbR) [3, 4]. An Up state thus shows up twice: as a fast electrical event in the multi-unit activity (MUA) band of the LFP, and as a slower vascular response. How exactly the elec-

trical activity couples to these changes in blood flow and oxygenation, and what its function is, remains an open question in systems neuroscience [6].

These two signals are usually recorded apart. Electrophysiology has microsecond time resolution but no direct access to blood flow information. Optical imaging (laser speckle contrast imaging, LSCI; multispectral HbO/HbR) covers millimetres of cortex, but it is slow – on the order of seconds to tens of seconds – and is filtered by the hemodynamic response [4]. Studies of slow waves in the rodent lesion model normally use one of the two, rarely both at the same time on the same animal. The focal-lesion model is a good case to combine them, because an electrically silent region does not need to be silent in blood flow.

In this work we present a software tool that puts the MEA LFP and the ICFO-preprocessed laser-speckle data on the same time axis. The raw speckle images and their reduction to cortical parcels are done at ICFO; here we deal with the signal-processing side: filtering, Up/Down detection, wave propagation, channel re-alignment, and the neurovascular-coupling block that links the two modalities.

II. METHODS

The tool reads one Open Ephys recording ($C = 32$ channels, $f_s = 5$ kHz) and the LSCI parcels supplied by ICFO, and refers them to a common time grid through a linear fit of the shared TTL pulses. The electrophysiology comes from a 32-channel planar surface array (*in-vivo32LH*) on the cortex of one hemisphere, a 4×8 grid with $550 \mu\text{m}$ centre-to-centre spacing spanning about 1.65×3.85 mm. Bad channels and parcels are propagated as NaN.

A. Band-pass filtering of the LFP

The user can inspect any band $[f_{\text{lo}}, f_{\text{hi}}]$. The tool builds each band-pass from the Butterworth low-pass prototype of order n , whose squared magnitude response is

$$|H_{\text{LP}}(\omega)|^2 = \frac{1}{1 + (\omega/\omega_c)^{2n}}, \quad (1)$$

through the standard low-pass-to-band-pass transformation; we use $n = 4$. A Butterworth filter has non-linear phase, which would bias any later timing estimate, so the tool filters forward and backward (`filtfilt`); the two passes cancel the phase, giving a zero-phase response,

$$y_c(t) = \mathcal{F}^{-1}\{|H(\omega)|^2 X_c(\omega)\}, \quad (2)$$

at the cost of squaring the magnitude (effective order $2n$). The analysis blocks each use a fixed band: 0.5–2 Hz for propagation, and 15–100 Hz and 200–1500 Hz for the two detection markers (Fig. 1(a)).

B. Up/Down state detection

Detection is not based on a single filtered band, but on a *score* that combines two markers, following Ruiz-Mejias et al. [5].

a. Log-MUA. The raw signal is cut into short windows of $f_s/200 = 25$ samples (5 ms). Each window is detrended, its power spectrum $P(f) = |\mathcal{F}\{x\}|^2$ is computed, only the multi-unit band $200 \leq f \leq 1500$ Hz is kept, and each frequency bin is normalized by its median over time (a per-frequency baseline). The log-MUA is the log of the band-averaged normalized power,

$$m_c(t) = \log \left\langle P_c(f, t) / \tilde{P}_c(f) \right\rangle_{200 \leq f \leq 1500 \text{ Hz}}, \quad (3)$$

smoothed over 40 ms. It rises in Up states and falls in Down states, and its output rate is 200 Hz.

b. High-band variance. As a second, independent marker we take the running standard deviation of the 15–100 Hz band over a sliding window $W(t)$ of 0.2 s,

$$k_c(t) = \left[\text{Var}_{\tau \in W(t)} \{(\mathcal{B}_{[15,100]}x_c)(\tau)\} \right]^{1/2}, \quad (4)$$

smoothed over 80 ms. It captures the broadband power increase of Up states even when the MUA band is noisy.

c. Fusion by principal-component analysis (PCA). The two markers are z-scored and combined into a single score by taking the first principal component,

$$s_c(t) = \text{PC}_1[\tilde{m}_c(t), \tilde{k}_c(t)], \quad (5)$$

with the sign fixed so that Up states are positive. PC_1 captures the variance shared by the two markers and reduces the contribution of the noise specific to each. On our recordings PC_1 accounts for a median of 0.66 of the joint variance (range 0.51–0.78 over channels), so a single score summarizes the two markers well.

d. Threshold. If the histogram of s_c is clearly bimodal, the threshold θ is the valley between the two most prominent peaks. Otherwise the tool uses Otsu's threshold [8], the value that maximizes the between-class variance,

$$\theta = \arg \max_{\theta} p_0(\theta)p_1(\theta)[\mu_1(\theta) - \mu_0(\theta)]^2, \quad (6)$$

where p_0, p_1 and μ_0, μ_1 are the weights and means of the two classes defined by θ . The typical score histogram is not two clean peaks, but a dominant Down bulk with a long Up tail. In that case Otsu is not the theoretically optimal choice, but it still gives a reasonable cut, and unlike the median it does not force a fixed 50% Up-fraction. The tool shows the histogram and the chosen θ so the user can judge the split.

e. State cleaning. The binary train $s_c(t) \geq \theta$ is cleaned by removing every Up or Down segment shorter than 100 ms: on each pass we flip the shortest segment below this limit and merge it with its neighbours; the procedure converges and treats both states the same way. Each transition is then refined to sub-window precision by fitting a third-degree polynomial to s_c in a ± 50 ms window and taking its crossing of θ . The detector returns the Up/Down train and the dominant slow-wave frequency $f_{\text{SW}} = 1/(\langle \tau_{\text{Up}} \rangle + \langle \tau_{\text{Down}} \rangle)$.

C. Wave propagation across the array

Slow waves travel across the cortex, so the same Up state reaches different MEA channels at slightly different times. The propagation block estimates these delays and turns them into a speed and a direction.

a. Pre-processing. Channels whose log-variance is an outlier (more than $z = 3.5$ in median-absolute-deviation units) are rejected as bad. The remaining channels are band-passed in the slow band (0.5–2 Hz by default) and a common-average reference (CAR) is subtracted: each electrode picks up a local component and a global one, and subtracting the mean across good channels removes the global part, which would otherwise push every correlation towards +1. The signals are then decimated to 200 Hz.

b. Coherence and lags. For every pair of channels the normalized cross-correlation is computed up to ± 500 ms,

$$\rho_{ij}(\tau) = \frac{\langle x_i(t) x_j(t + \tau) \rangle}{\sqrt{\langle x_i^2 \rangle \langle x_j^2 \rangle}}, \quad (7)$$

and summarized by the coherence $|\text{coh}_{ij}| = \max_{\tau} |\rho_{ij}(\tau)|$ and the lag $\ell_{ij} = \arg \max_{\tau} \rho_{ij}(\tau)$.

c. Grouping. Channels that share a wave are grouped by hierarchical (Ward) clustering [9] on the distance $d_{ij} = 1 - |\text{coh}_{ij}|$. Within each cluster the source channel is the one with the most negative mean lag (the earliest).

d. Plane-wave velocity. Each MEA channel has a known position (x_c, y_c) (in mm). Inside a cluster, the lags relative to the source are fitted to a two-dimensional plane wave by least squares,

$$\ell_c = a x_c + b y_c + c_0, \quad (8)$$

where $\mathbf{s} = (a, b)$ is the *slowness* vector (units s/mm, i.e. the inverse of a speed; not to be confused with a wave vector). The propagation speed and direction are

$$v = \frac{1}{\|\mathbf{s}\|}, \quad \phi = \text{atan2}(b, a), \quad (9)$$

and the goodness of fit R^2 is reported alongside; clusters with a speed outside the physiological range are flagged. Equation (8) is the 2D version of a constant-speed front: a 1D “lag versus depth” fit would only work for a linear probe, but our MEA is planar.

D. Channel re-alignment viewer

The lags ℓ_c returned by Eq. (7) feed a viewer that lets the user compare the *shape* of the wave across channels. Each channel is shifted by a controllable fraction $\alpha \in [0, 1]$ of its lag,

$$x_c^{\text{aligned}}(t) = x_c(t + \alpha \ell_{c_0c}), \quad (10)$$

so that $\alpha = 0$ shows the wave travelling and $\alpha = 1$ collapses the channels of a cluster onto their common reference, removing the inter-channel phase offset.

E. Neurovascular coupling (implemented, pending data)

This block is the main motivation of the tool: to relate the two signals once they are recorded together. The LFP shows the firing at the start of an Up state in milliseconds; the speckle signal shows the vascular answer a few seconds later, strongly low-pass filtered [3, 4]. After a focal lesion the gain or latency of this coupling may change, so measuring it tells us whether the slow waves

near the lesion still drive a vascular response. The MUA power is decimated to the speckle rate $f_R \simeq 50$ Hz, giving an envelope $M(t)$ matched to each parcel signal $R_i(t)$; the block then works at three levels.

a. (1) Coupling-delay map. For each parcel the block computes the normalized cross-correlation

$$c_i(\tau) = \frac{\langle M(t) R_i(t + \tau) \rangle}{\sqrt{\langle M^2 \rangle \langle R_i^2 \rangle}}, \quad (11)$$

and maps the peak lag $\tau_i^* = \arg \max_{\tau} c_i(\tau)$ (vascular delay) and the peak value $c_i(\tau_i^*)$ (coupling strength) over the cortex.

b. (2) Up-triggered average. Triggering on the Up-state onsets of Sec. IIB, the block averages each parcel over a window around onset, giving the mean hemodynamic transient evoked by an Up state.

c. (3) HRF by deconvolution. The fullest description treats the neurovascular coupling (NVC) as a linear time-invariant system, $R_i(t) = \sum_{\tau} h(\tau) M(t - \tau) + \varepsilon(t)$, and recovers the hemodynamic response function (HRF) h . Writing this as $\mathbf{r} = \mathbf{X}\mathbf{h}$ with $\mathbf{X}$ the Toeplitz matrix of $M(t)$, the least-squares solution is ill-conditioned because successive samples of $M(t)$ are correlated. The block therefore uses Tikhonov regularization,

$$\hat{\mathbf{h}} = (\mathbf{X}^T \mathbf{X} + \lambda \mathbf{I})^{-1} \mathbf{X}^T \mathbf{r}, \quad \lambda = \rho \frac{\text{tr}(\mathbf{X}^T \mathbf{X})}{L}, \quad (12)$$

with $L = f_R T_{\text{HRF}}$ taps and ρ a dimensionless penalty selected by generalized cross-validation (GCV). The recovered $\hat{\mathbf{h}}(\tau)$ is the impulse response: its peak amplitude is the coupling gain and its time-to-peak the vascular latency.

Without a simultaneous recording the block cannot be tested on real data; we validate it on synthetic ground truth in Sec. IIID.

III. RESULTS

We run the first three blocks on 60 s windows of a baseline (*prelesion*) and a post-lesion recording of the same anesthetized mouse, and validate the coupling block on synthetic data. Each caption lists the parameters needed to reproduce the panel from the interface.

A. Filtering and the slow-wave power

Figure 1(a) shows the three Butterworth bands the pipeline uses (0.5–2 Hz for propagation, 15–100 Hz and 200–1500 Hz for the two detection markers). Each band is flat over its own range and rejects the others by more than 40 dB, so the markers do not contaminate each other. Panel (b) shows why the slow band matters: the LFP power is concentrated below 4 Hz (shaded) and grows further after the lesion.

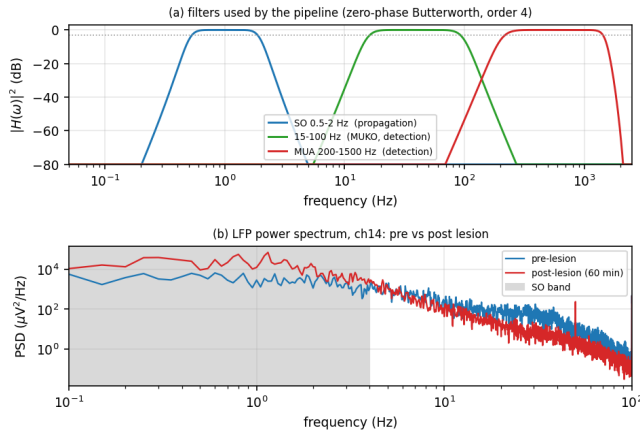


FIG. 1: (a) Frequency response $|H(\omega)|^2$ of the three Butterworth filters the pipeline uses (order 4, zero-phase), each labelled with the block that consumes it. (b) Welch power spectrum of channel 14 before and after the lesion; the slow-oscillation band is shaded. Interface: Spectral, *prelesion* vs *post 60*, ch. 14.

B. Up/Down detection

Figure 2 shows the detection score of Eq. (5), the Otsu threshold and the resulting Up/Down train for channel 14; the detected Up states coincide with the bursts of the score, with no false transitions between them. On this 60 s window the up-fraction is 0.14 and the mean Up and Down durations (0.26 and 1.56 s) give $f_{sw} = 0.55$ Hz, the slow-oscillation regime (< 1 Hz) of the anesthetized mouse [5]. The 100 ms state cleaner of Sec. II B reduces the raw threshold crossings to 64 transitions, with a median state duration of 0.30 s.

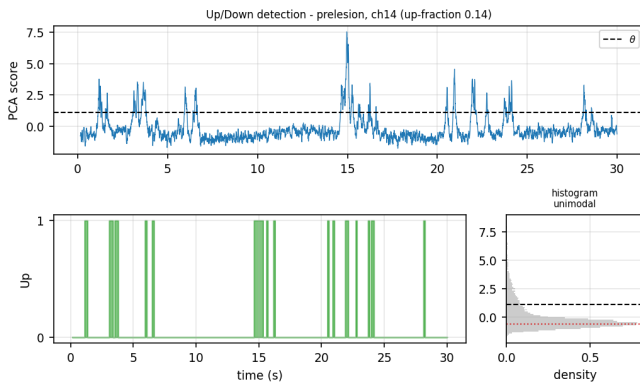


FIG. 2: Up/Down detection on real data. Top: detection score $s_c(t)$ with the Otsu threshold θ . Bottom left: detected Up states. Bottom right: histogram of the score with θ . Interface: Slow Waves, *prelesion*, ch. 14, 0–60 s.

C. Wave propagation

Figure 3 shows the coherence matrix of Eq. (7) and the plane-wave fit of one cluster; the empty rows/columns are the channels rejected as bad. The cluster shown ($N = 4$ channels) is the one the engine flags as dominant – the cleanest fit inside the physiological range – giving $v = 20.4$ mm/s with $R^2 = 1.0$, while the largest coherent block is a separate, slightly larger cluster of 5 channels at 7.8 mm/s ($R^2 = 0.97$). With $N = 4$ channels and 3 parameters in Eq. (8) the fit has a single degree of freedom, so $R^2 = 1.0$ on small clusters should be read together with the cluster size.

At the real spacing ($550 \mu\text{m}$ centre-to-centre) the clusters fall in 4–34 mm/s, in or just below the 10–100 mm/s physiological range for mouse slow waves [5, 7]. Since the positions in Eq. (8) scale as $x_c, y_c \propto \Delta$, the speed scales as $v \propto \Delta$ while the direction depends only on the ratio of the lags: the absolute speed is only as accurate as the entered spacing, but the direction is robust to it.

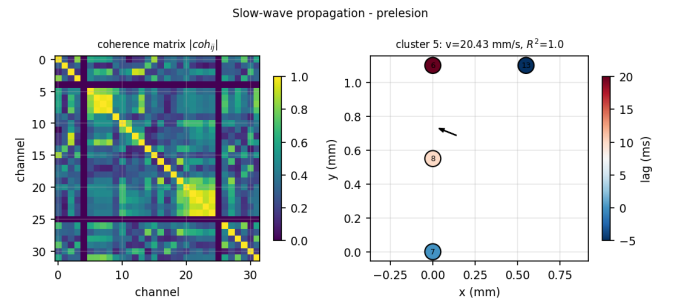


FIG. 3: Propagation block. Left: coherence matrix $|coh_{ij}|$ over the 32 channels; the empty lines are rejected channels. Right: lags of the dominant cluster on the MEA geometry, with the fitted plane-wave direction (arrow), speed and R^2 . Interface: Propagation, *prelesion*, 0–60 s, 0.5–2 Hz, CAR.

D. Neurovascular coupling: synthetic validation

Since no simultaneous recording exists yet, we validate the coupling block on synthetic ground truth and characterize it against the noise level. We build a synthetic MUA train of Poisson Up onsets, convolve it with a known canonical HRF (peak at 4 s, within the range reported for mouse regional cerebral blood flow, rCBF), add band-limited noise (0.1–1 Hz, the kind that actually corrupts the coupling), and run the same Tikhonov deconvolution the tool uses (Eq. 12). We sweep the signal-to-noise ratio (SNR) from 0.3 to 10 (8 noise realizations each) and report the L_2 error of the recovered HRF and its time-to-peak (Fig. 4).

As a reference, we measured the SNR of our own rCBF parcels by the standard Welch spectral method (slow-band 0.1–1 Hz power against the noise floor): about 0.7–2 (shaded band in Fig. 4), i.e. signal comparable to noise.

This is only a proxy of the true coupling SNR.

Fig. 4(a) shows the HRF shape error falling from about 0.5 at low SNR to 0.2 at high SNR, so the deconvolution is essentially exact when there is little noise. Inside the real range of our data the shape is noisy ($L_2 \approx 0.3$ – 0.4), but the time-to-peak – the read-out we use to compare before and after the lesion – is still recovered within about 0.5 s of the true 4 s (Fig. 4(b)), because the deconvolution averages over the hundreds of Up onsets of the synthetic train. The block is therefore ready for the first simultaneous recording.

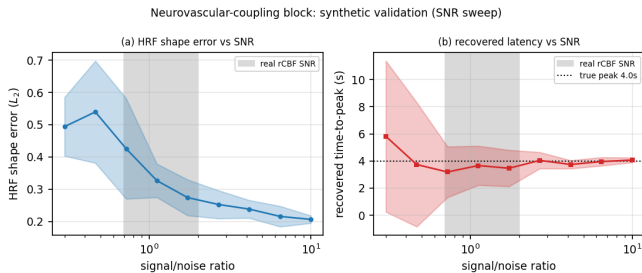


FIG. 4: Synthetic validation of the coupling block, swept over the signal-to-noise ratio (band-limited noise; mean $\pm$ SD over 8 realizations). (a) L_2 error of the recovered HRF vs. SNR. (b) Recovered time-to-peak vs. SNR (dotted: true 4s). The shaded band is the in-band SNR estimated on the real rCBF parcels (≈ 0.7 – 2); there the shape is noisy but the latency is recovered within ~ 0.5 s.

IV. CONCLUSIONS

We have built a tool that processes the MEA LFP and the laser-speckle data on a common time base. Four

blocks work on real data – filtering, Up/Down detection, propagation and re-alignment – and the coupling block is validated on synthetic data; each result can be reproduced from the interface with the parameters in the captions.

Some limitations remain. The propagation block gives speeds of 4–34 mm/s, in or just below the physiological range; the direction is robust to the electrode spacing, but the absolute speed scales linearly with it and the smallest clusters give the least reliable values, so the speed should be read together with the cluster size and R^2 . The detection score assumes two states; close to the lesion, where the cortex can stay silent for long stretches, the histogram loses its second mode and the Otsu threshold should be read with care (the tool shows the histogram for this reason). The slow-band CAR also removes any truly global synchronous component, so the block is better for *relative* timing across channels than for absolute synchrony.

The next step is what motivated the work: how the neurovascular coupling of slow waves changes in the acute phase after a focal lesion. The coupling block is ready for this, once a recording with LFP and LSCI taken at the same time on the same animal is available; the three read-outs of Sec. II E would then turn into real measurements of how the lesion changes the coupling.

Acknowledgments

I thank Roser Sala-Llonch and Maria V. Sánchez-Vives for their supervision, the ICFO Medical Optics group for the pre-processed laser-speckle data, and the IDIBAPS Systems Neuroscience laboratory for the electrophysiological recordings.

-
- [1] M. Steriade, A. Nuñez, F. Amzica, “A novel slow (< 1 Hz) oscillation of neocortical neurons in vivo”, *J. Neurosci.* **13**, 3252–3265 (1993).
 - [2] M.V. Sanchez-Vives, M. Massimini, M. Mattia, “Shaping the default activity pattern of the cortical network”, *Neuron* **94**, 993–1001 (2017).
 - [3] A. Devor et al., “Coupling of total hemoglobin concentration, oxygenation, and neural activity in rat somatosensory cortex”, *Neuron* **39**, 353–359 (2003).
 - [4] E.M.C. Hillman, “Coupling mechanism and significance of the BOLD signal: a status report”, *Annu. Rev. Neurosci.* **37**, 161–181 (2014).
 - [5] M. Ruiz-Mejias, L. Ciria-Suarez, M. Mattia, M.V. Sanchez-Vives, “Slow and fast rhythms generated in the cerebral cortex of the anesthetized mouse”, *J. Neurophysiol.* **106**, 2910–2921 (2011).
 - [6] P.J. Drew, “Neurovascular coupling: motive unknown”, *Trends Neurosci.* **45**, 809–819 (2022).
 - [7] M. Dasilva, A. Camassa, A. Navarro-Guzman et al., “Modulation of cortical slow oscillations and complexity across anesthesia levels”, *NeuroImage* **224**, 117415 (2021).
 - [8] N. Otsu, “A threshold selection method from gray-level histograms”, *IEEE Trans. Syst. Man Cybern.* **9**, 62–66 (1979).
 - [9] J.H. Ward, “Hierarchical grouping to optimize an objective function”, *J. Am. Stat. Assoc.* **58**, 236–244 (1963).

Una eina de software per a l'anàlisi simultània d'ones lentes corticals en registres electrofisiològics i hemodinàmics per laser speckle al model muri de lesió

Author: Abel Vázquez Santisteban, vazabel02@alumnes.ub.edu
Facultat de Física, Universitat de Barcelona, Barcelona, Spain.

UB Advisor: Roser Sala-Llonch
Universitat de Barcelona, Barcelona, Spain.

External Advisor: Maria V. Sánchez-Vives
IDIBAPS, Institut d'Investigacions Biomèdiques August Pi i Sunyer, Barcelona, Spain.

Resum: Les ones lentes corticals (< 2 Hz) es poden mesurar de dues maneres: elèctricament, a través del dispar de les neurones, o òpticament, a través dels canvis que produeixen al flux sanguini cerebral. Fins ara aquests dos punts de vista s'han estudiat amb instruments diferents i rarament al mateix animal. Aquest treball presenta una eina de software que ajunta els dos tipus de dades al model muri de lesió focal. L'eina llegeix un registre de 32 canals de potencials de camp local (LFP, Open Ephys, 5 kHz) d'una matriu de microelèctrodes (MEA) juntament amb imatges de contrast de speckle del flux sanguini cerebral, preprocessades pel grup de Medical Optics de l'ICFO, i les posa sobre un eix temporal comú. Descriu els quatre blocs de processament que implementa: (i) filtrat passabanda de Butterworth de fase nul·la; (ii) detecció d'estats Up/Down sobre una senyal de detecció construïda fusionant una estimació de log-MUA i una de variància d'alta banda mitjançant anàlisi de components principals, amb un llindar d'Otsu i una neteja per durada mínima; (iii) un bloc de propagació que agrupa canals per coherència de correlació creuada amb clustering jeràrquic i ajusta una ona plana bidimensional sobre la geometria del MEA per recuperar la velocitat i la direcció; i (iv) un visualitzador que realinea els canals pels retards retornats pel bloc de propagació. Els blocs s'executen sobre registres reals d'LFP d'un ratolí anestesià, i cada figura es reproduïx amb el mateix motor que mou la interfície gràfica. L'eina també implementa un cinquè bloc que relaciona el senyal elèctric i l'hemodinàmic per mapar l'acoblament neurovascular; està llest però encara no s'ha pogut provar, perquè no es disposa de cap registre amb les dues modalitats adquirides simultàniament al mateix animal. **Paraules clau:** Ones lentes, estats Up/Down, aacoblament neurovascular, laser speckle, propagació d'ones, processament de senyal.

ODSs: Aquest TFG està relacionat sobretot amb els ODS 3 (Salut i benestar), 4 (Educació de qualitat) i 9 (Indústria, innovació, infraestructures).

Objectius de Desenvolupament Sostenible (ODSs o SDGs)

1. Fi de les desigualtats		10. Reducció de les desigualtats	
2. Fam zero		11. Ciutats i comunitats sostenibles	
3. Salut i benestar	X	12. Consum i producció responsables	
4. Educació de qualitat	X	13. Acció climàtica	
5. Igualtat de gènere		14. Vida submarina	
6. Aigua neta i sanejament		15. Vida terrestre	
7. Energia neta i sostenible		16. Pau, justícia i institucions sòlides	
8. Treball digne i creixement econòmic		17. Aliança pels objectius	
9. Indústria, innovació, infraestructures	X		

Aquest TFG combina coneixements del grau de Física (processament de senyal, problemes inversos, estadística) amb una aplicació biomèdica. Es relaciona amb l'ODS 3 (fita 3.4: el desenvolupament d'una eina que permet caracteritzar simultàniament l'electrofisiologia i l'hemodinàmica del còrtex lesionat té interès traslacional per a patologies com l'epilèpsia o l'ictus), amb l'ODS 4 (fita 4.4: el treball conté una component formativa important en eines computacionals i analítiques) i amb l'ODS 9 (fita 9.5: el desenvolupament d'una eina reproducible i oberta enforteix la recerca científica interdisciplinària).

Appendix: experimental setup and interface

The recordings come from the setup of Fig. 5: the ICFO Medical Optics group performs the wide-field laser-speckle and multispectral (785/660 nm) imaging of regional cerebral blood flow and HbO/HbR, while the 32-channel MEA records the LFP through the same cranial window. These images are shown for context only.

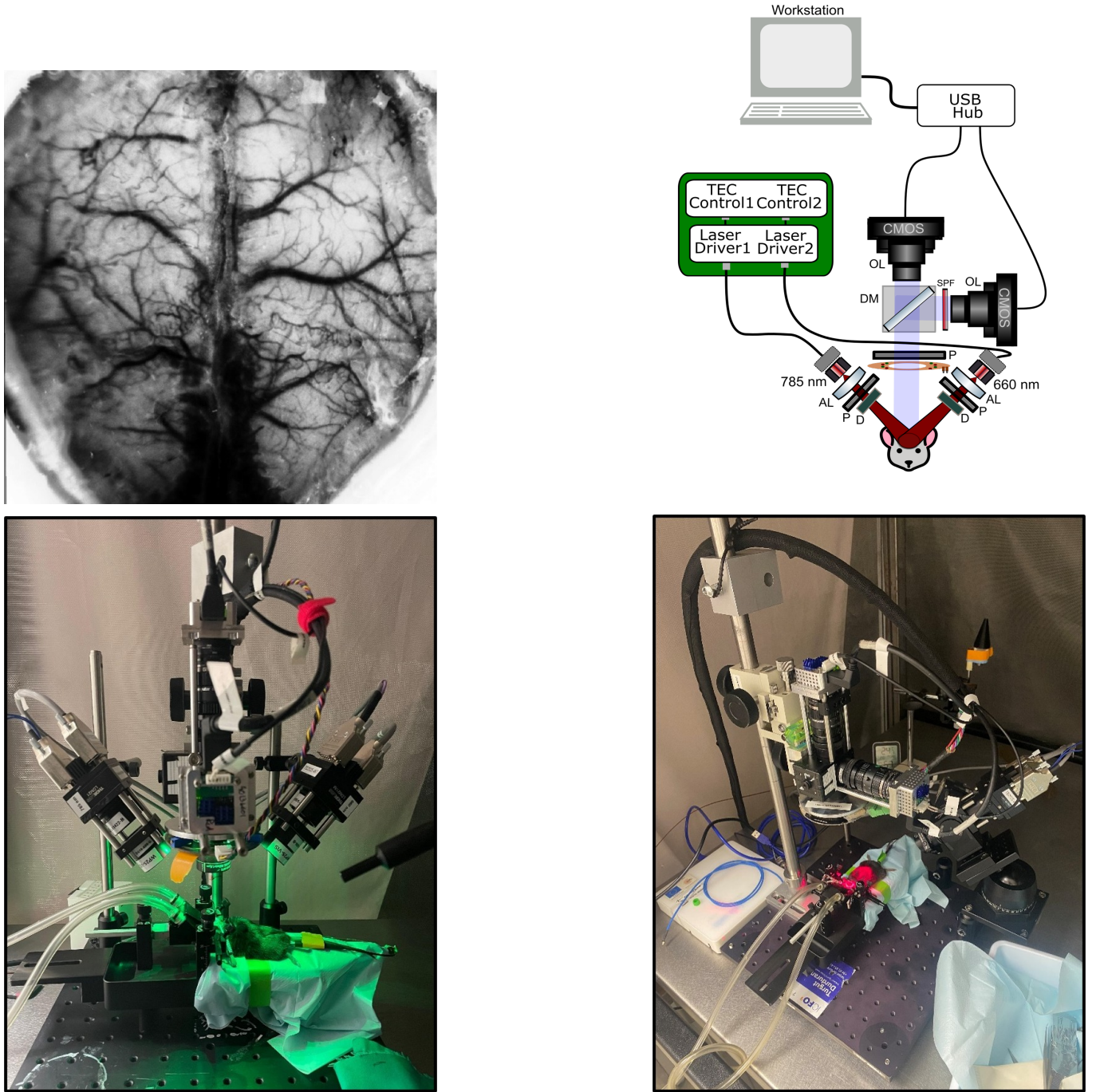


FIG. 5: Experimental setup: optical schematic (TEC-controlled 785/660 nm laser drivers and CMOS cameras), a wide-field laser-speckle image of the cortical vasculature, and two photographs of the imaging rig and the simultaneous laser-speckle + MEA preparation. Courtesy of the ICFO Medical Optics group.

The blocks of Sec. II–III run behind a graphical interface (Fig. 6).

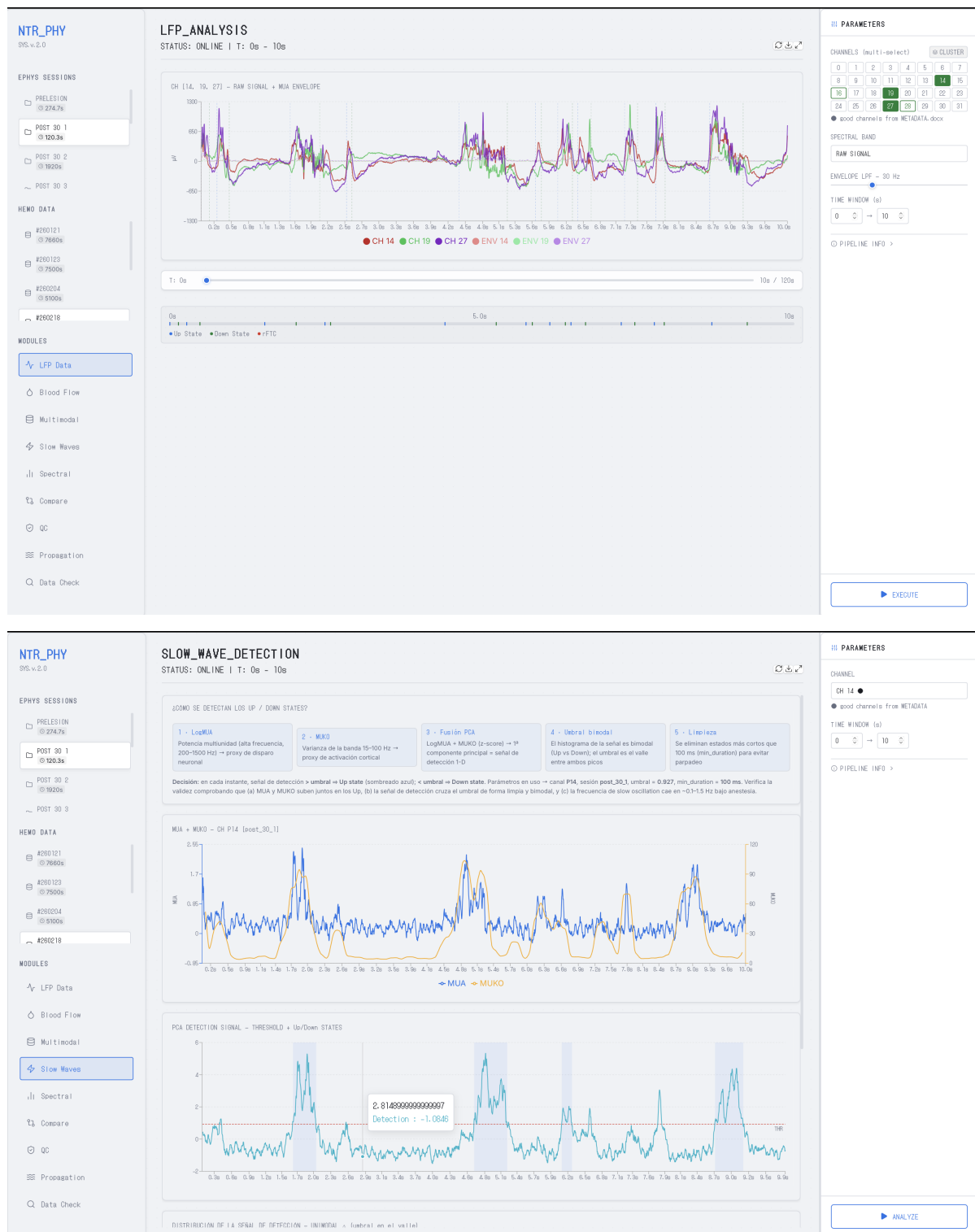


FIG. 6: The graphical interface. Top: the LFP viewer, with the raw signal and MUA envelope of three channels and the detected Up/Down events on the timeline below. Bottom: the Up/Down detection view, with the log-MUA and high-band-variance markers, the PCA detection signal and the detected Up states (shaded).