

EEG-fNIRS fusion for Practical Brain Computer Interfaces

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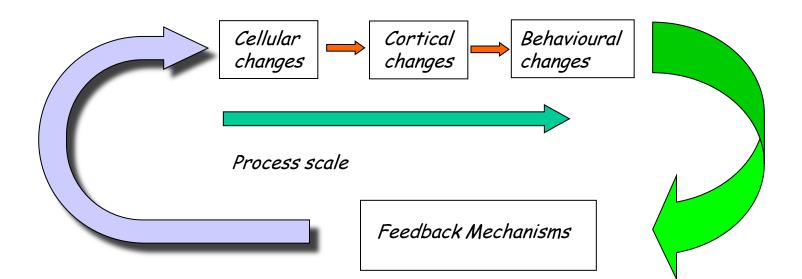
Stepping back: What is our 'anchor' problem?

- The problem is that 15 million people suffer a stroke annually. Of these 5 million are left with permanent disability which has severe economic and quality of life consequences.
- We are seeking to engineer technology (and science when required) which will improve recovery and function following stroke.



Stepping back: What is the science underpinning our approach?

The Science of Neuroplasticity Changes in brain structure as a result of experience and behaviour





What we are doing - specifically

• Science

- Discovering if/how, through appropriate feedback we can improve functional performance
 - Especially feedback of brain activity to improve motor function

• Engineering

- Novel instrumentation for cortical brain activity readout
 NIRS/EEG
- Game theoretic modelling of patient/therapist interaction
- Creation of web-technologies to facilitate distributed biosignal acquisition and processing

Application

 A neurorehabilitation system to improve functional outcome following stroke



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What I will be talking about today

• Science

Especially feedback of brain activity to improve motor function

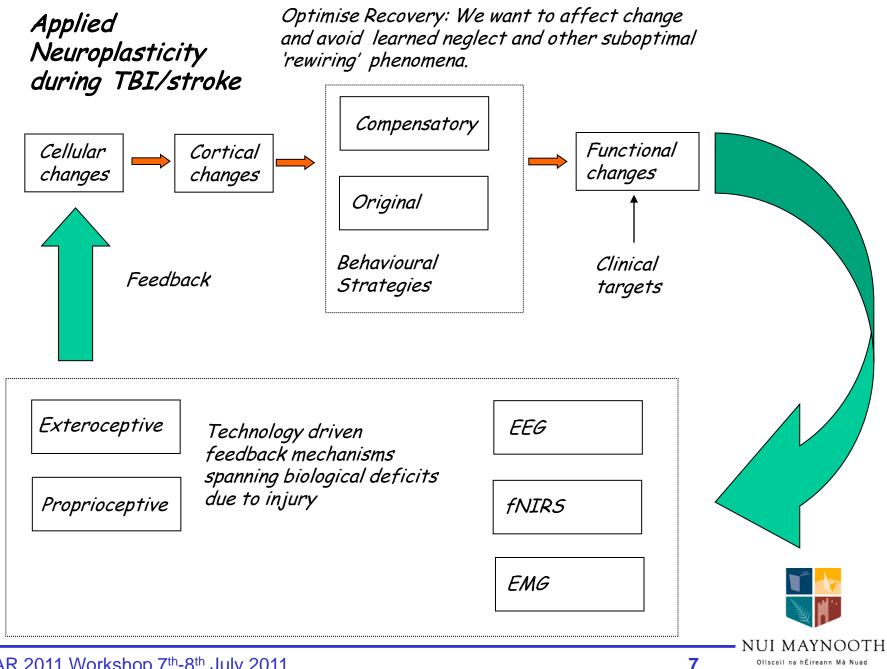
• Engineering

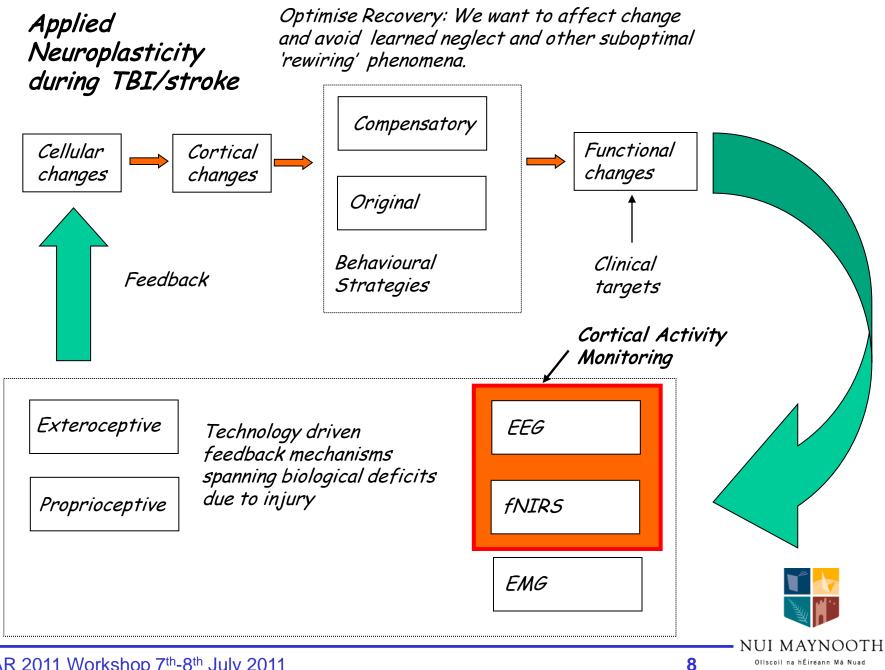
 Novel instrumentation for cortical brain activity readout – NIRS/EEG

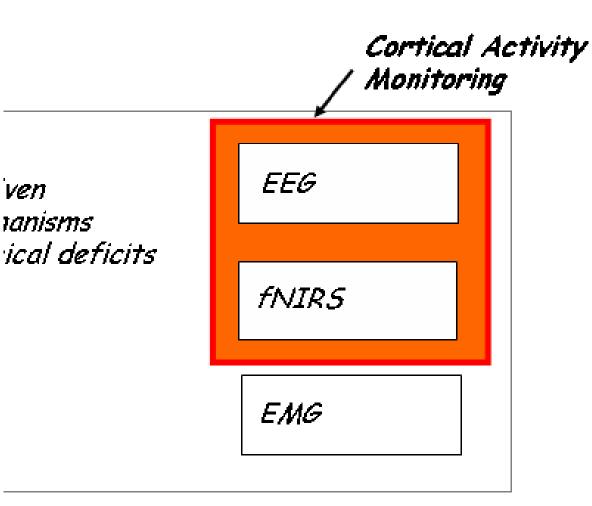
• Application

 A neurorehabilitation system to improve functional outcome following stroke









Q: So what are some of the practical problems for this subsystem?



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A: Usability versus Utility



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Usability



- The sensor system must be easy to use.
 - Simple: Unlikely that there is an neurophysiologist technician available every time patient wants to engage in therapy esp in domestic setting
 - Single: Single person required
 - Minimal: Minimise instrumentation complexity (e.g. number of application steps required – single step is best)
 - No gel if possible dry electrodes better, non contact even better!
 - Reliable: Must provide reliable measures of brain activation.
 - If patient tries to engage interface and nothing happens, patient != happy.



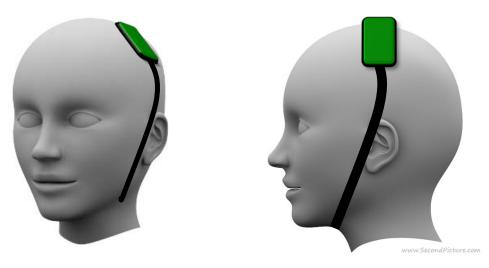
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Utility

- The system must be useful
 - Robust measures of brain activation required.
 - Unlike ALS patient, subject capable of head movement. The home is anything but an artefact free environment. Must make measurement in poorly controlled conditions.
 - **Precise** measures of brain activation required.
 - Something more than a binary signal may yield measures that can be related to outcome, neurovascular condition, cortical function.
 - Accurate measures of brain activation required.
 - False positives not a good idea for example



What we would wish our proposed solution to look like



- Usability: single step application via single cortical 'patch'
- Utility: dual NIRS/EEG to extract more useful signal per unit measurement area



Why dual modality? – redundancy

- We wish to measure over very specific cortical areas.
 - We access this area via the scalp
 - There is only so much scalp to work with for a specific cortical region.
 - NIRS optode positions do not overlap those of EEG for a given cortical target so maximising sensing area for a particular activation site



Why dual modality? – complementary

- EEG measures post synaptic potentials associated with neural activation
- fNIRS measures local haemodynamic changes associated with neural activation
- Provides better overall picture of cortical health and function and perhaps even state of recovery (hypothesis).
- Spatial resolution of fNIRS can be combined with temporal resolution of EEG



What else does fNIRS give us that might of use?

- Autonomic response through blood pressure fluctuations, which may be useful in more completely characterising motor effort:
 - Heart Rate
 - Heart Rate Variability
 - Respiration rate
 - Sympathetic nervous activity GSR-like measure
 - Mayer wave¹ changes may be indicative of systemic problems
 - Direct respiration rate
 - Oxygen saturation (pulse oximeter)

¹These are waves in arterial blood pressure brought about by oscillations in baroreceptor and chemoreceptor reflex control systems

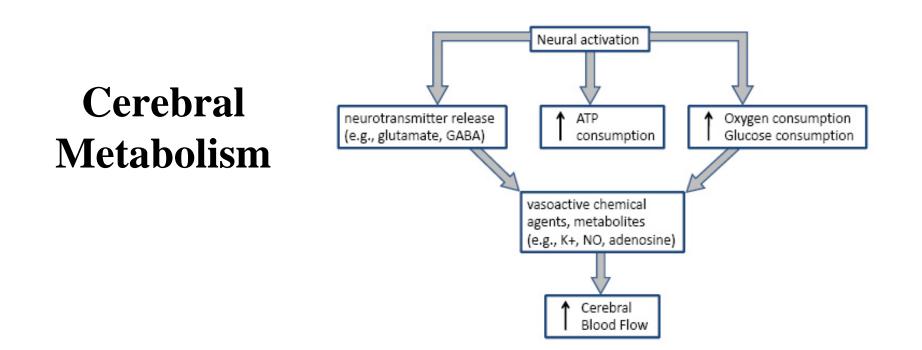


Q: What is the underlying physiology of the signal measured with fNIRS?

A: Haemodynamic changes associated with neural activation



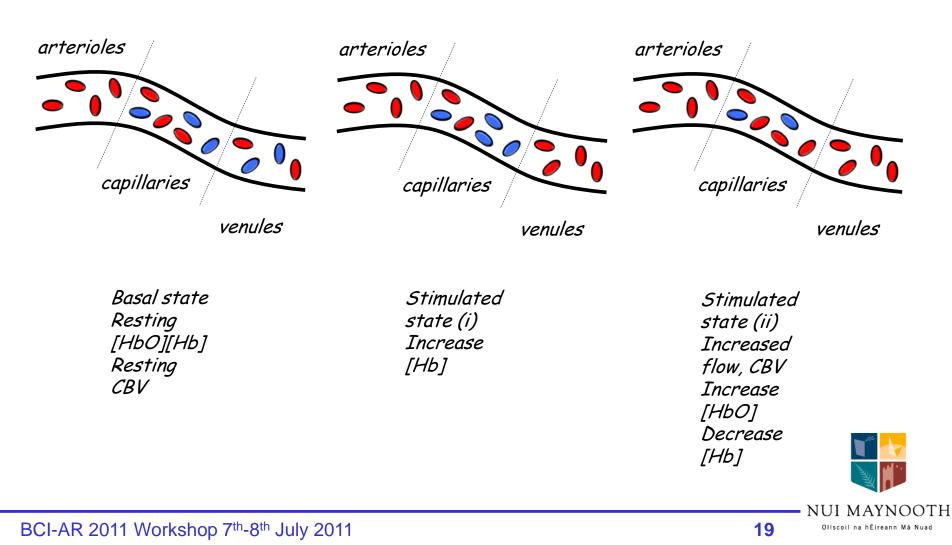
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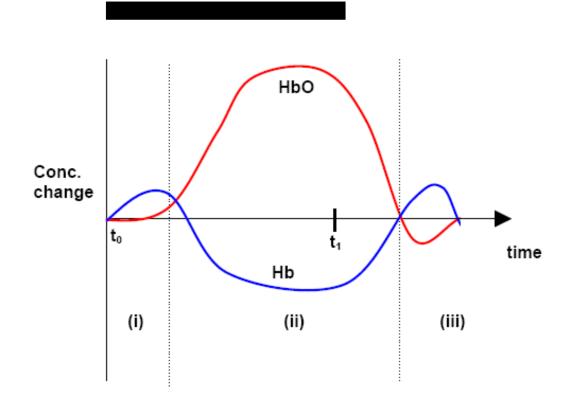
- Neural activation -> increase in consumption of glucose and oxygen -> increase in CBF
- While % increase in glucose consumption and CBF are similar, % increase in oxygen consumption is much less than CBF leading to net increase in amount of oxygen in blood in form of HbO
- This net increase in HbO then is source of signal



Haemodynamic Response Function



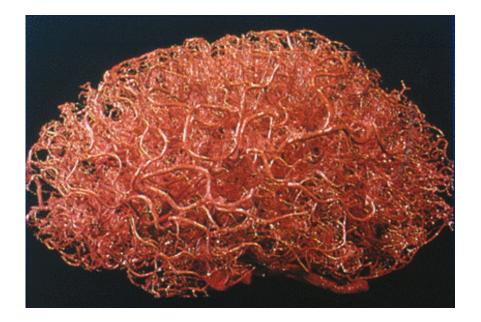
Haemodynamic Response Function



(i) Initial increase in Hb followed by (ii) increase in HbO, decrease in Hb and (iii) return to basal levels



What does the HR tell us about neural activity? - Spatial

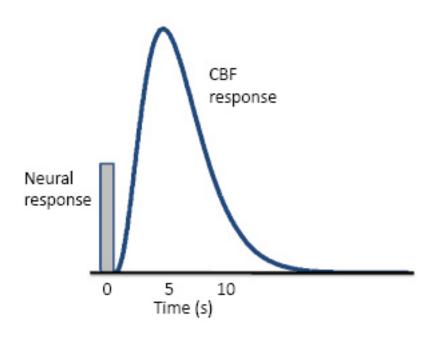


• Dense network of capillaries in grey matter (cortex) have spatial separation of ~25um so intrinsically high resolution if can separate contributions from larger arterial/venous vessels



What does the HR tell us about neural activity?- Timing

• Timing: CBF impulse response is delayed 1-2 seconds with 4-6 seconds to peak





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What does the HR tell us about neural activity?- Magnitude

- Amplitude: Generally linear for neural activity greater than a few seconds.
 - Some evidence that low intensity neural responses do not evoke a measurable CBF (fMRI)
 - Also while neural activity might saturate CBF can continue to grow



Q: How does fNIRS measure this haemodynamic response?

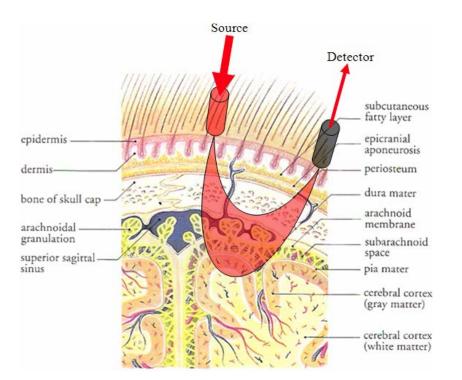
A: The haemodynamic response is associated with changes in optical absorption and scattering in the near infrared region (700-900nm)



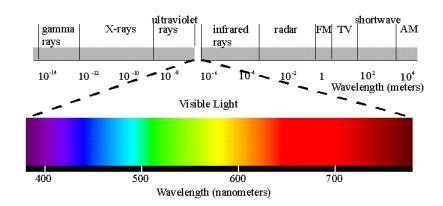
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Near infrared Spectroscopy

• It is an optical method for measuring haemodynamic signals at the cortex.



NIRS detects localised changes in cortical activity and associated blood flow due to an "Optical Window" (700nm - 900nm)

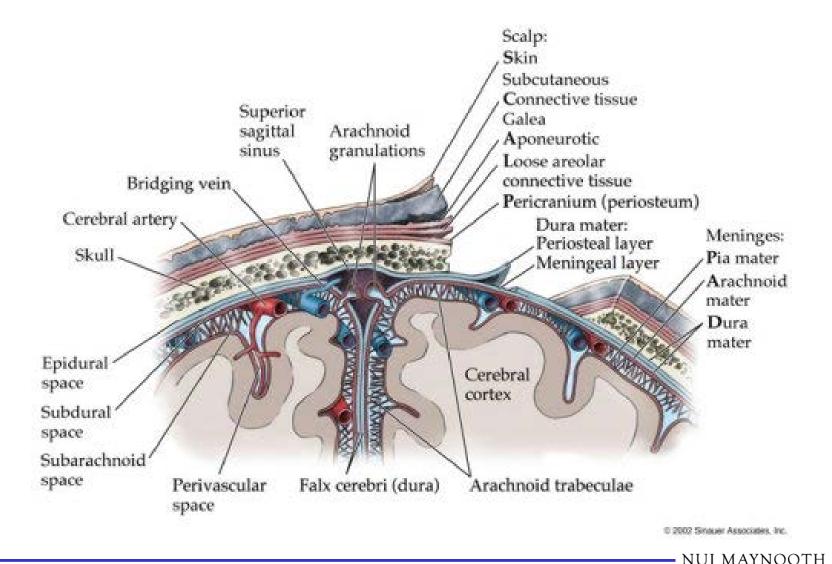


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Near-infrared light can penetrate to depths of 2-3cm below the surface of the scalp.

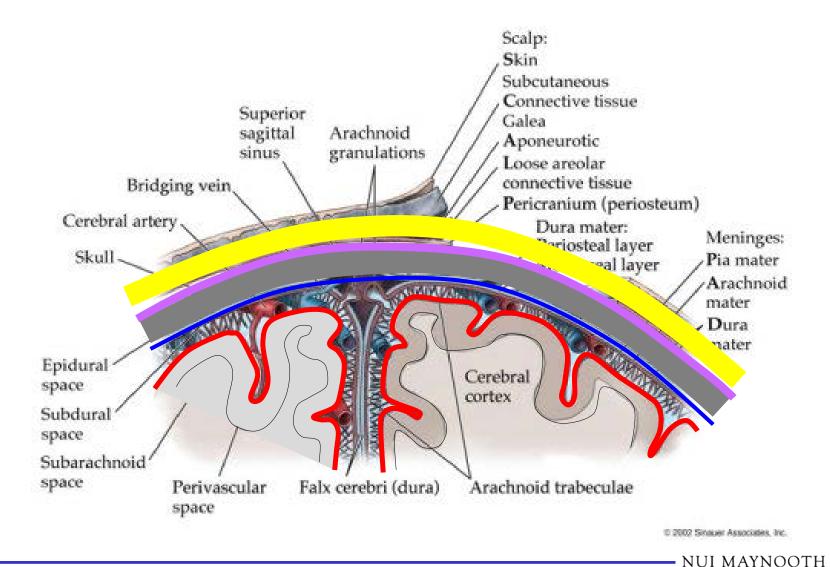






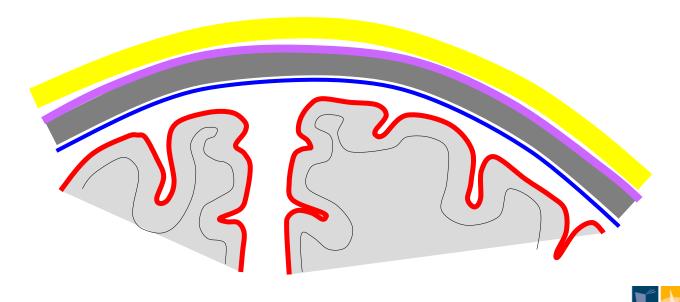
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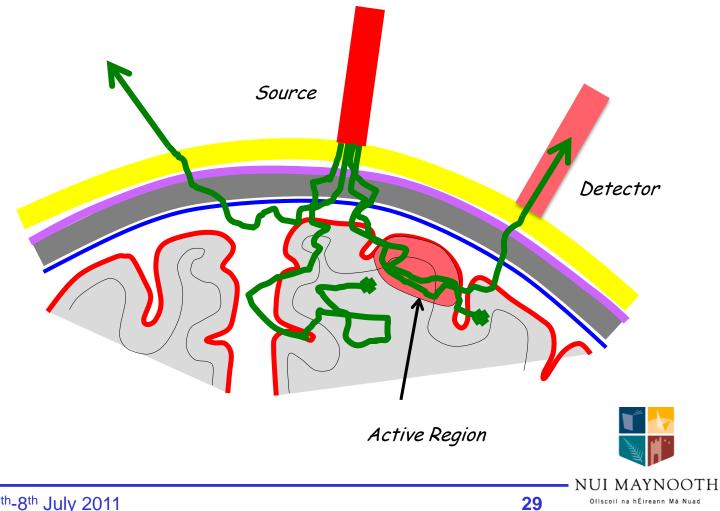
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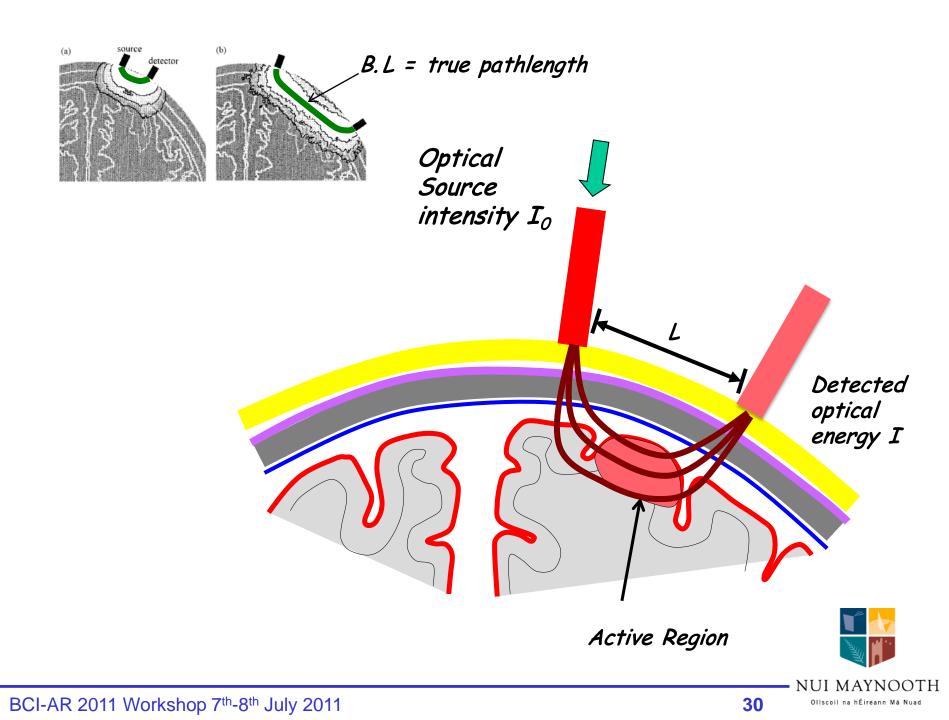
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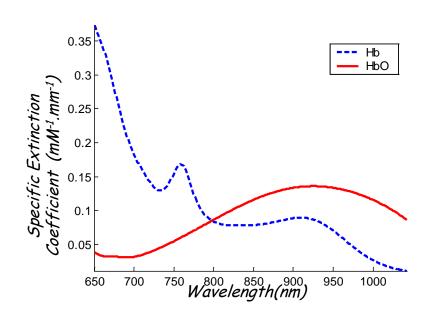




Absorption Changes - Quantitative

• The primary chromophores which are dynamic during activation are [HbO] and [HbR].

 Relative concentrations of Hb, HbO change during activation – due to neurovascular coupling





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Absorption Changes – Modified Beer-Lambert Law

$$A_{\lambda} = \log(\frac{1}{T}) = \left(\alpha_{\lambda,Hb} \cdot [Hb] + \alpha_{\lambda,HbO} \cdot [HbO]\right) B_{\lambda} \cdot L + G$$
(1).

Where *T* is the transmittance which is the ration of incident power to transmitted power, i.e. $\frac{I_{\lambda}}{I_{0,\lambda}}$.

$$\Delta A_{\lambda} = (\alpha_{\lambda,Hb} \Delta [Hb] + \alpha_{\lambda,HbO} \Delta [HbO]) B_{\lambda} L$$
(2),



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Modified Beer-Lambert Law Example

$$\frac{\Delta A_{760nm}}{B_{760nm}.L} = \left(\alpha_{760nm,Hb}.\Delta[Hb] + \alpha_{760nm,HbO}.\Delta[HbO]\right)$$
(3)

$$\frac{\Delta A_{880nm}}{B_{880nm}.L} = \left(\alpha_{880nm,Hb} \cdot \Delta [Hb] + \alpha_{880nm,HbO} \cdot \Delta [HbO] \right)$$
(4)

In matrix form these can be expressed as

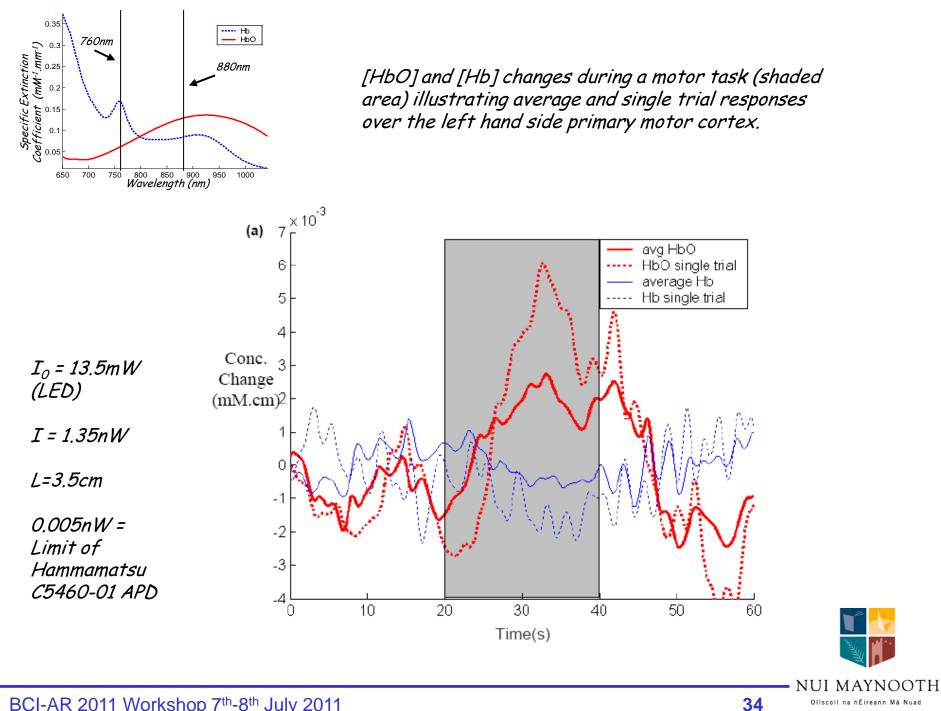
$$\mathbf{A}/\mathbf{B}\mathbf{L}=\boldsymbol{\alpha}\mathbf{C}$$
(5)

Where

$$\mathbf{A} = \begin{pmatrix} \Delta A_{760nm} \\ \Delta A_{880nm} \end{pmatrix}, \boldsymbol{\alpha} = \begin{pmatrix} \alpha_{760nm,Hb} & \alpha_{760nm,HbO} \\ \alpha_{880nm,Hb} & \alpha_{880nm,HbO} \end{pmatrix} \text{ and } \mathbf{C} = (\Delta [Hb] \quad \Delta [HbO])$$

Equation (5) is solved to extract **C** for each time sample as

 $\mathbf{C} = \boldsymbol{a}^{-1} \cdot \mathbf{A} / B \cdot L \tag{6}$

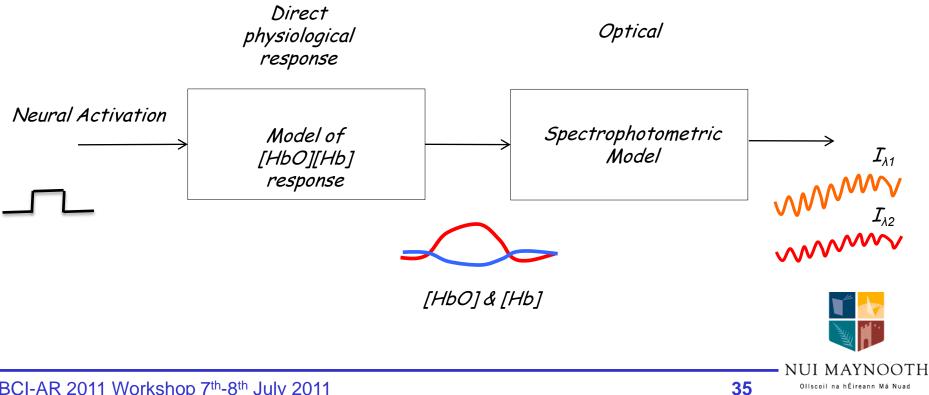


BCI-AR 2011 Workshop 7th-8th July 2011

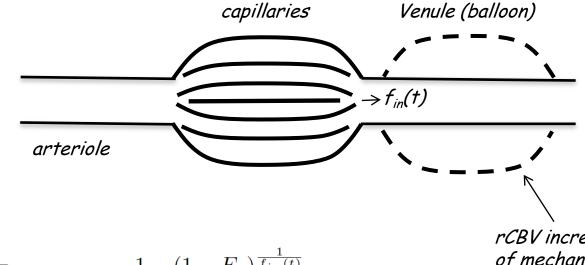
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Putting this altogether: A signal model for **fNIRS**

• Simulate fNIRS signal for signal processing research and development – key to advance performance outside the lab



Physiological Model for Neurovascular Coupling



$$\begin{split} E(t) &= 1 - (1 - E_0)^{\frac{1}{f_{in}(t)}} \\ \dot{q}(t) &= \frac{f_{in}(t)}{\tau_0} \left[\frac{E(t)}{E_0} - \frac{q(t)}{v(t)}\right] + \frac{1}{\tau_v} \left[f_{in}(t) - v^{\frac{1}{\alpha}}\right] \frac{q(t)}{v(t)} \\ \dot{v}(t) &= \frac{1}{\tau_v} \left[f_{in}(t) - v^{\frac{1}{\alpha}}\right] \\ \dot{p}(t) &= \frac{1}{\tau_v} \left[f_{in}(t) - v^{\frac{1}{\alpha}}\right] \frac{p(t)}{v(t)} \end{split}$$

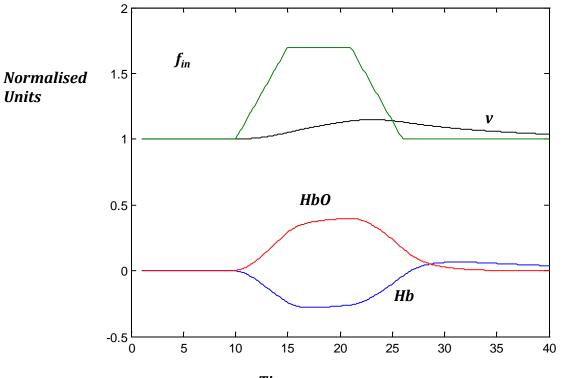
rCBV increase is as a result of mechanical distension of venule due to increase in CBF

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E= O₂ extraction rate q=[Hb] v=CBV p=[HbO]*



Simulated balloon model output



Time

Figure: The balloon model of the neurovascular response relating neural activation to changes in cerebral blood volume. From top to bottom the variables plotted are CBF, normalised blood volume, [HbO] and [Hb].



Spectrophotometric model

• Relate changes in scattering and absorption to light levels via MBLL

$$\Phi_s^{\lambda}(t) = \Phi_b^{\lambda}(t) + \Phi_c^{\lambda}(t) + \Phi_m^{\lambda}(t) + \Phi_n^{\lambda}(t) + \Phi_o^{\lambda}$$
$$\Phi_b^{\lambda}(t) = exp(-\Delta A_b^{\lambda}(t))$$
$$\Phi_c^{\lambda}(t) = K_c^{\lambda} f(k(t), R(t))$$
$$\Phi_m^{\lambda}(t) = K_m^{\lambda} \sin(2\pi f_m t + \theta_m)$$
$$\Phi_n^{\lambda}(t) = N(0, (\sigma_n^{\lambda})^2)$$



Synthetic signal comparisons

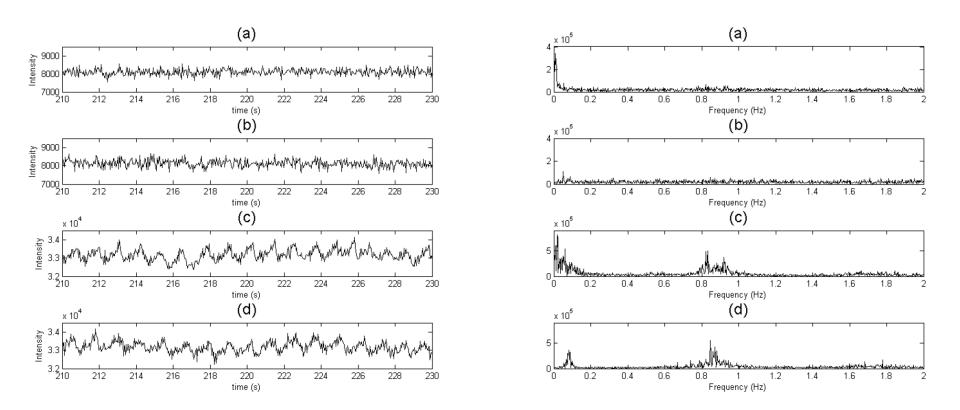


Figure: Visual comparison of synthetic fNIRS model with real optical density signals. The left hand side is the time domain while the corresponding spectra are on the right hand side. (a) Actual measurement at 690nm, (b) synthetic output for 690nm, (c) actual measurement at 830nm and (d) synthetic output at 830 nm.



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Averaged modelled [HbO][Hb] concentrations

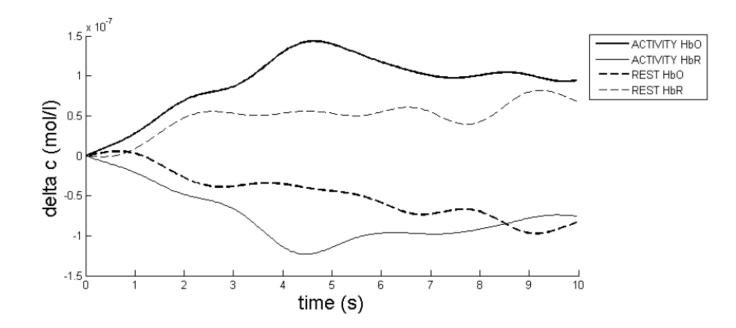


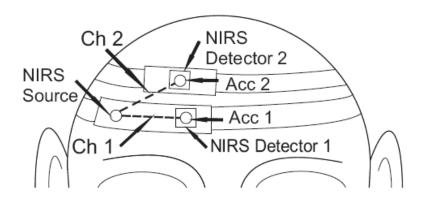
Figure: Averaged derived changes in [HbO] and [Hb] responses both at rest and in response to activation using the synthetic signal model



Value of this model

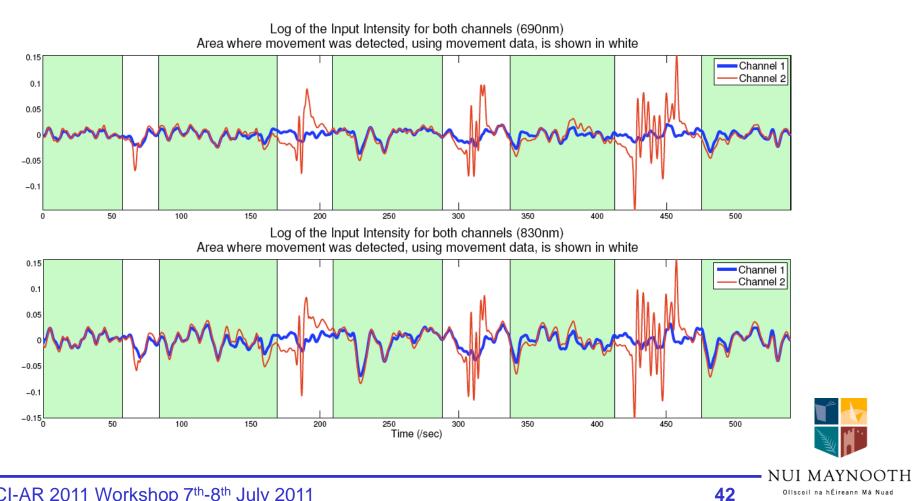
- Investigate the nature of artefact.
 - For example motion artefact can be induced through modulation of *L* (*next slide*).
- Adaptive filtering methods can be designed and investigated for removal of respiratory rate, cardiac pulsations, etc. (*next slide*)
 - Examine role of superficial channels
- Correlate with real responses
- Can be integrated with EEG model to produce a comprehensive compound signal future work





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Motion Artefact



Using Finger-derived PPG to remove cardiac pulse

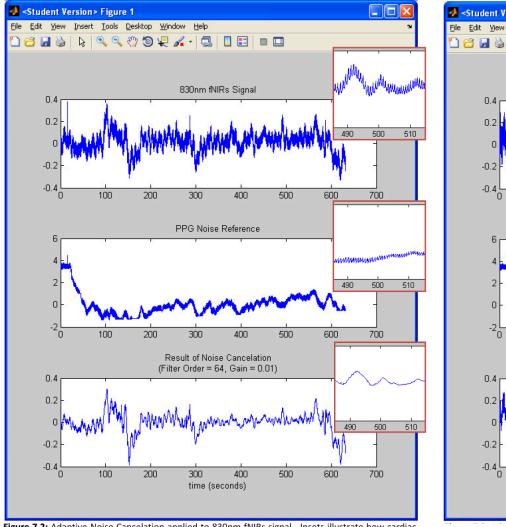
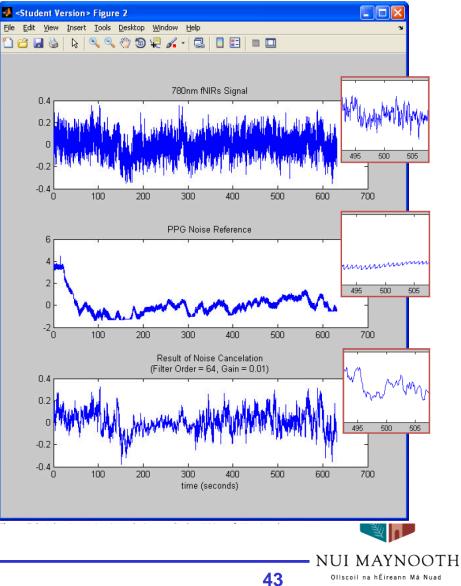


Figure 7.2: Adaptive Noise Cancelation applied to 830nm fNIRs signal. Insets illustrate how cardiac noise has been almost completely removed.

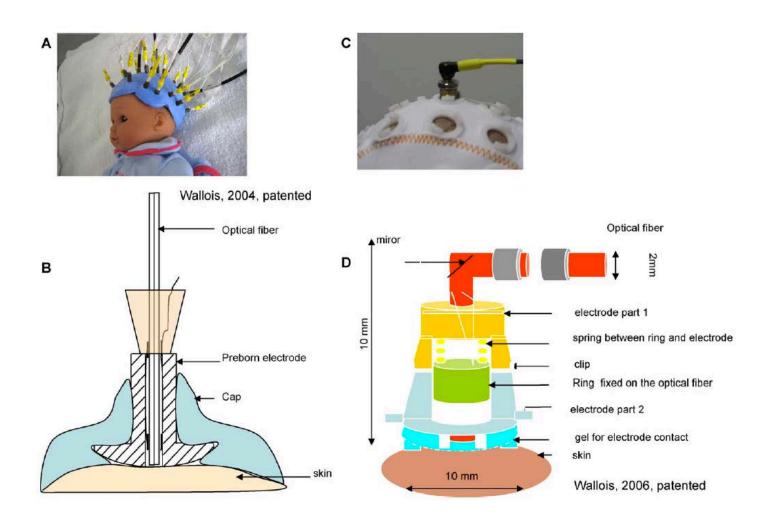


Current combined electro-optical probes



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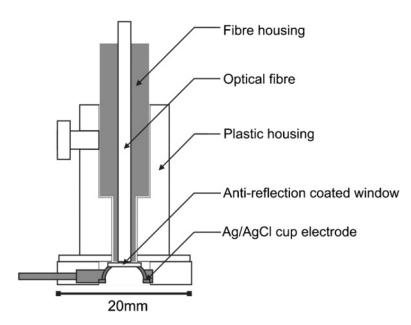
Wallois Electropode





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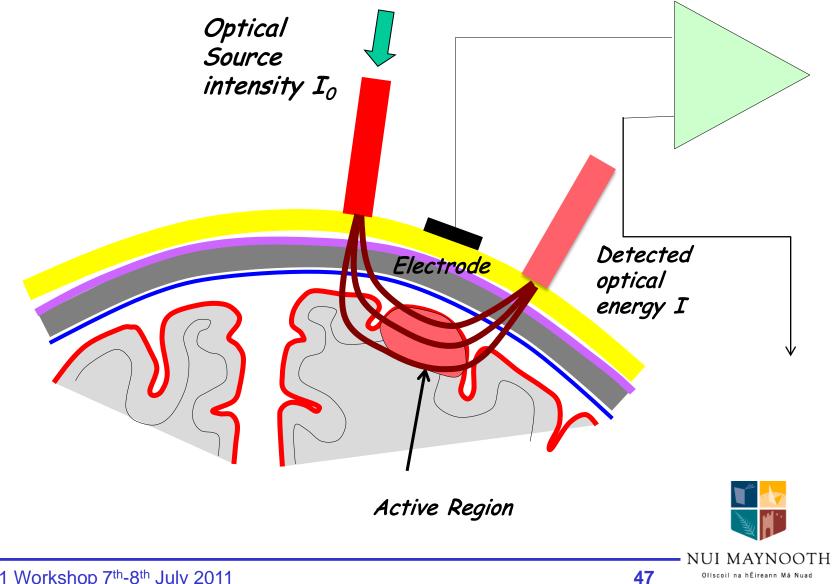
Cooper Electrode/Optode



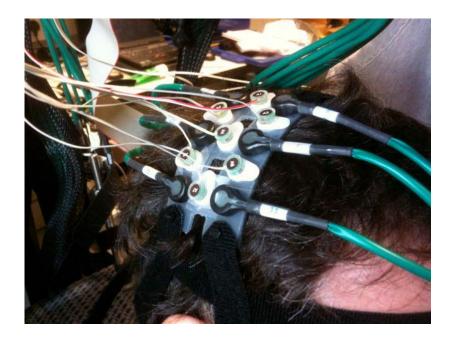
R. J. Cooper, N. L. Everdell, L. C. Enfield, A. P. Gibson, A. Worley, and J. C. Hebden, "Design and evaluation of a probe for simultaneous EEG and near-infrared imaging of cortical activation," Phys. Med. Biol. 54(7), 2093-2102 (2009)

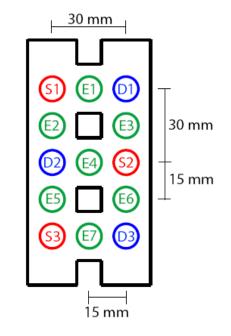


Maynooth Electrode/Optode



Probe Geometry





fNIRS data was recorded using a TechEn CW6 system (TechEn Inc., USA). Wavelengths used were 690 nm and 830 nm, sampled at 25 Hz. EEG data was recorded using a BioSemi Active-Two system (BioSemi Inc., The Netherlands). DC coupled data was recorded at 2048 Hz.



Simple Experiment

- Overt finger tapping
- Imagined finger tapping
- Postioned C3/C4 handedness dependent
- 20 trials per subject
 - No extensive training naïve performance
- EEG features Premovement μ -ERD/ postmovement β -ERS
- fNIRS features– Elevation in [HbO], fall in [Hb]
- Classifier LDA EEG separately, fNIRS separately, combined space



Classification with LDA – Overt finger tapping

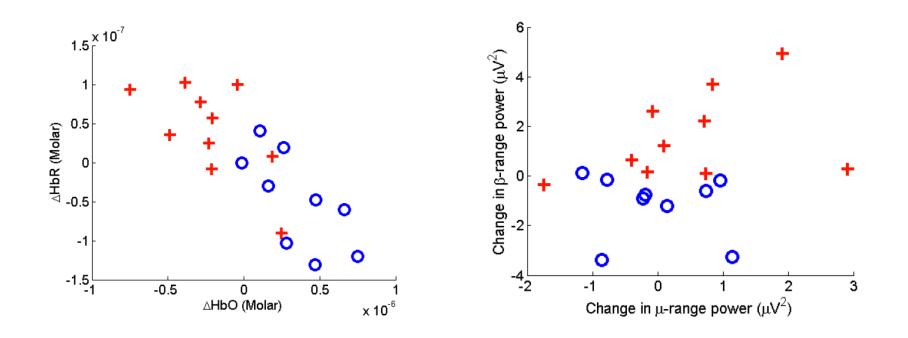


Figure 1: 2D fNIRS feature space for Channel 2 of Subject A, Trial 1. Crosses indicate feature locations when subject is in a rest period. Circles indicate feature locations when subject is in a fingertapping period.

Figure 2: 2D EEG feature space for Channel 2 of Subject A, Trial 1. Crosses indicate feature locations when subject is in a rest period. Circles indicate feature locations when subject is in a finger-tapping period.

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Classification with LDA – Overt finger tapping

	Subject A						Subject B					
	Trial 1			Trial 2			Trial 1			Trial 2		
Channel	fNIRS	EEG	Comb.	fNIRS	EEG	Comb.	fNIRS	EEG	Comb.	fNIRS	EEG	Comb.
1	84%	79%	90%	100%	84%	95%	79%	84%	95%	79%	84%	84%
2	79%	79%	84%	95%	79%	95%	47%	79%	63%	53%	84%	84%
3	100%	74%	95%	100%	84%	100%	79%	74%	79%	84%	84%	84%
4	95%	84%	95%	84%	84%	79%	74%	74%	63%	53%	90%	79%
5	74%	74%	84%	42%	74%	58%	47%	68%	47%	74%	79%	95%
6	100%	90%	100%	95%	74%	90%	58%	74%	68%	79%	84%	79%
7	58%	84%	84%	68%	68%	79%	68%	63%	68%	58%	74%	63%
Average	84%	80%	90%	83%	78%	85%	65%	74%	69%	68%	83%	81%



Classification with LDA – Imagined Movement

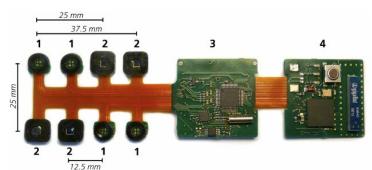
	S	ubject A		Subject B			
Channel	f NIRS	EEG	Dual	f NIRS	EEG	Dual	
1	59%	51%	64%	64%	46%	62%	
2	56%	59%	67%	51%	54%	59%	
3	56%	54%	64%	61%	41%	56%	
4	69%	67%	72%	64%	59%	67%	
5	61%	51%	72%	41%	36%	46%	
6	56%	77%	64%	74%	59%	69%	
7	56%	59%	62%	15%	43%	49%	
Average	59%	60%	66%	53%	48%	58%	



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Future Work

- More subjects more trials
- Bilateral measurements
- Stroke patients
- Minature wireless apparatus for EEG/fNIRS
- Improved signal models
- Simpler application



Opt Express. 2008 Jul 7;16(14):10323-30. Wireless miniaturized in-vivo near infrared imaging. Muehlemann T, Haensse D, Wolf M.



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Summary

- fNIRS/EEG has potential as a BCI interface technology which may be particularly useful for damaged brain
- fNIRS/EEG probe makes more efficient use of scalp area
 - Given move to dry electrodes with poorer SNR, combination with fNIRS may restore performance
- fNIRS/EEG may give good spatial resolution AND good temporal resolution
- fNIRS still in development better and cheaper technology will come



End



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