Cerebral Autoregulation Assessment using Electroencephalograms

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ABSTRACT

This paper presents the possibility of using Electroencephalograms (EEG) signals of an individual for quantitative interpretation of Cerebral Autoregulation (CA). EEG data was recorded during arm cuff inflation to induce dynamic changes in arterial blood pressure and then, Cerebral Blood Flow (CBF) was estimated from EEG using canonical hemodynamic response function (HRF). The assessment of CA was carried out by using power of the various frequency bands of EEG signal.

Keywords

Cerebral Autoregulation, Cerebral Blood Flow, Blood Pressure, Electroencephalograms

1. INTRODUCTION

Cerebral blood flow (CBF) is the measure of blood supply to the brain in a given time and typically varies between 50 to 54 ml/100g/min (100g of brain tissue). Brain needs continuous adequate supply of blood for its proper functioning. Cerebral Autoregulation (CA) refers to the intrinsic ability of the brain to maintain constant cerebral blood flow despite changes in perfusion pressure due to active modulation of cerebrovascular resistance/conductance as shown in Figure 1 [8].

It becomes increasingly important to monitor CA in a number of common disorders such as hyper/hypotension, stroke, trauma or other serious mental illness requiring intensive care. Monitoring of cerebral blood flow is extremely useful for population at higher risk of developing stroke. It comes handy as an alternate way of planning effective strategies to minimize consequences of cerebral ischemia. The symptoms of a brain injury detected by hospital examination often occur at late stages of brain health deterioration [3]. Some clinical ways of CBF measurement include using Xenon-133, Thermal Diffusion Technique, Laser Doppler Flowmetry (LDF), Jugular Venous Oximetry, Transcranial Doppler Sonography and Near Infrared Spectroscopy (NIRS)[12]. MRI techniques for calculating CBF in acute settings is not feasible for practical applications. All the above mentioned techniques lack the possibility of detecting flow abnormalities that can occur between short spans over different times before causing a significant/irreversible brain damage. The focus of the present study was to explore the possibility of a quantitative measure of cerebral autoregulation by non-invasive means using individuals' EEG signals.

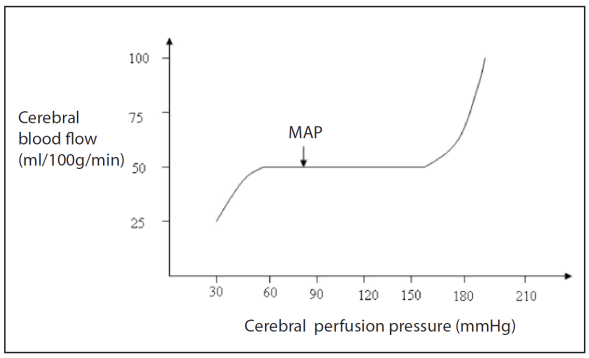

Figure 1: Relationship between Cerebral Blood Flow and Cerebral Perfusion Pressure [8]

2. PHYSIOLOGY OF AUTOREGULATION

Ample literature confirms the existence of neurovascular coupling between EEG signals and CBF [2][6]. EEG-CBF correlation can be considered as a result of the change in local neuronal activity, which affects local blood flow regulation. This is because regional metabolic demand increases, requiring more oxygen and energy. As a result, the byproducts of metabolism (CO2, NO, lactate etc.) act as vasodilators causing relaxation of smooth blood vessels and increases blood flow. As CBF serves to deliver metabolic substrates and also washes away waste products of metabolism, focal increases in CBF closely follow neural activity and hence, measures local changes in EEG signal. Figure 2 shows the mechanism of neurovascular coupling [4].

Using Statistical Parametric Mapping's (SPM) canonical HRF function that serves as a good link function for the neurovascular coupling [9], we demonstrate observed changes in CBF with EEG recorded activities. The estimated CBF was obtained from EEG signals via a linear convolution of canon-

BODYNETS 2013, September 30-October 02, Boston, United States

DOI 10.4108/icst.bodynets.2013.253703

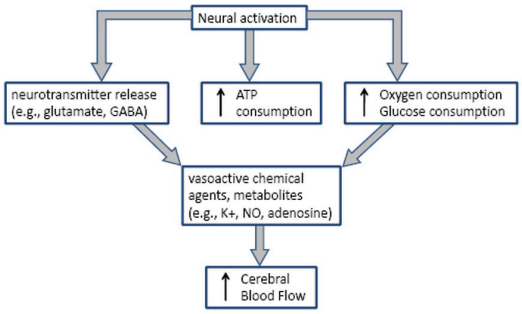

Figure 2: Flowchart showing vascular response as a result of neuronal activity [4]

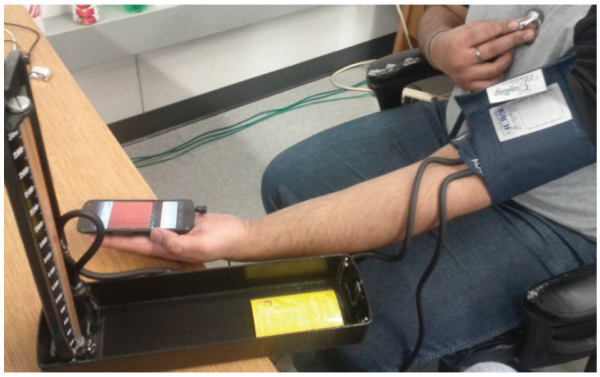

Figure 3: Experimental setup to acquire simultaneous recording of blood pressure and EEG signals during arm cuff inflation

ical HRF with the neural activity (as measured from the instantaneous EEG signal activity).

CBF is dependent on a number of factors but can be broadly related to cerebral perfusion pressure (CPP) and cerebrovascular resistance (CVR) [11] by the equation given below

$$CBF = \frac{(CPP)}{(CVR)} \quad (1)$$

$$CPP = MAP - ICP \quad (2)$$

$$MAP = DP + \frac{(SP - DP)}{3} \quad (3)$$

where MAP = Mean Arterial Pressure, ICP = Intracranial pressure, DP = Diastolic Pressure, SP = Systolic Pressure.

3. EXPERIMENTAL SETUP

Cerebral auto regulation refers to maintaining a constant flow of blood over a range of mean arterial pressures (60-160 mmHg) assuming in normal adults ICP is less than 10mmHg (constant) [5]. In order to quantify cerebral autoregulation, variation in MAP was evoked by using arm cuff inflation described in the following section. The estimated CBF derived from corresponding EEG activity was also observed, analysed and discussed.

3.1 Data Collection

Large quantitative variation in EEG signals corresponding to changes in MAP can be induced pharmacologically but may expose subjects to risk of stroke. Previously, mean arterial pressures have been elevated with the application of leg compression cuffs with no changes in heart rate, cardiac output, thoracic impedance and central venous pressure [7][10]. Similar approach was adopted in this experiment using a sphygmomanometer and the EEG headband. Figure 3 shows the experimental setup to study the changes in EEG signals corresponding to changes in mean arterial pressure. It is harder to measure MAP compared to the blood pressure. Hence, the change in MAP can be reflected by evoking changes in instantaneous blood pressure as they are related according to equation-3. The EEG signals were recorded on the laptop via a Bluetooth interface using the single dry electrode headband from Neurosky Incorporation.

The subjects were seated in a chair with closed eyes and data was recorded from the FP1 location according to the 10-20 international system of electrode placement. The cuff was inflated in the arm to restrict blood flow (140 mmHg) and held constant for the time the EEG signals were recorded from the subject (10 secs). The blood pressure was also recorded simultaneously along with the EEG data by using the blood pressure android application on a smart phone as shown in Table 1. Data was recorded from two subjects with five trials each for the baseline and arm cuff inflation activity.

Table 1: Systolic and Diastolic variation in blood pressure during arm cuff inflation

Baseline		Arm Cuff Inflation	
Systolic	Diastolic	Systolic	Diastolic
117	74	130	81
119	75	128	79
118	74	129	80
115	72	125	77

4. OBSERVATIONS

This section provides a discussion of the observations in terms of the derived EEG parameters and estimated CBF variations.

4.1 EEG Parameters

Studying EEG signals in time domain or only its power spectrum, changes in the underlying brain activity were difficult to observe as illustrated by Figures 4 and 5. EEG parameters were derived from the decomposed frequency bands of the power spectrum of EEG signal. They were used to quantify the changes in EEG activity compared to the baseline activity. The relative frequency power change was defined as Delta (1-4)(δ), Theta (4-8)(θ), Alpha (8-12)(α), Beta (12-30)(β), Gamma (30-50) (γ) Hz frequency power divided by the Total power (1-50 Hz), calculated over every 0.25 seconds time windows.

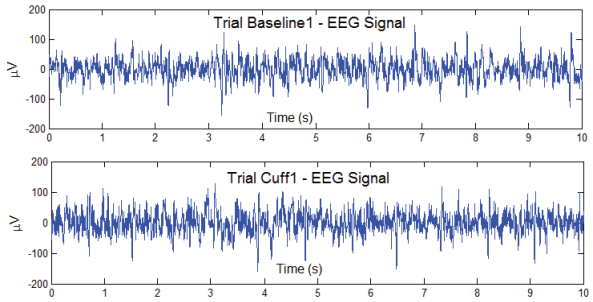

Figure 4: EEG signal recording during baseline and arm cuff inflation activities

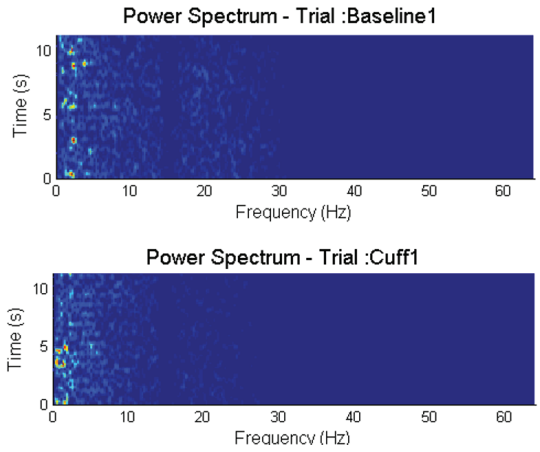

Figure 5: EEG power spectrum of baseline and arm cuff inflation activities

Figure 6 shows the power variation in these bands during arm cuff inflation activity compared to baseline activity. A higher order relative power during cuffing compared to the baseline activity was observed. Using such EEG parameters, changes related to EEG with blood pressure could be studied quantitatively.

4.2 CBF Variations

CBF was estimated from the EEG signal data using the hemodynamic response as described in section-II. A comparison in the variation of estimated CBF from the two activities is discussed here. It took around two seconds for the estimated CBF to reach to a peak value before it settled down to a base value as shown in Figure 7. It was interesting to see that inflating arm cuffs caused temporary increase in mean arterial pressure reflected in the increased estimated CBF values as shown compared to base value whereas no visible changes were observed in EEG waveforms in time domain to detect any such phenomenon. The fall back of estimated CBF to the base value in arm cuff activity took about six seconds which again strengthened our observation of detecting increase in estimated CBF with increase in blood pressure. This indicated that autoregulation mechanisms could be reflected by analyzing the behavior of EEG signals.

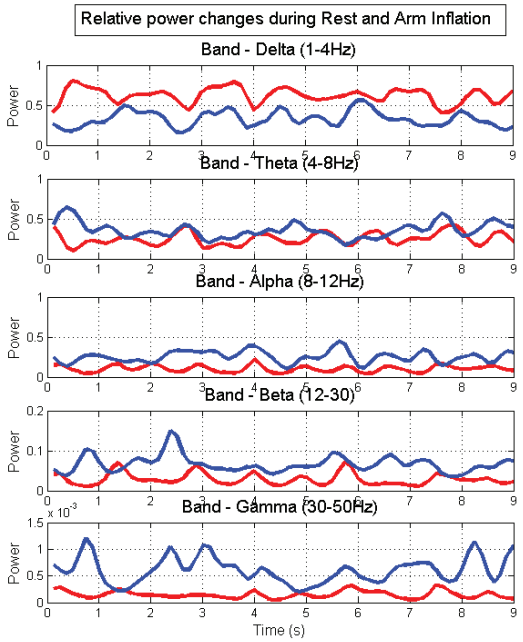

Figure 6: Relative power variation in frequency bands during Baseline (red) and Arm Cuff Inflation (blue)

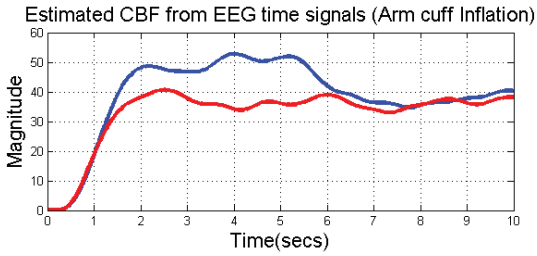

Figure 7: Comparison of estimated CBF for Baseline (red) and Arm Cuff Inflation (blue)

5. CONCLUSION

This paper presented the scope of EEG as a monitoring tool for the mechanism of Cerebral Autoregulation. It was shown by studying quantitative EEG parameters such as relative power spectrum which exhibited variability with a change in mean arterial pressure. The observation was strengthened by analyzing the change in behavior (rise and fall) of estimated cerebral blood flow as well. Thus, the system attempted to make quantitative EEG as a firsthand measure to check impaired autoregulation in the brain.

6. FUTURE WORK

We are currently working on porting our quantitative EEG measures over to an Android mobile platform which is capable of recording and transmitting EEG signals in real-time [1]. Although, we are able to show the possibility of quantifying Cerebral Autoregulation via EEG, appropriate cut-offs have to be determined to state if the change in parameters are significant to be alarmed. Last, it is important to ana-

lyze the sensitivity of this approach to avoid confusion with artifacts.

7. ACKNOWLEDGEMENTS

This work is partially supported by the National Science Foundation under grants CNS-0751205, CNS-0821736 and CNS-1229700.

8. REFERENCES

[1] G. Bajwa, R. Dantu, M. Mohamed Issadeen, and R. Joseph. Self-tracking via brain-mobile-cloud interface. *AAAI Spring Symposium Series, North America*, Mar 2013.

[2] R. B. Buxton, K. Uludag, D. J. Dubowitz, and T. T. Liu. Modeling the hemodynamic response to brain activation. *NeuroImage*, 23, Supplement 1(0):S220 – S233, 2004. Mathematics in Brain Imaging.

[3] M. Diop, K. M. Tichauer, J. T. Elliott, M. Migueis, T.-Y. Lee, and K. S. Lawrence. Time-resolved near-infrared technique for bedside monitoring of absolute cerebral blood flow. In *Proc. SPIE*, volume 7555, page 75550Z, 2010.

[4] R. D. Freeman, B. N. Pasley. Neurovascular coupling. *Scholarpedia*, 3(3):5340, 2008.

[5] E. Gao, W. L. Young, J. Pile-Spellman, E. Ornstein, and Q. Ma. Mathematical considerations for modeling cerebral blood flow autoregulation to systemic arterial pressure. *American Journal of Physiology - Heart and Circulatory Physiology*, 274(3):H1023–H1031, 1998.

[6] Z. Y. C. W. Liu X, Zhu X-H. Tightly coupled spontaneous eeg and cbf signals in the anesthetized rat brain. In *Proceedings of the 16th Annual Meeting of ISMRM*, page 755, Toronto, ON, CA, 2008.

[7] R. B. Panerai, R. P. White, H. S. Markus, and D. H. Evans. Grading of cerebral dynamic autoregulation from spontaneous fluctuations in arterial blood pressure. *Stroke*, 29(11):2341–2346, Nov. 1998.

[8] E. Shardlow and A. Jackson. Cerebral blood flow and intracranial pressure. *Anaesthesia and Intensive Care Medicine*, 12(5):220 – 223, 2011.

[9] SPM. Statistical parametric mapping available: http://www.fil.ion.ucl.ac.uk/spm/.

[10] J. Williamson, J. Mitchell, H. Olesen, P. Raven, and N. Secher. Reflex increase in blood pressure induced by leg compression in man. *J Physiol*, 475(2):351–7, 1994.

[11] A. Zauner and J. P. Muizelaar. *HEAD INJURY: Pathophysiology and management of severe closed injury; Brain metabolism and cerebral blood flow*. 1997.

[12] A. Zauner and J. P. Muizelaar. *HEAD INJURY: Pathophysiology and management of severe closed injury; Measuring Cerebral Blood Flow and Metabolism*. 1997.