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Measurements of Cerebral Arterial Oxygen Saturation using a Fiber-optic Pulse Oximeter

Justin P. Phillips, *Member, IEEE*, Richard M. Langford, Serene H. Chang, Kishore Maney, Panayiotis A. Kyriacou, *Senior Member, IEEE*, Deric P. Jones, *Senior Member, IEEE*.

Abstract— This pilot investigation was undertaken to assess the performance of a novel fiber-optic cerebral pulse oximetry system. A fiber-optic probe designed to pass through the lumen of a cranial bolt of the type used to make intracranial pressure measurements was used to obtain optical reflectance signals directly from the brain tissue. Preliminary results from seven patients measured in the operating theatre and ITU are presented. Estimations of cerebral arterial oxygen saturation derived from a frequency domain-based algorithm are compared with pulse oximetry (SpO_2) and hemoximeter (SaO_2) blood samples. The mean ($\pm SD$) difference between cerebral oxygen saturation ($ScaO_2$) and finger SpO_2 (in saturation units) was $-7.47(\pm 3.4)\%$. The mean ($\pm SD$) difference between $ScaO_2$ and blood SaO_2 was $-7.37(\pm 2.8)\%$.

I. INTRODUCTION

Neurological damage following traumatic head injury or brain surgery does not all occur at the time of the impact or operation (primary injury), but can develop afterwards sometimes many hours or days later [1]. The commonest causes of such secondary injury are intracranial bleeding and swelling of the brain leading to raised intracranial pressure (ICP), reduced cerebral blood flow and perfusion and subsequent hypoxic brain damage [2]. The various ways to clinically monitor potential ischemia of the injured brain include the use of near-infrared spectroscopy [3].

Near-infrared spectroscopy (NIRS) is a noninvasive optical technique and is a modality that can be used for monitoring brain tissue oxygen saturation, changes in blood volume and, indirectly, brain blood flow and oxygen consumption [4]. With NIRS however, there is a high degree of uncertainty in the exact brain volume being assessed. Computer models have shown that the volume of tissue interrogated by NIRS typically consists of 30% brain and 70% non-cerebral tissues (i.e. scalp and skull) [5]. Absorption of light by these tissues, particularly the blood within the scalp vasculature complicates algorithms used in NIRS systems and can lead to inaccuracies in these systems' calculation of oxygen saturation [6, 7].

To avoid the problems associated with the absorption of light by the superficial tissues, it has been proposed that pulse oximetry measurements be made directly from the brain tissue [8]. An optical fiber based system for directly monitoring brain tissue hemoglobin oxygen saturation using photoplethysmography (PPG) has been developed. The fiber-optic probe is placed in direct contact with the brain tissue via a cranial bolt (Integra Neurosciences, Plainsboro, NJ, USA) of the type used for the measurement of intracranial pressure (ICP), thereby eliminating tissue such as the scalp, skull and dura from the optical path. The aim is to produce a rapidly responding indicator of cerebral hypoxemia, which may be used for patients in whom the use of a cranial bolt is indicated for measurement of ICP.

Short-duration measurements were conducted in patients undergoing neurosurgery at the Royal London Hospital, London UK. The primary aim was to determine whether adequate photoplethysmographic (PPG) signals could be obtained from the brain tissue. The study also aimed to verify whether the cranial bolt access system would serve as a suitable conduit for the fibers and whether the acquired signals would be suitable for calculation of oxygen saturation. Measurements of longer duration were also taken from a patient recovering from an intracerebral hemorrhage in the London Hospital ITU.

II. MATERIALS AND METHODS

A. Measurement system

The measurement system for this study [9] was developed and evaluated in our laboratory, based around a fiber optic reflectance PPG probe comprising two optical fibers, one of which was used to transmit light from red and infrared LEDs to the tissue and the other to return a fraction of the backscattered light to a photodiode. A signal processing system was interfaced to a 16-bit data acquisition card (National Instruments Inc. Austin, TX, USA) installed into a notebook computer. The optical fiber probe consisted of two silica optical fibers (a transmitting fiber and a receiving fiber) each with a core diameter of $400\ \mu\text{m}$, an outer cladding diameter of $730\ \mu\text{m}$ and a numerical aperture (NA) of 0.39 (Ocean Optics Inc., Dunedin, FL, USA). The distal end of each fiber was cut and polished flat and the proximal end terminated with a male SMA connector. The fibers were coated in a protective PVC jacket, which is stripped away over a length of 16 cm from the distal end. The probe

J. P. Phillips, P.A. Kyriacou and D.P. Jones are with the School of Engineering and Mathematical Sciences, City University, London, EC1V 0HB, UK (e-mail: justin.phillips.1@city.ac.uk)

S. H. Chang, K. Maney and R. M. Langford are with the Anaesthetic Laboratory, St Bartholomew's Hospital, London, EC1A 7BE, UK (e-mail: r.m.langford@btconnect.com).

materials are biomedically compatible, and allow steam sterilization.

B. Cranial access system

The Integra Neurosciences IM-3 Cranial Access System was used in conjunction with the fiber-optic probe, allowing the tips of the optical fibers to be placed directly within the tissue of the cerebral cortex. The IM-3 bolt has three lumens, one of which was used for intracranial pressure measurement and the remaining two for the fiber-optic probe. Figure 1 shows a diagram of the intra-cranial bolt containing the optical fibers in situ.

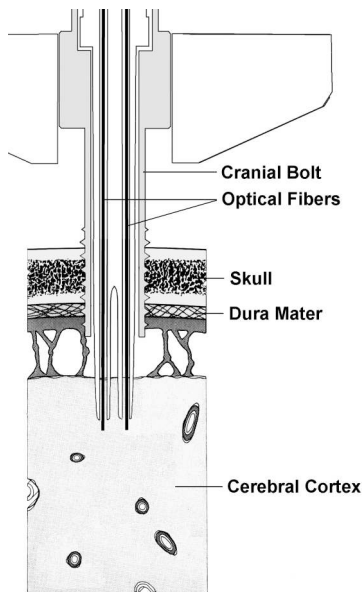


Fig. 1. Cranial bolt and fibers *in situ*. The inter-fiber separation was 2.0 mm.

C. Short duration measurements

This study was approved by the local Research Ethics Committee and patients' written informed consent was sought prior to the study. After induction of anesthesia, the patient was intubated and ventilated and the cranial bolt was inserted by the neurosurgeon. The fibers were then inserted via the bolt, approximately 5 mm into the brain and PPG signals recorded for four minutes. After the monitoring period, the fibers and the bolt were removed and the surgery resumed. To date, six patients aged between 46 and 70 requiring elective neurosurgery have been studied.

D. Short duration measurement

One patient was studied; a 78-year old female who presented to hospital with a Glasgow coma score (GCS) of 1. A CT scan revealed the presence of a frontal temporal intracerebral hematoma. The patient underwent a craniotomy to evacuate the hematoma. The patient was sedated with Propofol (an intravenous anesthetic) and stabilised according to established treatment protocols and bolts were placed by a

neurosurgical registrar and ICP monitoring, together with routine ITU monitoring commenced. The cranial bolt was inserted into the right frontal part of the skull. After obtaining assent, the optical fiber probe was introduced into the cranial bolt, so that the probe was penetrating the right frontal lobe of the cerebral cortex. Signals were recorded from the brain tissue using the cerebral oximeter for a period of six hours. The patient remained lightly sedated throughout the monitoring period and was mechanically ventilated throughout the monitoring period. After approximately six hours the patient was moving frequently, and appeared to be regaining consciousness so the optical fibers were removed from the bolt and monitoring stopped. The patient spent several days on the intensive care unit where she made a satisfactory recovery.

E. Estimation of oxygen saturation values

All the short-duration signals were analysed retrospectively using a 60-second sample of each waveform. This sample was chosen by visual inspection, selecting the 'best' signal in terms of overall quality of the waveform, where waveforms with large amplitude and minimal artifact were preferred. The arterial oxygen saturations were estimated from the cardiac-modulated components of the discrete Fourier transforms. These were taken from the same 60 second samples of each waveform, using a LabVIEW (National Instruments Inc. Austin, TX, USA) virtual instrument incorporating a fast Fourier transform algorithm.

The cardiac frequency f_{card} was derived from the discrete Fourier transform of the acquired signal. The amplitude of the pulse peaks in each amplitude spectrum were then calculated and normalised by dividing by the amplitude at zero Hz in each spectra. The cerebral arterial oxygen saturation, $ScaO_2$ was estimated using an empirically derived formula for estimating oxygen saturation in pulse oximetry [10]:

$$ScaO_2 = 110 - 25 \left(\frac{A_{card,R}/A_{DC,R}}{A_{card,IR}/A_{DC,IR}} \right) \quad (1)$$

where $A_{card,R}$ and $A_{card,IR}$ are the corresponding cardiac peak amplitudes and $A_{DC,IR}$ and $A_{DC,R}$ are the amplitudes of the infrared and red spectra at zero Hz. The long duration data was broken down into blocks of 60 seconds. A continuous estimation of the arterial and venous oxygen saturations was thus produced from successive data blocks using the same software algorithm used for the short duration measurements.

III. RESULTS

A. Short duration measurements

Signals were successfully obtained at both wavelengths for all six patients. To allow comparison of waveforms between patients, each waveform was divided by the total intensity

(i.e. the AC signal was divided by the AC+DC signal). Figure 2 shows an example waveform. The infrared and red PPG signals are of good quality and the dichrotic notch is clearly visible on both channels.

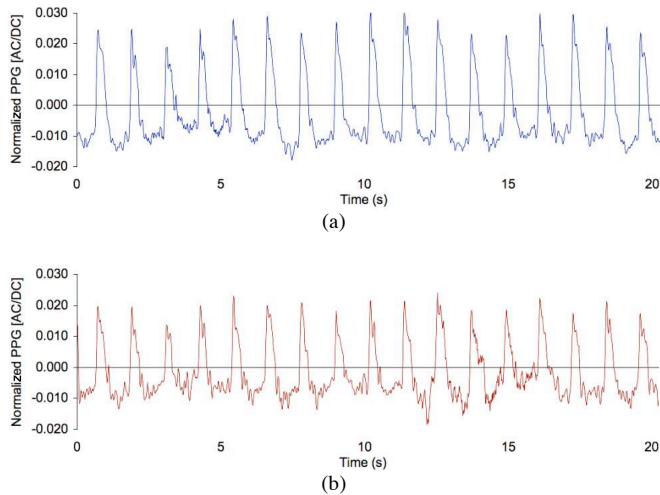


Fig. 2. Infrared (a) and red (b) normalised PPG signals recorded from Patient #6.

B. Estimation of oxygen saturation using frequency domain analysis

TABLE I
OXYGEN SATURATIONS ESTIMATED FROM SHORT DURATION MEASUREMENTS

| Patient # | Peak frequency f_{card} (Hz) | Ratio-of-ratios | ScaO ₂ (%) |
|-----------|--------------------------------|-----------------|-----------------------|
| 1 | 1.06 | 0.474 | 98.2 |
| 2 | 1.08 | 0.380 | 100.5 |
| 3 | 0.94 | 0.608 | 94.8 |
| 4 | 0.94 | 0.784 | 90.4 |
| 5 | 1.10 | 2.32 | 52.0 |
| 6 | 0.86 | 0.694 | 92.6 |

The acquired DC signals for all the neurosurgical patients were used to estimate the cerebral arterial oxygen saturation ScaO₂, using an FFT algorithm. The results are summarized in Table 1. The oxygen saturation values calculated for patient 5 are considerably lower than the results obtained for the other patients. The finger pulse oximeter reading indicated a value of 99% for the systemic arterial oxygen saturation in this patient. In the absence of any measurement errors, this result suggests that a high proportion of the blood in the pulsating vessels close to the probe contained blood of low oxygen saturation. This effect may be unusual, and may have arisen as a result of the patient's individual pathology. Alternatively the cerebral veins may be pulsatile in all patients but the effect is not apparent unless the probe is positioned in a region where the density of veins is particularly high. The incompressibility of the brain,

cerebro-spinal fluid and other cranial contents provides a likely mechanism for venous pulsation. Changes in cerebral arterial pressure during the cardiac cycle would cause the total volume of the cerebral arteries to increase. The intracranial pressure would increase momentarily, compressing the cerebral veins so the veins are partially emptied of blood, causing their diameter to decrease.

C. Long duration measurement

Signals were successfully recorded from the patient throughout the six-hour monitoring period. Table 2 shows the cardiac frequency, normalised infrared and red peak amplitudes, ratio-of-ratios and ScaO₂ calculated from the Fourier transforms obtained from the acquired data. The main peak occurring in each one-minute red and infrared spectrum between 0.8 and 1.6 Hz (i.e. in the cardiac frequency range) was identified by the VI and the values averaged for the entire measurement period.

TABLE II
MEAN (\pm SD) CEREBRAL ARTERIAL SATURATION AND OTHER VARIABLES ESTIMATED FROM LONG DURATION MEASUREMENT

| f_{card} (Hz) | Normalised peak amplitude /10 ⁻³ | | Oxygen saturation ScaO ₂ (%) |
|--------------------|---|-----------------------|---|
| | $A_{card,IR}/A_{DC,IR}$ | $A_{card,R}/A_{DC,R}$ | |
| 1.05 (\pm 0.12) | 1.63 (\pm 0.73) | 1.29 (\pm 0.74) | 90.54 (\pm 2.73) |

The arterial oxygen saturation (SpO₂) was manually recorded from the commercial finger pulse oximeter (GE Healthcare Clinical Systems, Wauwatosa, WI, USA) incorporated into the patient monitor and the arterial oxygen saturation (SaO₂) was measured from a blood sample using a hemoximeter (IL682 CO-oximeter, Instrumentation Laboratory Inc., Lexington, MA, USA). A total of 54 pulse oximetry measurements and 5 hemoximeter measurements were recorded during the six hour period. The ScaO₂ was significantly lower than the finger SpO₂ ($P < 0.001$) and the blood SaO₂ ($P < 0.001$) as confirmed by paired Student's t -tests. A steady trend of decreasing ScaO₂ was seen for the first four hours followed by a gradual rise. The mean (\pm SD) difference between ScaO₂ and finger SpO₂ (in saturation units) was $-7.47(\pm 3.4)\%$; $n=54$. The mean (\pm SD) difference between ScaO₂ and blood SaO₂ was $-7.37(\pm 2.8)\%$; $n=5$.

IV. DISCUSSION

It was shown that satisfactory quality red and near-infrared PPG signals can be obtained directly from human brain tissue using a fiber optic probe. The proximity of a large vein to the probe tip seems a likely explanation for the extremely low cerebral arterial oxygen saturation reported for Patient 5. Unfortunately the exact position of large

cerebral blood vessels is not known prior to drilling of the burr hole and placement of the bolt. Even after the burr hole is made, the situation does not really improve as the dura mater covers the cerebral surface and the large vessels running over it. If venous pulsation occurs in all cerebral tissue, it would complicate the process of estimating arterial oxygen saturation using the principle of pulse oximetry. The PPG signal, assumed to arise solely from pulsating arterial blood would be 'contaminated' by the venous pulse. Until more measurements are made, the extent of this potential problem is difficult to quantify.

For the long-duration measurement, it is not clear why the oxygen saturation measured in the frequency domain (ScaO₂) was significantly lower than the finger SpO₂ and the blood SaO₂: mean difference (ScaO₂ - SpO₂) = -7.81(±2.9)%, (ScaO₂ - SaO₂) = -7.37(±2.8)%. Although the SpO₂ and the ScaO₂ were measured at different sites, no significant difference in these values was expected. One suggestion was that a component of the cardiac pulsation 'seen' by the probe was due to veins rather than arteries as was suggested to explain the extremely low oxygen saturation recorded in Neurosurgical Patient #5. Despite some unexpected results, it was felt that the results of this study were encouraging and interesting and warranted further evaluation of this technique to establish whether good quality signals can be maintained for a clinically relevant monitoring period in other patients, and to compare oxygen saturation values obtained using this method with those from other observations such as NIRS.

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