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INVESTIGATION OF OXYGEN SATURATION DERIVED FROM CARDIAC PULSATIONS MEASURED ON THE ADULT HEAD USING NIR SPECTROSCOPY

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1. INTRODUCTION

Cardiac related pulsatile signals can be detected in different parts of the human body, including the finger, ear lobe and forehead [1] by using near infrared (NIR) monitoring. These pulsatile signals are due to attenuation of light by the increase of arterial blood volume during systole in the cardiac cycle. Pulse oximetry exploits these pulsatile signals to calculate oxygen saturation (S_pO_2) [2]. There are two types of pulse oximetry: (1) the transmission type where the light source and detector are facing each other across the measurement site (e.g. ear lobe or finger), and (2) the reflectance type where both the light source and detector are in the same plane (e.g. forehead or forearm) [1] with the source detector spacing typically less than 1 cm. With more sensitive optical instruments, it has been shown that these pulsatile signals can be measured on the forehead at a greater spacing using either a CCD spectrometer [3] or a phase resolved system [4]. Both of these two methods can be considered as operating in reflectance mode with large source detector spacings of 3 or 3.5 cm. These pulsatile signals were also thought to be mainly caused by the change in arterial blood volume. However, at source detector spacing larger than ~2 cm, NIR light can penetrate through the skull into the brain and the measured pulsatile signals are likely partly to include components caused by brain movement [5] as well as arterial blood pulsations in the scalp and the brain. The objective of this paper is to compare three algorithms used to calculate oxygen saturation from the head pulsatile signals, (signified by SpO_2^h to distinguish it from SpO_2 measured at other sites) with large source detector spacing. Two of the algorithms implicitly allow the possibility of venous blood contributing to SpO_2^h. We will show the SpO_2^h calculated by three algorithms in 8 adult subjects during normoxia and hypoxia. Examples of phase differences between the oxy and deoxy-haemoglobin (ΔHbO_2 and ΔHHb) signals, which could imply a venous contribution, will also be presented.

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2. METHODS

2.1 Experimental Study

Eight adult subjects (mean age 31 ± 3 years) participated in this study which was approved by the UCL Hospital Ethics Committee. An optical probe with a source detector spacing of 3.5 cm was placed on the left side of the foreheads of subjects. The light source was provided by a tungsten halogen lamp (Model 77501, Oriel Instruments) via an optical fibre bundle. The transmitted light was collected by another fibre bundle linked to an imaging spectrograph (SPEX 270M, JY Optical Systems Instruments SA, Inc.) which dispersed the light on to a cooled CCD detector (Wright Instruments). Intensity spectra were collected between 670 and 990 nm with a spectral resolution of 5 nm and exposure time of 50 ms. A pulse oximeter probe (Novametrix 500) operating in beat-to-beat mode was attached to subjects' ear lobes to monitor SpO_2. In the first part of the experiment when the subjects were resting, they breathed room air through a face mask. In the second part of the experiment, the fraction of inspired oxygen (FiO_2) was reduced from 21% to 10-15% such that SpO_2 fell from 98% to 90%. At this point the subjects were given 100% oxygen for 5 breaths which caused SpO_2 to rapidly return to 98-100%, then they returned to air breathing. The same manoeuvre was repeated three times.

2.2 The Three Algorithms to Calculate SpO_2^h

The intensity spectra were converted to attenuation spectra with respect to a reference spectrum which was the average of 200 spectra (10 seconds) and further smoothed by a 3rd order Savitsky-Golay filter. The attenuation spectra calculated in this way can be interpreted as changes in attenuation $\Delta A(\lambda)$ from a nominal baseline and were used in the following analyses. For the conversion from $\Delta A(\lambda)$ to ΔHbO_2 and ΔHHb, the wavelength range 746 – 906 nm was used. The wavelength dependence of the differential pathlength was taken into account. Of the three methods described below, method B and C were developed by the authors.

Method A A flow diagram for this method is shown in Fig.1(a). This method is that used by [3,4] who calculated SpO_2^h using data from a CCD multi-wavelength spectrometer and a phase resolved system, respectively. At each wavelength λ_i, $\Delta A(\lambda_i,t_j)$ a block of 256 samples (12.8 s) was Fourier transformed (FT) and the subsequent FTs were carried out at 1 s time intervals. For each FT spectrum, the spectral peak around the heart rate frequency (~ 1 Hz) and its 1st harmonic were identified and the sum of their magnitudes, which was taken as the energy of the cardiac pulsations, over a bandwidth of 0.8 Hz was calculated. With the sum of magnitudes calculated at all wavelengths, an attenuation spectrum for the cardiac pulsations was thus obtained, i.e. $\Delta A_p(\lambda)$ which was then converted to ΔHbO_2 and ΔHHb by least square fitting to the specific extinction coefficient spectra. The SpO_2^h was approximated by:

$$SpO_2^h = \frac{\Delta HbO_2}{\Delta HbO_2 + \Delta HHb} \times 100\% \quad (1)$$

The SpO_2^h calculated is therefore a running average over 12.8 s (256 samples).