

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

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ΔHHb) signals, which could imply a venous contribution, will also be presented.

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:

$$\text{SpO}_2^h = \frac{\Delta\text{HbO}_2}{\Delta\text{HbO}_2 + \Delta\text{HHb}} \times 100\% \quad (1)$$

The SpO₂^h calculated is therefore a running average over 12.8 s (256 samples).

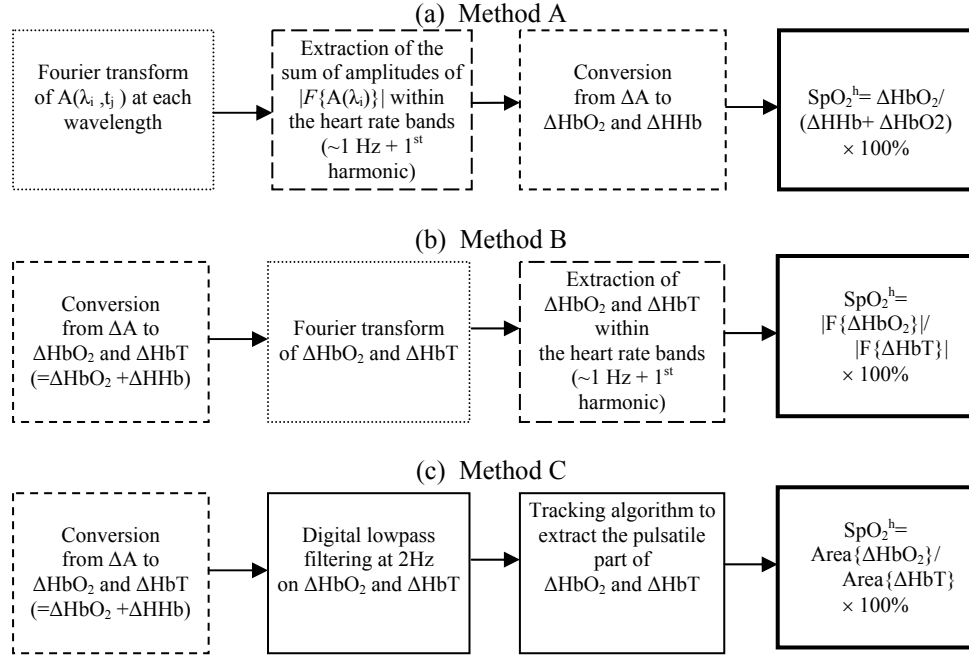


Figure 1. The three methods implemented to calculate SpO₂^h

Method B As shown in Fig.1(b), the basic elements are the same as method A but in a different sequence. For each $\Delta A(\lambda, t_j)$ spectrum at t_j , one value of ΔHbO_2 and ΔHHb were obtained respectively by least square fitting. A FT was then performed on 256 samples of ΔHbO_2 and ΔHbT ($= \Delta HbO_2 + \Delta HHb$) separately with the time interval between subsequent FTs of 1 s. The corresponding spectral peaks around the heart rate frequency were identified as well as its first harmonic. The sums of the magnitudes around the spectral peaks were then calculated which were regarded as the energy of the cardiac pulsations in ΔHbO_2 and ΔHbT . The SpO₂^h was calculated as

$$SpO_2^h = \frac{\sum_i |F\{\Delta HbO_2\}|_i}{\sum_i |F\{\Delta HbT\}|_i} \times 100\% \quad (2)$$

where $F\{\}$ signifies the FT and i includes the indexes of the frequency bins within the heart rate bandwidth and that of its 1st harmonic. As in Method A, the calculated SpO₂^h is a running average over 12.8 s.

Method C As depicted in Fig.1(c), each $\Delta A(\lambda, t_j)$ spectrum at t_j was first converted to one value of ΔHbO_2 and ΔHHb , respectively. Subsequently, a digital lowpass filter (5th order Butterworth with a cut-off frequency of 2Hz) was used to remove high frequency noise from the ΔHbO_2 and ΔHbT signals. A tracking algorithm based on trough detection and cubic spline fitting was implemented to track the baseline movement and to isolate

the pulsatile component of the ΔHbO_2 and ΔHbT signals. The SpO_2^h was then calculated on a beat-to-beat basis from the areas under each cardiac pulsation in ΔHbO_2 and ΔHbT :

$$\text{SpO}_2^h = \frac{\text{Area}\{\Delta\text{HbO}_2\}}{\text{Area}\{\Delta\text{HbT}\}} \times 100\% \quad (3)$$

4. RESULTS

4.1 Normoxia

During normoxia, 60 seconds worth of data were analysed for each subject. The individual means \pm standard deviations of SpO_2^h for all 8 subjects calculated by methods A, B and C are summarised in Fig.2 alongside the group means \pm standard deviations among the mean SpO_2^h in 8 subjects, i.e. $90.0 \pm 5.8\%$ (method A), $100.0 \pm 9.1\%$ (method B) and $94.0 \pm 9.5\%$ (method C). While the means of method A and B, and those of method B and C are statistically different ($p < 0.05$), those of method A and C are not. The SpO_2 measured by the pulse oximeter was 98% for all subjects.

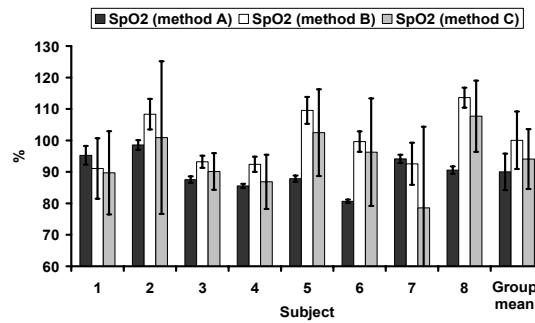


Figure 2. Individual and group means \pm standard deviations of SpO_2^h calculated by methods A, B and C

4.2 Hypoxia

An example of SpO_2^h calculated by methods A, B and C, together with SpO_2 measured by the pulse oximeter as FiO_2 was changed is shown in Fig.3. The correlations between SpO_2 measured by the pulse oximeter and SpO_2^h calculated by method A, B and C during approximately 700 seconds of FiO_2 variations are summarised in Table 1. For comparison purpose, SpO_2^h calculated by method C and SpO_2 measured by the pulse oximeter have been averaged for 12.8s (same as methods A and B).

Table 1 Correlation coefficient (r) between SpO_2 measured by the pulse oximeter (P.O.) and SpO_2^h calculated by methods A, B and C (\checkmark for $p < 0.005$)

Subject	r for Method A ($p < 0.005$)	r for Method B ($p < 0.005$)	r for Method C ($p < 0.005$)
1	0.42 (\checkmark)	0.31 (\checkmark)	0.66 (\checkmark)
2	0.67 (\checkmark)	0.58 (\checkmark)	0.73 (\checkmark)
3	0.49 (\checkmark)	0.37 (\checkmark)	0.66 (\checkmark)
4	0.18 (\checkmark)	0.45 (\checkmark)	0.68 (\checkmark)
5	0.35 (\checkmark)	0.45 (\checkmark)	0.50 (\checkmark)
6	0.56 (\checkmark)	0.31 (\checkmark)	0.48 (\checkmark)
7	-0.08 (\times)	0.05 (\times)	0.20 (\times)
8	0.29 (\checkmark)	0.64 (\checkmark)	0.76 (\checkmark)

5. DISCUSSION AND CONCLUSIONS

Method A involves performing FTs and taking the magnitudes of $\Delta A(\lambda)$ at each wavelength λ as the first procedure. Since magnitudes contain no information about the phase at each signal, method A intrinsically ignores any phase difference between ΔHbO_2 and ΔHHb , i.e. it assumes that both ΔHbO_2 and ΔHHb are in phase. This assumption is reasonable when the pulsatile signals are solely due to a change in arterial blood volume (a central assumption of the work carried out in [3,4]). The final values of ΔHbO_2 and ΔHHb are both mainly positive leading in most cases to $\text{SpO}_2^{\text{h}} < 100\%$. Each $\Delta A(\lambda, t_j)$ spectrum is subject to certain instrumental noise and using 256 $\Delta A(\lambda, t_j)$ spectra to calculate one value of ΔHbO_2 and ΔHHb improves the signal-to-noise ratio (SNR). This is shown in Fig.2 where SpO_2^{h} calculated by method A has the smallest inter and intra subject standard deviation of the three methods. Method B converts $\Delta A(\lambda)$ to ΔHbO_2 and ΔHHb as a first step and hence preserves the phase difference between ΔHbO_2 and ΔHHb . Since each value of ΔHbO_2 and ΔHHb is calculated from only one $\Delta A(\lambda)$ spectrum, the results are more noisy than those of method A. Each SpO_2^{h} is calculated from the FT of 256 samples of ΔHbO_2 and ΔHHb and thus can be considered as rolling average of 12.8 s. Method C also preserves the phase difference between the ΔHbO_2 and ΔHHb signals but suffers the same SNR problem as method B due to the use of only one $\Delta A(\lambda)$ spectrum for the conversion. Method C calculates SpO_2^{h} on a beat-to-beat basis but the algorithm may introduce certain errors while isolating the pulsatile part of ΔHbO_2 and ΔHHb . These errors contribute to the high inter and intra subject variation in the SpO_2^{h} values calculated by this method (Fig 2). With a rolling average of 12.8s, however, the resulting SpO_2^{h} have, in most cases, the highest correlation with SpO_2 from the pulse oximeter. From the results of the normoxia and hypoxia studies, method A seems to offer the most reasonable results; SpO_2^{h} has the lowest standard deviation and is generally less than 100% which fits with the idea of SpO_2^{h} being a ratio defined as $\Delta \text{HbO}_2 / \Delta \text{HbT}$ and $\Delta \text{HbT} > \Delta \text{HbO}_2$. Using method C for beat-to-beat analysis however, we found several instances where ΔHbO_2 and ΔHHb were out of phase and occasionally going in opposite directions (180° out of phase) during a cardiac cycle as shown in Fig.4(b). As a result, the ΔHbT is smaller than ΔHbO_2 and hence $\text{SpO}_2^{\text{h}} > 100\%$. This also happens in Method B which preserves phase change. This kind of phase change is unlikely to be caused by a change in arterial blood volume. Arterial HHb cannot decrease by more than 2% in a cardiac cycle (normal SpO_2 varies between 98 and 100%) and yet we see a larger drop (14% drop in ΔHHb with respect to ΔHbO_2) from Fig.4(b). Venous ΔHHb , on the other hand, can decrease noticeably during a cardiac cycle. Two possible mechanisms for the observed changes are: (1) the arterial to venous volume ratio increases during a cardiac cycle because the arterial compartment expands and presses against the venous compartment during systole, (2) the small rise in cerebral blood flow during systole increases the proportion of ΔHbO_2 (and hence reduces the proportion of ΔHHb) in the venous compartment. During normoxia the mean SpO_2^{h} calculated by methods A, B and C were different from the arterial SpO_2 measured by the pulse oximeter. Also, during hypoxia, SpO_2^{h} calculated by methods A and B were only partially correlated to the arterial SpO_2 as shown in Table 1 suggesting that the arterial contribution to SpO_2^{h} is only partial. To conclude, method A is only suitable if one is prepared to assume that the pulsatile signals from the head are totally arterial. Otherwise, other algorithms which preserve phase changes between ΔHbO_2 and ΔHHb , such as methods B and C should be used.

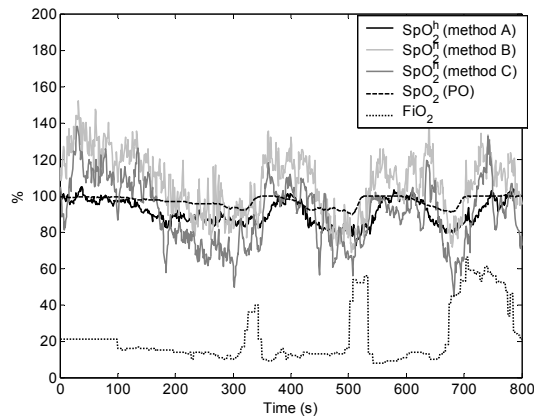


Figure 3. Changing FiO_2 : examples of SpO_2^h calculated by methods A, B & C, and SpO_2 measured by the pulse oximeter

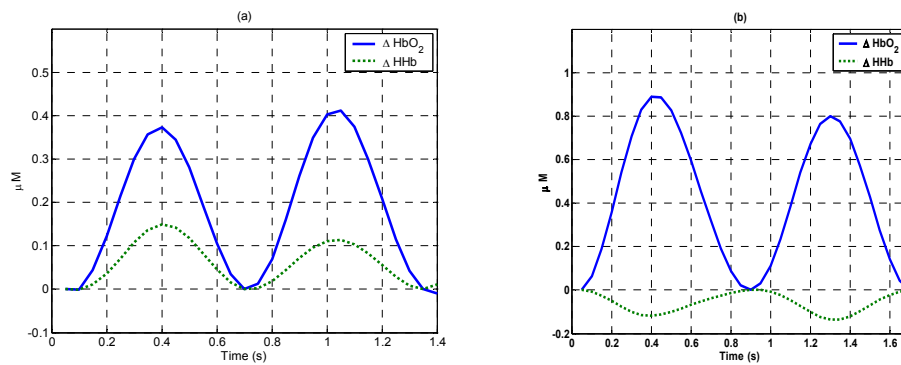


Figure 4. Examples of ΔHbO_2 and ΔHb during a cardiac cycle: (a) both ΔHbO_2 and ΔHb are positive, (b) ΔHbO_2 is positive but ΔHb is negative

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