Low Oxygen Saturation Quantification in Human Arterial and Venous Circulation

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Abstract— Conventional pulse oximetry has limited accuracy in measuring blood oxygen saturation in low saturation and perfusion scenarios. This limits the application of pulse oximetry in patients suffering from peripheral vascular afflictions. We present a novel pulse oximetry system which proposes solutions to these low saturation and perfusion scenarios, by inducing an artificial pulse in the detected photoplethysmograph. A novel arterio-venous hypothesis was formulated to extract arterial and venous saturation data from the artificial photoplethysmograph using arterial-to-venous compliance ratios. Sensor wavelengths were selected to provide high and low saturation accuracy, followed by an in vitro sensor calibration procedure. System performance was validated by means of an in vivo procedure. In vivo results indicate good accuracy for high saturation, with limited accuracy in low saturation scenarios. The arterio-venous hypothesis was validated, indicating that venous saturation can be extracted from the artificial photoplethysmograph. The results indicate that the proposed system might be able to accurately monitor arterial and venous saturation in low or no perfusion scenarios. It is recommended that further studies into the system’s performance are conducted.

Index Terms— Pulse oximetry, low saturation, low perfusion, venous saturation, photon diffusion theory, in vitro calibration.

I. INTRODUCTION

Continuous, non-invasive and reliable blood oxygen saturation information can play a vital role in the choice of medical intervention in treating patients suffering from peripheral vascular afflictions. In treating patients suffering from diseases such as meningococccemia and diabetes mellitus, medical intervention often includes the surgical removal of tissue affected by advanced hypoxia as a result of ischemia [1].

The decision to excise tissue and the severity of the excision are currently based on an intermittent and qualitative evaluation by the medical practitioner and not on a continuous and quantitative measurement of the oxygen supply. Although a continuous saturation recording cannot serve as the sole parameter on which excision is based, it will often enable a more informed decision by the medical practitioner.

The current benchmark for the continuous and non-invasive monitoring of arterial saturation ($S_aO_2$) is pulse oximetry. Typical commercial pulse oximeter sensors consist of two high-output light emitting diodes (LEDs) and a highly sensitive photodetector. The LEDs emit red and infrared light into a pulsating vascular bed, while the photodetector detects the light intensity after interaction with the tissue components. Light intensities detected by the photodetector take the form of a photoplethysmographic (PPG) signal. The PPG results from the time-varying amount of blood that is injected into the vascular bed during systole and diastole. The determination oxygen saturation using pulse oximetry ($S_pO_2$) is based upon a modulation ratio $R$, which is highly dependent on the relative amplitudes of the AC components in the red and infrared PPGs and relatively independent of the static components in the PPGs, caused by static light absorption and scattering by bloodless tissue, residual blood and bone. Arterial oxygen saturation using pulse oximetry ($S_aO_2$) is related to $R$ by means of empirical calibration curves, obtained for commercial pulse oximeters by way of in vivo desaturation procedures [2].

The application of conventional pulse oximetry principles in monitoring peripheral vascular afflictions is however, limited. Although accurate for $S_aO_2$ values in excess of 70-80%, studies have shown a marked decrease in $S_pO_2$ values for $S_aO_2$ values below 70–80% [3-6]. In this paper we follow the method proposed by Bland and Altman [7] to describe pulse oximeter accuracy in the form of a bias error and standard deviation (SD). Razi and Hossein [5] and Bickler et al. [6] conducted studies demonstrating an increase in pulse oximeter bias and SD for $S_aO_2 < 75-80%$. Additional to low $S_aO_2$ inaccuracy, commercial pulse oximeters have also demonstrated questionable performance in low perfusion scenarios. As a result of the dependence of the calculation of $R$ on the AC component in the PPG, the performance of older pulse oximeter models has shown significant deterioration in the event of very small AC components [8,9]. The state-of-the-art adaptive filtering technology for PPG processing,
introduced by the Masimo Corporation, has significantly improved pulse oximeter performance in low perfusion scenarios [10].

Pulse oximetry principally depends on the presence of a pulsating arterial component in the PPG. This pulsating component is often missing in ischemic tissue. As conventional pulse oximeters are rendered unusable by these ischemic conditions and only measures $S_O^2$, there is a need to develop an alternative system capable of continuously and non-invasively monitoring both $S_O^2$ and venous saturation ($S_{pv}^2$). $S_O^2$ may be useful for assessing the status of tissue affected by recent or extended occurrences of ischemia, with similar $S_O^2$ and $S_{pv}^2$ indicating a lack of tissue oxygen consumption resulting from extended tissue damage, or arterio-venous shunting. Although Chan and Smith [11] addressed the absence of an alternating current (AC) pattern by inducing an artificial AC pattern in the PPG, a system specifically designed for $S_{pa}^2$ and $S_{pv}^2$ measurement accuracy in low saturation and no severe hypoperfusion scenarios is still needed.

This paper presents the development and testing of a modified pulse oximetry system to non-invasively monitor $S_{pa}^2$ and $S_{pv}^2$ in low saturation and perfusion scenarios, to address the needs described above. The selection of the sensor hardware to facilitate low saturation measurement is discussed, followed by a design overview of an artificial pulse generator for low perfusion applications. Thereafter, a system calibration by means of an in vitro calibration procedure to facilitate low saturation accuracy is presented. An in vivo validation procedure and results are then presented to validate system performance in differing physiological and saturation scenarios. Throughout the calibration and validation procedures, results were tested against a photon diffusion theory model provided by Schmitt [12], which was modified to incorporate the effect of venous pulsations. The modified photo diffusion model is presented in Appendix A.

II. ARTIFICIAL PULSE OXIMETER

The artificial pulse oximeter (APO), shown in Fig. 1(a), operates on the basis of generating a clearly detectable AC component in the PPG by means of a peristaltic action that is generated pneumatically. Residual blood in the tissue under consideration (in this case an extended index finger), is artificially pulsed into the vascular bed of the finger tip by inflating three pressure cuffs sequentially around the proximal segment of the finger.

In conventional pulse oximetry, AC behavior in the PPG is assumed to be the result of arterial pulsating behaviour only [13]. In the case of the APO however, pulsating behaviour is induced in both arterial and venous circulation. The modulation ratio $R$ calculated from the AC component in the PPG would thus be dependent on both $S_{pa}^2$ and $S_{pv}^2$. For the APO, this effect is taken into account by considering the saturation value calculated from the APO’s $R$ as a linear combination of $S_{pa}^2$ and $S_{pv}^2$. This relationship can be summarized as follows:

$$SO^2 = PS_{pa}^2 + (1-P)S_{pv}^2$$  \(1\)

with $SO^2$ the original APO saturation value, and $P$ the arterial pulse volume fraction relating artificial pulse amplitudes in arterial and venous circulation. $P$ is defined by the arterial volume fraction, also called the arterial-to-venous compliance ratio (i.e. if the compliance ratio is $1:1$, $P = 1/2$, and if the compliance ratio is $1:5$, $P = 1/6$). If $S_{pv}^2$ can be determined from a symmetrically similar, but healthy, measuring site, (1) can be solved for $S_{pa}^2$. The application of an arterial-to-venous compliance ratio is a novel approach in quantifying the combined effects of $S_{pa}^2$ and $S_{pv}^2$ on a PPG. We term this the “arterio-venous hypothesis”. In designing the APO, two important aspects had to be addressed:

- accuracy at high and low saturation, and
- the generation of clearly detectable AC behaviour in the PPG.

High and low saturation accuracy was addressed by selecting LED wavelengths ideally suited for a specified saturation range, while the generation of an artificial pulse entailed the development of an artificial pulse generator (APG).

The sensitivity and accuracy of pulse oximeters are highly dependent on the combination of LED wavelengths used in the sensors. In commercial pulse oximeters, LED wavelengths are selected to maximize sensor sensitivity. Unfortunately, this wavelength selection is only suited for saturation values in excess of 70%. To counter the amplified scattering effects at

![Fig. 1. a) APO overview and b) artificial pulse generator](image-url)
low saturation, our LED wavelength selection for low saturation was based on a study conducted by Mannheimer et al. [14], where LED selection was based on an attempt to match scattering perturbations resulting from differing physiological parameters. Mannheimer et al. [14] suggested the use of a 735/890 nm LED pair, but due to availability of identical LEDs, a 740/880 nm LED pair was used in the design of the low saturation sensor presented here.

Consequently two pairs of LEDs were used in the APO, namely a conventional 660/910 nm LED pair for high saturation measurement and a 740/880 nm LED pair for low saturation measurement. Both LED pairs used the PDB-C171SM photodetector from Advanced Photonics Inc., which has an almost linear responsivity ranging from 0.33 A/W – 0.55 A/W in the 660 – 910 nm wavelength range.

The APG is a computer-controlled system capable of inducing a clearly detectable artificial AC component in the finger. Two APG has two subsystems, namely the pressure cuff and compressor.

The pressure cuff consists of a set of three separately inflatable and expandable tubes, contained in a non-stretch cloth enclosure. The cuff is wrapped around the finger and fixed at a comfortable diameter using Velcro. The cuff setup is similar to a conventional blood pressure cuff, both in design and operation. By inflating the tubes of the pressure cuff in a sequence starting proximally and progressing distally on the finger, residual blood is pushed forward into the fingertip, creating light absorption modulation and thus an AC component in the PPG.

The compressor, as seen in Fig. 1(b), consists of a pressure controlled pneumatic pump, a reservoir and a PC-controlled 5/8 valve bank. Two small compressors had to be used in order to have enough pneumatic capacity to drive the system. System pressure is regulated at 600 mmHg by means of a PC-independent electronic pressure sensor. This relatively high pressure was selected to ensure that pulsating behaviour is induced in both the arterial and venous circulation.

III. CALIBRATION

Most modern pulse oximeters rely on a calibration procedure to obtain \( R \) values for specific \( S_pO_2 \) values. These calibration curves can be formulated by means of empirical or theoretical procedures, with most pulse oximeter manufacturers resorting to an in vivo empirical calibration approach for optimized accuracy [2]. We followed a theoretical (using the modified photo diffusion model presented in Appendix A), and an empirical approach.

A. Theoretical calibration

Theoretical arterial calibration curves for the 660/910 nm and 740/880 nm sensors can be seen in Fig. 2, where it is clear that the 660/910 nm calibration curve has a steeper slope than that of the 740/880 nm curve. A steeper slope indicates that \( R \) is more sensitive to a change in the light intensity and for this reason most manufacturers choose the 660/910 nm LED combination for conventional pulse oximeters.

B. Empirical calibration

An in vitro empirical calibration approach was followed to calibrate the APO, due to the need for low saturation calibration and the ethical barriers that prevent human in vivo calibration procedures at low saturations [14].

A calibration setup similar to the one presented by Edrich et al. [15] was used for the APO’s calibration, depicted in Fig. 3(a). Two separate, closed systems, representing the arterial and venous circulation, were incorporated into the setup to enable the independent control of \( S_pO_2 \) and \( S_vO_2 \). A Lilliput infant oxygenator was used to saturate or desaturate the blood using either a 100% \( O_2 \) or a 95% \( N_2 \) and 5% \( CO_2 \) mixture. Homogenized 2% low fat milk was used as a diffuser to simulate the scattering properties of bloodless tissue, skin and bone [15].

A tri-chamber stainless steel cuvette, based on the dual-chamber cuvette designed by Edrich et al. [15], was used to provide separate chambers for the arterial and venous circulation and the diffuser. Blood and diffuser chamber dimensions were 0.3 mm x 20 mm x 44 mm and 4 mm x 20 mm x 44 mm respectively, to simulate a blood-tissue volume percentage of 10%. The chambers were separated by flexible membranes that could deform outwardly if pressure was applied to the chambers [16]. A cross-sectional view of the cuvette can be seen in Fig. 3(b).

The overall goal of the calibrating procedure was to obtain a calibration curve, where the APO’s output ratio \( R \) could be correlated with blood oxygen saturation values. This process entailed measuring \( R \) whilst systematically desaturating fully saturated human blood to saturation values lower than 20%. Recently expired whole human blood from the blood repository at Tygerberg Academic Hospital was used as the principal absorber during the calibration test.

The calibration procedure was divided into two stages: Firstly, calibration of the 660/910 nm and 740/880 nm sensors for conventional pulse oximetry applications in low and high saturation scenarios and secondly, empirical testing of the arterio-venous hypothesis.
For the normal empirical calibration procedure, only the arterial circuit in the calibration setup was used, with the venous circuit inactive. The blood was firstly fully saturated using 100% O₂ and then stepwise desaturated using a 95% N₂ and 5% CO₂ mixture. An external blood gas analysis of SₐO₂ values was conducted at each step by means of a Bayer Rapidlab 865 blood gas analyzer. This system is a co-oximetry system and values measured with the blood gas analyzer will thus be indicated as co-oximetry saturation (SₐO₂). These SₐO₂ values served as the reference saturation values to build the calibration curve in conjunction with the \( R \) values calculated at each desaturation step.

The calibration setup was also used to conduct in vitro empirical tests on the validity of the arterio-venous hypothesis. Both arterial and venous circuits were used during this procedure. The arterial and venous blood was firstly fully saturated using the above-mentioned approach. A three-way valve assembly was then used to isolate the arterial circuit from the oxygenator, after which venous blood was stepwisely desaturated. \( SₐO₂ \) would thus remain fairly constant, while \( SₐO₂ \) decreased, providing the differing saturation values needed to validate the arterio-venous hypothesis. Reference \( SₐO₂ \) and \( SₐO₂ \) values were taken at each desaturation step to validate the \( SₐO₂ \) and \( SₐO₂ \) values calculated by means of (1).

Calibration curves were obtained for the 660/910 nm and 740/880 nm sensors in the 20–100% saturation range. These curves were obtained by fitting the data collected during calibration with quadratic polynomials based on commercial calibration curves [17]. Calibration data and the corresponding quadratic fits for both sensors can be seen in Fig. 4, while the hematocrit was maintained at 38%.

Unfortunately the in vitro validation of the arterio-venous hypothesis did not deliver repeatable results (eight calibration trials were conducted). This was likely due to the complexity of controlling simulated physiological parameters and their interaction in the calibration setup. Due to these contributing factors, more importance was placed on an in vitro approach to prove the arterio-venous hypothesis, which is discussed in the following section.

IV. SYSTEM VALIDATION

The system validation entailed collecting and analyzing PPG data from volunteers and using this data in correlation with the calibration curves obtained from the in vitro empirical calibration to calculate either \( SₐO₂ \) or \( SₐO₂ \). These calculated values were compared with reference \( SₐO₂ \) and \( SₐO₂ \) values obtained using the Bayer Rapidlab 865 blood gas analyzer to analyze blood samples obtained from the volunteers.

The study population was a group of 12 healthy volunteers, recruited from the Stellenbosch University campus. Ethical approval for the study was granted by the Committee for Human Research at Stellenbosch University. Each volunteer had to give written informed consent and had to have blood parameters within set limits to participate in the study.

A high saturation pulse oximetry reading with a naturally induced PPG was firstly taken on the index finger of the volunteer’s left hand using the 660/910 nm sensor. A capillary blood sample was then taken from the middle finger on the volunteer’s left hand and analyzed for the reference \( SₐO₂ \). A blood pressure cuff was then inflated around the left forearm of the volunteer to occlude the hand under consideration. This was done to eliminate all pulsating behaviour in the finger vascular bed to ensure that cardiac-induced AC components did not contaminate the artificial PPG. Immediately after that, the APO was used on the occluded index finger of the volunteer’s left hand. A venous blood sample was taken from a vein on top of the volunteer's left hand to analyze for the reference \( SₐO₂ \). Using the initial \( SₐO₂ \) calculated during the high saturation validation, \( SₐO₂ \) could thus be calculated from (1). The blood pressure cuff was kept in an inflated state for a period of approximately five minutes to allow the saturation values in the occluded tissue to decrease to a suitably low value. This was done to simulate the low saturation and no perfusion scenario of tissue suffering from ischemia resulting.
from peripheral vascular afflictions. The APO was again used on the occluded left-hand index finger with two separate measurements taken with the 660/910 nm and 740/880 nm sensors. A capillary blood sample was taken from middle finger of the occluded left hand to determine the reference $S_{ca}O_2$.

The initial validation stage was the high saturation validation of the 660/910 nm sensor. A graphical representation of empirical and theoretical performance can be seen in Figs. 5(a) and 5(b). In Fig. 5(a), the different symbols indicate data points collected for different volunteers (this notation will be used in all subsequent figures of similar format). $S_{pa}O_2$ values calculated by means of the theoretical and empirical calibration curves, where volunteer-specific physiological parameters were used for the theoretical development, demonstrated bias and SD values of -6.2±4.35 and -4.8±3.86. The empirical calibration curve thus demonstrated superior performance to the theoretical calibration curve. As this was the case for all the validation stages, only graphical illustrations of empirical performance will be provided for the next stages.

The arterio-venous validation stage entailed the validation of the arterio-venous hypothesis with the 660/910 nm sensor. During this stage, $S_{pv}O_2$ values calculated using (1) was compared to reference $S_{cv}O_2$ values. A graphical illustration of arterio-venous performance can be seen in Fig. 6. Bias and precision values calculated for the theoretical and empirical arterio-venous calibration curves were -23.3 ±19.45 and 11.07±18.05, respectively. The empirical development again outperformed the theoretical.

The low saturation validation phase entailed the low saturation validation of 660/910 nm and 740/880 nm sensor accuracy. Low $S_{pa}O_2$ values calculated were compared to low $S_{ca}O_2$ reference values. Graphical illustrations of the low saturation performance of the 660/910 nm and 740/880 nm sensors can be seen in Figs. 7(a), 7(b) and 7(c). For the 660/910 nm sensor, theoretical and empirical bias and precision were -14.5 ±23.07 and -5 ±24.75. The 740/880 nm sensor's results demonstrated theoretical and empirical bias and precision of -34.4 ±18.4 and -13.6 ±21.38. Empirical calibration again outperformed the theoretical development for both sensors.

V. DISCUSSION

In pulse oximetry, the accuracy of any $S_{p}O_2$ measurement is dependent on the accuracy of the pulse oximeter's calibration curve. This is the approach followed by most pulse oximeter manufacturers and the reason behind using an in vivo calibration procedure. In this study however, this approach was not possible, with an in vitro approach being followed instead. This in vitro approach also has its shortcomings however, with the mismatch of optical properties, as explained by Mannheimer et al. [14], being a major contributor to errors. A comparison of theoretical and empirical calibration curves for the 660/910 nm and 740/880 nm sensors can be seen in Figs. 8(a) and 8(b). Fig. 8(a) shows theoretical calibration curves developed using physiological parameters specific to the calibration setup and volunteers three and seven. These volunteers were selected as illustration due to their blood parameters being notably different from that of the calibration setup. It is evident from Fig. 8(a) that the in vitro calibration curve and the theoretical calibration curve calculated by using the physiological properties of the in vitro calibration, is remarkably similar. In adapting the model for a finger
diameter of 10 mm, the calculated theoretical curve did not deviate significantly in the saturation range 80–100%. The calibration curves presented by Schmitt [12] also showed good correlation with in vivo commercial calibration curves. It can thus be concluded that the in vitro calibration curve is a relatively good substitute for an in vivo calibration curve in the high saturation range, verified by the low bias and SD of the $S_pO_2$ results presented in Fig. 5(a). It has to be noted however, that the bias in $S_pO_2$ using the in vitro calibration curve is -4.8%, which is more than the ±1-2% for conventional in vivo curves. The errors in $S_pO_2$ values presented in Fig. 5(a) are most likely due to the use of a blood cuvette to simulate tissue optical transmission.

The empirical and theoretical calibration curves obtained for the 740/880 nm sensor were not correlating, as can be seen in Fig. 8(b). The theoretical calibration curve was however similar to results presented by Mannheimer et al. [14]. The constant DC offset between the curves was most likely caused by hardware issues such as emitting intensity discrepancy between the selected 740 nm and 880 nm LEDs or LED emitting angle differences.

Fig. 8(a) also displays the large deviations in theoretical calibration curves that variations in physiological parameters can cause at low saturations. The theoretical curves presented were calculated by using subject-specific physiological parameters measured during the in vivo validation phase. It was noted that these deviations were only noticeable in the 0-80% saturation range. This indicated that the in vitro calibration curves would not be accurate in the low saturation range, as the physiological parameter changes between the in vitro calibration and in vivo validation would have a pronounced effect on the calibration curve. Low saturation accuracy seemed to be best when using the empirical calibration curves for both the 660/910 nm and 740/880 nm sensors.

The bias and precision errors of the in vivo low saturation measurements could have been affected by the operating principle of the pressure cuff. By fixating the cuff with Velcro...
strips, no repeatable and sustainable method was available to ensure that each pneumatic pressure pulse would induce a repeatable blood pulse. It was thus not possible with the current design to ensure that all artificial blood pulses had similar pulse volumes, a factor that has a major effect in low saturation scenarios where physiological parameter variations have a large influence on sensor performance [14]. Pulse volume variations in a subject PPG and its effect on \( R \) can be seen in Fig. 9. An unusually high pressure of 600 mmHg was used in the peristaltic pressure cuff to ensure that a clear arterial pulse was induced in both the arterial and venous circulations. Due to the yielding of the Velcro and the resultant inefficient pressure transfer, lower pressures would actually be exerted on the finger.

When comparing low saturation results for the 660/910 nm and 740/880 nm sensors (Fig. 7(b)), it is evident that the 740/880 nm sensor has better precision compared to the 660/910 nm sensor. Additionally, the lower sensitivity of the 740/880 nm sensor’s calibration curve requires calculated \( R \) values which are much more repeatable to achieve the same saturation repeatability than that shown for the 660/910 nm sensor.

The arterio-venous results seem to indicate that the measurement of \( S_{\text{pO}_2} \) is possible, taking into account that an arterial-to-venous compliance ratio of 1:1 was used as first approximation throughout the calculations. To demonstrate the effect of the arterial-to-venous compliance ratio, consider the \( S_{\text{pO}_2} \) values measured for one of the volunteers. The original \( S_{\text{pO}_2} \) value was calculated at 51% with the reference \( S_{v\text{O}_2} \) at 82%. If the arterial-to-venous ratio is changed from 1:1 to 1:5, the calculated \( S_{\text{pO}_2} \) value changes to 70%. Hence more research into an appropriate value for the arterial-to-venous ratio is required to improve the APO’s performance.

Pneumatic pressure discrepancies might also have a pronounced effect on \( S_{\text{pO}_2} \) measurements. In the arterio-venous hypothesis, it is assumed that the pressure exerted on the finger is higher than systolic pressure and that an artificial pulse is thus induced in both the arterial and venous circulation. This assumption is only valid if the pressure cuff’s diameter is sufficiently small to ensure efficient pressure transmittance from the higher-than-systolic pneumatic pressure in the cuff to the finger. If the Velcro yields during data acquisition or if the cuff is too loosely fitted around the finger, the combined arterial and venous \( R \) to be representative of only venous pulsations. If \( S_{\text{pO}_2} \) is used in conjunction with this venous \( R \), the calculated \( S_{\text{pO}_2} \) would be much lower than the reference \( S_{v\text{O}_2} \).

VI. CONCLUSION

It can be concluded that the APO shows promising results for the non-invasive real-time measurement of \( S_{\text{aO}_2} \) and \( S_{v\text{O}_2} \) in low saturation and perfusion scenarios. Improved results in terms of accuracy and repeatability may be achieved by implementing design changes based on the experience with the current prototype.

VII. APPENDIX A: PHOTO DIFFUSION MODEL FOR APO

A mathematical model was used to verify the arterio-venous hypothesis theoretically and to study physiological scenarios not approximated in an in vitro or in vivo experiment. The photon diffusion theory model developed by Schmitt [12], was used due to its demonstrated correlation with other models and empirical results [12,14].

Photon diffusion theory approximates photon propagation through a medium as a diffusive process. Tissue is well-suited to diffusive theory modelling due to a high optical depth and short mean free path. The governing equation which describes photon propagation as a diffusive process

\[
\frac{1}{\rho^2} \frac{d}{d\rho} \left( \rho^2 \frac{d\psi}{d\rho} \right) = \alpha^2 \psi \rho = -\frac{1}{D} S \rho 
\]

was derived by Schmitt [12] using an angle-independent solution of the general transport equation. In (2) \( \rho [\text{mm}] \) is the physical distance from the photon source, \( \psi(\rho) \) and \( S(\rho) \) are the scalar photon density and source functions, respectively, at point \( \rho \), \( \alpha [\text{mm}^{-0.5}] \) is the attenuation coefficient and \( D [\text{mm}] \) is the diffusion coefficient. \( \alpha \) and \( D \) can be described in terms of the bulk absorption coefficient \( \Sigma_a [\text{mm}^{-1}] \) and the transport corrected bulk scattering coefficient \( \Sigma_s' [\text{mm}^{-1}] \) as follows:

\[
\alpha = \sqrt{3\Sigma_a \Sigma_s' + \Sigma_s} 
\]

\[
D = \frac{1}{\Sigma_a + \Sigma_s} 
\]
\[ D = \frac{1}{3} \sum_{a} + \Sigma_{a} \] (4)

From the subsequent solution of (2) by Schmitt [12], blood oxygen saturation \( S_{a}O_{2} \) was related to the red/infrared ratio \( R \) through the following

\[ S_{a}O_{2} = \frac{R\sigma_{0\%}^{air} - K_{t}\sigma_{0\%}^{air} - \sigma_{0\%}^{air} + R \sigma_{100\%}^{air} - \sigma_{100\%}^{air}}{K_{t}' \sigma_{100\%}^{air} - \sigma_{100\%}^{air} + R \sigma_{100\%}^{air} - \sigma_{100\%}^{air}} \] (5)

with

\[ \Sigma_{a,f} = \left( \alpha_{f} \sigma_{f} d - 1 \right) \]
\[ \Sigma_{a,i} = \left( \alpha_{i} \sigma_{i} d - 1 \right) \] (6)

Where \( \sigma_{0\%}^{air} \) and \( \sigma_{100\%}^{air} \) are the optical absorption cross-sections for red blood cells containing totally deoxygenated and oxygenated haemoglobin, and \( d \) is the finger diameter. A subscript \( f \) or \( i \) indicates an optical property at the lower red wavelength \( (f) \) or higher infrared wavelength \( (i) \).

For the APO, the bulk absorption coefficient was calculated according to Schmitt [12]. Mie algorithms provided by Michel [18] were used to calculate the absorption cross-sections of red blood cells, and the absorption by bloodless tissue was approximated according to Jacques [19]. The transport corrected scattering coefficient \( \Sigma_{a} \) was also derived from Schmitt [12], with scattering coefficients for whole blood and bloodless tissue taken from Meinke et al. [20] and Simpson et al. [21]. A complete description of the mathematical procedures is available in Schoevers [22].

In the derivation of the solution of \( R \) by Schmitt [12], the AC component of the PPG is seen as a function of variation in the bulk absorption coefficient \( \Sigma_{a} \) due to a time-varying increase in arterial blood volume. This AC component was summarized as follows [12]:

\[ \Sigma_{a} + \frac{\Delta \Sigma_{a}}{} = \left( V_{a} + \Delta V_{a} \right) \Sigma_{a}^{art} + \left( V_{v} + \Delta V_{v} \right) \Sigma_{a}^{ven} + \left[ 1 - \left( V_{a} + \Delta V_{a} + V_{v} + \Delta V_{v} \right) \right] \Sigma_{a}^{bs} \] (7)

with \( V_{a} \) and \( V_{v} \) the volume fractions of arterial and venous blood, and \( \Sigma_{a}^{art} \) and \( \Sigma_{a}^{ven} \) the absorption coefficient for arterial and venous blood respectively. \( \Sigma_{a}^{bs} \) is the absorption coefficient of bloodless tissue.

As can be seen in equation (7), only changes in arterial blood volume were addressed. The solution for \( R \) derived from this relationship would not be suitable for modelling the APO’s behavior. The AC component represented by equation (7) was thus modified to include the effects of venous blood volume changes. The modified representation of the AC component then becomes:

\[ \Sigma_{a} + \frac{\Delta \Sigma_{a}}{} = \left( V_{a} + \Delta V_{a} \right) \Sigma_{a}^{art} + \left( V_{v} + \Delta V_{v} \right) \Sigma_{a}^{ven} + \left[ 1 - \left( V_{a} + \Delta V_{a} + V_{v} + \Delta V_{v} \right) \right] \Sigma_{a}^{bs} \] (8)

with \( \Delta V_{a} \) the influx of arterial blood during systole. Hence:

\[ \Delta \Sigma_{a} = \Delta V_{a} \Sigma_{a}^{art} - \Sigma_{a}^{bs} + \Delta V_{v} \Sigma_{a}^{ven} - \Sigma_{a}^{bs} \] (9)

If it is assumed that \( \Sigma_{a}^{art} \) and \( \Sigma_{a}^{ven} \) >> \( \Sigma_{a}^{bs} \), it can be concluded that

\[ \Delta \Sigma_{a} = \Delta V_{a} \Sigma_{a}^{art} + \Delta V_{v} \Sigma_{a}^{ven} \] (10)

Using the same logic as Schmitt [12] in obtaining equation (5), the ratio \( R \) can now be expressed as

\[ R = \frac{K_{t}' \Sigma_{a}^{art} \Delta V_{a} + \Sigma_{a}^{ven} \Delta V_{v}}{\Sigma_{a,br} \Delta V_{a} + \Sigma_{a,br} \Delta V_{v}} \] (11)

A relationship between \( V_{a} \) and \( V_{v} \) is thus required to solve \( R \). This was the origin of the arterio-venous hypothesis. If it is assumed that pressure pulses of equal magnitude are created in the arterial and venous circulation by the APO, the relationship between \( V_{a} \) and \( V_{v} \) will be determined by the arterial-to-venous compliance ratio. Research into arterial-to-venous compliance ratios in literature led to widely varying values stated in different formats [23-25]. We used an arterial-to-venous ratio of 1:1 as a first approximation. The solution for \( R \) in the presence of arterial and venous pulsating behaviour thus becomes:

\[ R = K_{t}' \frac{S_{a}O_{2}}{\Sigma_{a}^{art} \Sigma_{a,br} \Delta V_{a} + \Sigma_{a}^{ven} \Sigma_{a,br} \Delta V_{v}} + 2 \sigma_{0\%}^{air} \frac{S_{a}O_{2}}{\Sigma_{a}^{art} \Sigma_{a,br} \Delta V_{a} + \Sigma_{a}^{ven} \Sigma_{a,br} \Delta V_{v}} + 2 \sigma_{0\%}^{air} \frac{S_{a}O_{2}}{\Sigma_{a}^{art} \Sigma_{a,br} \Delta V_{a} + \Sigma_{a}^{ven} \Sigma_{a,br} \Delta V_{v}} \] (12)

By stating \( S_{a}O_{2} \) as a constant and specifying \( S_{a}O_{2} \) as values ranging from 0% to 100%, \( R \) could thus be calculated. The calculation can be done for any selected compliance ratio.

REFERENCES

Low Oxygen Saturation Quantification in Human Arterial and Venous Circulation

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