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TOWARDS A HEMODYNAMIC MODEL TO CHARACTERIZE INACCURACIES IN FINGER PULSE OXIMETRY

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Abstract

A pulse oximeter monitors a patient's functional arterial oxygen saturation (SpO$_2$) by illuminating vascularized tissue. However, the optical signals measured, called photoplethysmograms (PPGs), are easily distorted by motion, leading to inaccurate SpO$_2$ readings. Motion artifacts in PPGs are partly attributed to hemodynamic variations, though the exact mechanism is not understood. This paper introduces a model-based approach to improve insight in the effects of hemodynamic variations on SpO$_2$. To make a first step towards an improved understanding of hemodynamic variations, a hemodynamic fingertip PPG model has been developed, including hydrostatic pressures. Measurements on a healthy male subject show that the PPG model can explain changes in PPG baseline and pulsatility in a limited range of arterial and venous pressures. The measurements moreover indicate that modeling of blood flow regulations is required to explain transients in PPGs and inaccurate SpO$_2$ readings in more situations.

1 Introduction

Pulse oximeters are commonly used to monitor a patient's blood oxygenation. SpO$_2$ computed by a pulse oximeter is an estimate of the percentage of arterial hemoglobin that carries oxygen. SpO$_2$ is obtained from the periodic decreases in a red and an infrared PPG, which reflect the cardiac pulses in blood volume (Fig. 1). It can be obtained from the pulses, because hemoglobin that carries oxygen (oxyhemoglobin, HbO$_2$) absorbs less red and more infrared light compared to hemoglobin that does not carry oxygen (reduced hemoglobin, Hb).

Performance of pulse oximeters deteriorates significantly during motion, which is partly attributed to hemodynamic variations. To make a first step towards an improved understanding of the effect of hemodynamic variations on SpO$_2$, a hemodynamic fingertip PPG model has been developed, including hydrostatic pressures. Hydrostatic pressures have been included, because these contribute significantly to motion induced hemodynamic variations [3]. The model derived insights improve understanding of the

clinical performance of pulse oximeters, which can be used to make them more robust to motion.

This paper first describes the calculation of oxygen saturations in Section 2. Section 3 explains the development of the fingertip's hemodynamic model. Next, Section 4 describes how the blood volumes obtained from the hemodynamic model are used in the Beer-Lambert law to simulate a PPG. Simulation and experimental results are shown and discussed in Sections 5 and 6 respectively. Finally, Sections 7 and 8 respectively state future work and conclusions.
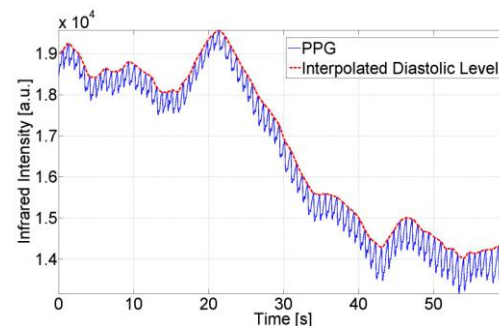
Fig. 1: Recorded infrared PPG (solid blue) and its diastolic level (dashed red). Venous congestion causes the decrease in diastolic level.

2 Arterial and Venous Oxygen Saturations

The operation of pulse oximeters is often modeled using the Beer-Lambert law, which describes the exponential relationship between light absorbance and intensity [2]. The pulses in a PPG can be modeled as follows by the Beer-Lambert law:

$$I_d = I_0 \exp\left(- \sum_{i \in \{a,mv,v\}} \left(\varepsilon_o S_i - \varepsilon_r \left(1 - S_i\right)\right) c_i l_{\text{tip}} \right), \quad (1)$$

in which I_d [W/m^2] is the light intensity detected, I_0 [W/m^2] the light intensity emitted, a, mv and v indicate arterial, microvascular and venous blood respectively, ε_o [M^{-1}m^{-1}] and ε_r [M^{-1}m^{-1}] are the wavelength dependent molar extinction coefficients of HbO$_2$ and Hb respectively, S_i (dimensionless) is the oxygen saturation, c_i [M] the hemoglobin concentration, and l_{tip} [m] the optical pathlength

through the fingertip. The exponent in Eq. (1) models the light's total absorbance by blood.

SpO$_2$ is derived from the ratio Γ of cardiac induced changes in the red and infrared absorbances:

$$\text{SpO}_2 = \frac{\varepsilon_{rR} - \varepsilon_{rIR}\Gamma}{(\varepsilon_{rR} - \varepsilon_{oR}) - (\varepsilon_{rIR} - \varepsilon_{oIR})\Gamma} \qquad (2)$$
$$= n_a S_a + n_{mv} S_{mv} + n_v S_v,$$

in which subscripts R and IR respectively indicate red and infrared light, and $n_i = \Delta c_i / (\Delta c_a + \Delta c_{mv} + \Delta c_v)$ is the relative change in hemoglobin concentration. Equation (2) shows that SpO$_2$ is a weighted average of the arterial, microvascular and venous saturations. If the PPG pulses are caused by arterial blood only, as assumed in pulse oximetry, SpO$_2$ provides an estimate of the arterial oxygen saturation.

Changes in venous blood volume in the fingertip can be used to determine venous oxygen saturation (SvO$_2$) [3]. By inflating a cuff wrapped around the arm one can increase venous blood pressure causing venous congestion. Venous congestion is reflected as a decrease in the PPG's diastolic level (Fig. 1). The change in diastolic level can be used to compute the ratio Γ of simultaneous changes in red and infrared absorbances. Ratio Γ can subsequently be used in Eq. (2) to obtain SvO$_2$.

3   Hemodynamic Model

Blood flow in a vascular tree can be modeled by an electrical equivalent network, in which voltages represent blood pressures and currents represent blood flows. The proposed model of the blood flow in the fingertip is shown in Fig. 2, in which P [mmHg] indicates pressure, i [l/min] flow and t [s] time.

The vasculature in the fingertip has been lumped in an arterial (subscript a), a microvascular (subscript mv) and a venous compartment (subscript v). Flow resistance R [mmHg s/m^3] has been modeled by a Poiseuille resistance and inertance has been discarded, because of the low flow velocity and the small diameters of the vessels in the fingertip. The arterial and venous resistances depend on their respective volumes $V_a(t)$ [l] and $V_v(t)$ [l]. The microvascular resistance is assumed constant.

The arterial pressure-volume (PV) relationship in the fingertip can be described by the following empirical relation [1]:

$$V_a(P_a(t)) = V_{a1} - V_{a2}\exp(-nP_a(t)), \qquad (3)$$

in which V_{a1} [l], V_{a2} [l] and n [mmHg^{-1}] are constants. The arterial compliance $C_a(P_a(t))$ [l mmHg^{-1}] is defined as the derivative of arterial volume (Eq. (3)) with respect to arterial pressure. The venous PV relationship has been assumed linear:

$$V_v(P_v(t)) = C_v P_v(t) + V_{vo}, \qquad (4)$$

in which C_v [l mmHg^{-1}] is the constant venous compliance and V_{vo} [l] the nonzero volume required to have both a realistic venous volume and compliance. The microvessels' volume is assumed constant because pulsations are very small in the "relatively nondistensible" microvessels.

A hydrostatic pressure has been added to a time-varying arterial pressure wave to obtain $P_{a\_in}(t)$ [mmHg] and to a constant venous pressure to obtain $P_{v\_in}(t)$ [mmHg].
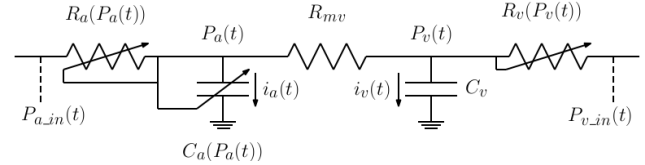
Fig. 2: Electrical equivalent model of the blood flow in a human fingertip.

4   Optical Model

The red and infrared PPGs are modeled by the Beer-Lambert law (Eq. (1)), using the blood volumes which result from the hemodynamic model. So the fingertip is considered a single compartment of constant thickness l_{tip} which contains uniformly distributed arterial, microvascular and venous blood. The amount of light absorbed by each of the blood volumes depends on its saturation S_i. The concentration of arterial, microvascular and venous hemoglobin $c_i(t)$ in the fingertip depends on the respective volumes $V_i(t)$ according to:

$$c_i(t) = \frac{\rho_{Hb}V_i(t)}{m_{Hb}V_{tip}}, \quad i \in \{a, mv, v\}, \qquad (5)$$

in which ρ_{Hb} [g/l] is the density of hemoglobin in blood, m_{Hb} [u] the gram molecular weight of hemoglobin and V_{tip} [l] the total blood volume in the fingertip. Equations (3) and (4) respectively describe the pressure-dependent time-varying arterial and venous blood volumes in the fingertip. The microvascular volume is assumed constant. The red and infrared PPGs are simulated using Eq. (1) normalized by I_0.

5   Simulation Results

Figure 3 shows a simulated infrared PPG when arterial and venous hydrostatic pressures are subsequently 0, 10 and 20 mmHg. A decrease in diastolic level and pulsatility can be observed as a result of increased blood volume and increased arterial pressure respectively. Simulations have shown that the blood pressures, flows and volumes agree with the data presented in medical literature.
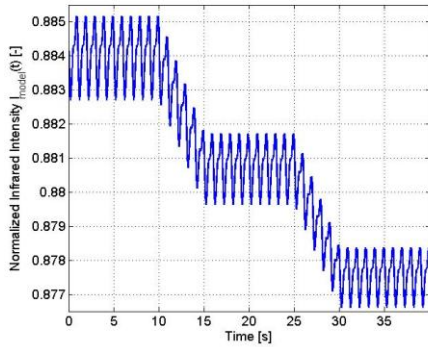
Fig. 3: Simulated IR PPG with arterial and venous hydrostatic pressures of 0, 10 and 20 mmHg.

6 Experiment Design and Results

Because measured SpO_2 depends on fluctuations in blood volumes (Eq. (2)), experiments have been designed to validate the modeled PV relationships. All measurements have been obtained from a single healthy person in a sitting position (25 year old male), because the experiments are intended to obtain initial evidence of the model's capabilities. The model has been validated by comparing measured PPGs to PPGs obtained from simulations similar to the experiments.

Blood volume in the fingertip is measured via a 659 nm and an 880 nm PPG. Finger arterial blood pressure is measured using a Portapres Model-2 (FMS, Finapres Medical Systems BV Amsterdam, The Netherlands). The Portapres' hydrostatic sensor has been used to measure hydrostatic pressure.

Experiments have been designed to ensure reproducibility. Measurements have been done in a temperature controlled environment (22°C-24°C). During the measurements the subject does not take deep breaths and refrains from speaking to prevent irregular changes in thoracic pressure. The subject is not using any medication and has not consumed any coffee during a period of three hours preceding the experiments. Measurements are done on different days to account for day to day changes in vascular smooth muscle tone.

6.1 Hydrostatic Challenges by Altering Height

To validate the fingertip's combined arterial and venous PV relationship, the arm has been positioned at different heights such that hydrostatic pressures of approximately -30, -20, 0, 10, 20, 40 and 50 mmHg are attained. The arm is returned to a resting level between subsequent heights, at which the hydrostatic pressure equals approximately 10 mmHg.
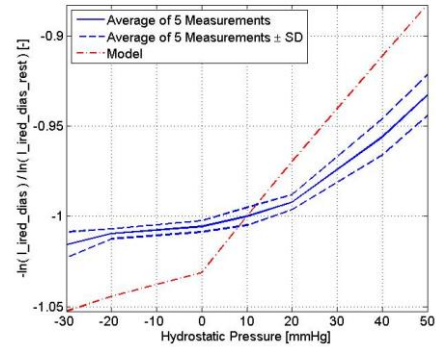
Fig. 4: IR light absorbance vs. hydrostatic pressure.

Figure 4 shows the average diastolic light absorbance as a function of hydrostatic pressure. It has been normalized by the absorbance measured when the arm is at rest level to compensate for differences in blood volume and differences in I_0 between the measurements and the model. Because absorbance is a measure of blood volume, Fig. 4 reflects the fingertip's PV relationship. Both the measured and simulated absorbances show two regimes. A slow increase is observed when the arm is elevated so arterial blood volume is influenced mostly. A fast increase is observed when the arm is lowered so venous volume increases as well. These results suggest that the model approximates the fingertip's PV relationships reasonably.
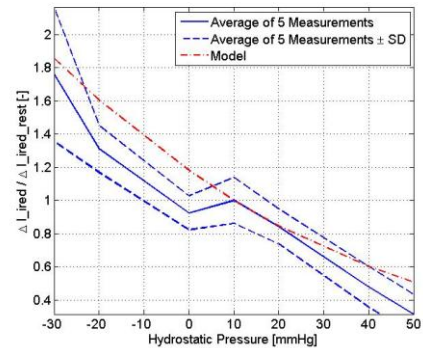
Fig. 5: IR PPG pulsatility vs. hydrostatic pressure.

Figure 5 shows the average PPG pulsatility (peak to peak amplitude) as a function of hydrostatic pressure. It has been normalized by the pulsatility measured when the cuff is deflated to compensate for day to day changes in vascular smooth muscle tone. The arterial compliance model used is reasonable up to hydrostatic pressures of 20 mmHg: at higher pressures simulated PPG pulsatility levels off, whereas measured pulsatility keeps decreasing. This discrepancy can be explained by the venoarteriolar response (VAR), which constricts arterial flow when venous pressure exceeds 25 mmHg, and the myogenic reflex, which constricts arterioles in response to an increase in arterial

pressure [4]. The increased arterial smooth muscle tone decreases arterial compliance, resulting in a diminished arterial pulsatility.

Simulated PPGs follow changes in input pressures directly, whereas measured PPG transients last 10 s to 80 s. The slow transients are likely a result of the VAR and myogenic reflex, as they decrease arterial flow. Moreover, the venous blood column may build up slower than the arterial hydrostatic column, because venous valves slow down back flow [4].

6.2 Venous Congestion by Cuff Inflation

To validate the PV relationship of the veins in the fingertip separately, a cuff wrapped around the upper arm has been inflated from 25 mmHg to 60 mmHg in steps of 5 mmHg. Since venous pressure in the fingertip can be considered equal to cuff pressure, cuff inflation results in venous congestion. The cuff is deflated between subsequent pressure levels.

Measured diastolic light absorbance increases linearly as a function of cuff pressure, reflecting a linear PV relationship. Venous oxygen saturations obtained from the measured PPGs indicate that venous blood causes the volume increase. So a linear PV relationship seems adequate for the veins.
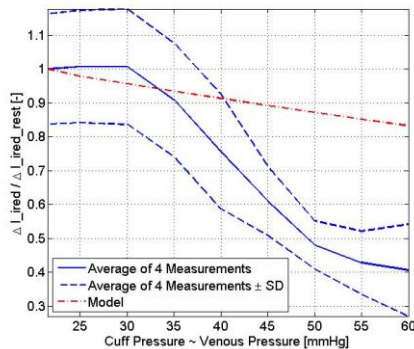
Fig. 6: IR PPG pulsatility vs. cuff pressure.

Figure 6 shows the average PPG pulsatility as a function of cuff pressure. It has been normalized by the pulsatility measured when the cuff is deflated to compensate for day to day changes in vascular smooth muscle tone. Measured pulsatility decreases significantly when the cuff pressure exceeds 30 mmHg. Simulated pulsatility decreases only gradually. A decrease in average SpO_2 from 99% to 97% coincides with the measured decrease in pulsatility. This indicates a decrease in arterial pulsatility (Eq. (2)), which is attributed to the VAR.

7   Future Work

The influences of motion on blood pressure waveforms can be modeled by adding motion induced acceleration forces to the model's pressure inputs. The effects of external pressures applied to the fingertip can be captured by modeling vessel wall motion by mass-spring-damper systems. By varying model parameters, the effects of motion on the fingertip blood flow and PPG can be characterized for different patient conditions. The results improve understanding of the clinical performance of pulse oximeters, based on which SpO_2 algorithms can be made more robust to motion.

8   Conclusions

Measurements have demonstrated that the proposed hemodynamic PPG model explains the main changes in absorbance caused by changes in hydrostatic pressure or venous congestion. This implies that the model approximates the fingertip's arterial and venous PV relationships. Since the model partly captures the arteries' compliance characteristics, it describes changes in arterial pulsatility within a limited range of arterial pressures.

The study identified the VAR and myogenic reflex as regulations responsible for SpO_2 inaccuracies and slow PPG transients. These regulations are to be modeled to be able to describe these phenomena.

The study indicated that also in stationary situations venous pulsations contribute to SpO_2 computed from PPG pulses. Therefore, if accurate SpO_2 readings are to be obtained from the PPG pulses, the venous component is to be accounted for.

References

[1] López-Beltrán, E.A. et al. Non-invasive studies of peripheral vascular compliance using a non-occluding photoplethysmographic method. *Medical & Biological Engineering & Computing*, 36, 748–753, 1998.

[2] Mannheimer, P.D. The light-tissue interaction of pulse oximetry. *Anesthesia & Analgesia*, 105(6), S10–S17, 2007.

[3] Nitzan, M. et al. Measurement of oxygen saturation in venous blood by dynamic near infrared spectroscopy. *Journal of Biomedical Optics*, 5(2), 155–162, 2000.

[4] Okazaki, K. et al. Vasoconstriction during venous congestion: effects of venoarteriolar response, myogenic reflexes, and hemodynamics of changing perfusion pressure. *The American Journal of Physiology*, 289, R1354–R1359, 2005.

[5] Tobin, R.M. et al. A characterization of motion affecting pulse oximetry in 350 patients. *Anesthesia & Analgesia*, 94, S54–S61, 2002.