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# Measurement of oxygen saturation in venous blood by dynamic near infrared spectroscopy

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**Abstract.** A method for the measurement of oxygen saturation in the venous blood, $SvO_2$, based on optical measurements of light absorption in the infrared region is presented. The method consists of applying relatively low external pressure of 25 mm Hg on the forearm, thereby increasing the venous blood volume in the tissue, and comparing the light absorption before and after the external pressure application. $SvO_2$ has been determined from light absorption measurements in two wavelengths, before and after the pressure application, using a formula derived for two adjacent wavelengths. The method has been applied to the hands and fingers of 17 healthy male subjects, using wavelengths of 767 and 811 nm. $SaO_2$, the oxygen saturation for arterial blood, was also obtained from photoplethysmographic measurements in these two wavelengths (pulse oximetry) using the same formula. The mean ($\pm$SD) value of $SaO_2$ was 94.5%($\pm$3.0). The mean value of $SvO_2$ was 86.2%($\pm$4.1) for the finger and 80.0%($\pm$8.2) for the hand. These $SvO_2$ values are reasonable for the finger and the hand where arterio-venous anastomoses exist. The method enables the measurement of $SvO_2$ in the limbs, a parameter which is related to tissue blood flow and oxygen consumption.  [S1083-3668(00)01102-3]

Keywords: oxygen saturation; arterial blood; venous blood; pulse oximetry; photoplethysmography; venous occlusion.

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## 1 Introduction

The different light absorption spectrum for oxygenated and deoxygenated hemoglobin had motivated the development of several noninvasive optical methods for the assessment of oxygen saturation in the blood. The most interesting region of the light spectrum for tissue diagnosis is the red to near infrared region (600–1300 nm), in which the absorption coefficient is low enough to allow significant intensity of light to be transmitted through or diffusely reflected from the organ under investigation. The measurement is simple for thin organs, such as fingertip or earlobe, but thicker organs such as breast and head can also be examined by transmission.[1,2] The cutaneous vascular system can be examined using spectroscopic absorption measurements of diffused reflected light.[3,4]

The transmission of light through a given tissue depends on the light absorption and the light scattering coefficients of the arterial and venous blood and of the various components of the tissue. The contribution of the blood to the light absorption can be isolated by using a greater number of wavelengths,[4,5] or by assessment of tissue light absorption and scattering after squeezing the blood out of the tissue.[6,7] Another approach for the isolation of the contribution of blood to the light absorption uses photoplethysmography (PPG)—the measurement of light absorption changes due to the cardiac induced blood volume changes.[4,8] Since the PPG signal originates from the arterial blood volume increase during systole, the measurement of the PPG signal in several wavelengths—pulse oximetry—enables the assessment of the oxygen saturation in the arterial blood, $SaO_2$.

Pulse oximetry provides information on the arterial blood oxygen saturation. Oxygen saturation in the peripheral venous blood $SvO_2$ also has physiological and clinical significance, as lower blood flow to the tissue results in higher utilization of the oxygen in the blood, hence lower value of $SvO_2$. The assessment of blood supply to the limbs can provide information on its adequacy and also on the severity of shock or cardiac failure, in which diversion of blood flow from the peripheral circulation towards more vital organs takes place. However, in contrast to the routine use of pulse oximetry to $SaO_2$ measurement, no accepted method for the measurement of $SvO_2$ is available. Iwasaki et al.[9] suggested an invasive method based on introducing a fiber optic transmission catheter into a peripheral vein for the measurement of the regional $SvO_2$. Similar to pulse oximetry, venous blood oxygen saturation can be obtained noninvasively by measuring the change in light absorption in two wavelengths after inducing change in the venous blood volume. Changes in oxyhemoglobin or deoxyhemoglobin concentration ($\Delta[HbO_2]$ or $\Delta[Hb]$ respectively) in the venous blood were measured by means of a commercial near-infrared spectroscopy (NIRS) device (NIRO 500, Hammamatsu, four wavelengths) during venous occlusion and $SvO_2$[10,11] or oxygen consumption[12] were derived from the values of $\Delta[HbO_2]$ and $\Delta[Hb]$. Similar measure-

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ments, also based on four wavelengths NIRS device, were performed on the brain, after inducing a change in the venous blood volume by means of tilting the head[1,13] or by occluding the jugular vein.[14–16] $\Delta[HbO_2]$ and $\Delta[Hb]$ were also measured in the forearm after venous occlusion using reflection measurements in two wavelengths only,[17] but these changes were only qualitatively evaluated, and were not used for quantitative assessment of $SvO_2$.

In the following, a different method for the assessment of venous blood oxygenation from measurements of light transmission through the tissue after venous occlusion is described. Measurements of the transmission of light in two adjacent wavelengths through venous blood of variable volume induced by venous occlusion were used for the assessment of $SvO_2$, using a simple method which is a modification of pulse oximetry for the measurement of $SaO_2$. The same two wavelengths were also used for the evaluation of $SaO_2$ from the PPG curves.

## 2 Theory

The transmitted light intensity, $I_t$, through a sample of hemolized blood is given by the Beer–Lambert law[4]

$$I_t = I_o \exp(-\alpha d), \tag{1}$$

where $I_o$ is the incident light intensity, $\alpha$ is the absorption constant of the blood, and $d$ is the width of the tissue sample. The transmitted light intensity, $I_t$, through a tissue sample which includes vessels with whole blood is given by

$$I_t = I_o \exp(-\alpha l - \varepsilon c l), \tag{2}$$

where $l$ is the effective optical path length, which is higher than $d$ because of scattering in the tissue, $\varepsilon$ and $c$ are the extinction coefficient and the concentration of the blood, respectively.

During systole the tissue blood volume increases and consequently the light transmission through the tissue decreases. Photoplethysmography (PPG) is the measurement of the oscillatory changes in light transmission through tissue due to the cardiac induced blood volume changes in the tissue. If $I_s$ is the light transmission through the tissue during the maximal increase in tissue blood volume and $I_d$ is the light transmitted through the tissue during end diastole (when the tissue blood volume has its minimal value), then

$$I_s = I_d \exp(-\varepsilon_a \Delta c_a l),$$

$$\ln(I_d / I_s) = \varepsilon_a \Delta c_a l, \tag{3}$$

where $\varepsilon_a$ is the extinction coefficient for the arterial blood, and $\Delta c_a$ is the increase of blood concentration due to the maximal systolic increase of blood volume. If the light transmission is measured for two wavelengths $\lambda_1$ and $\lambda_2$, then

$$\ln(I_d / I_s)_1 = \varepsilon_{a1} \Delta c_{a1} l_1,$$

$$\ln(I_d / I_s)_2 = \varepsilon_{a2} \Delta c_{a2} l_2. \tag{4}$$

For small blood volume changes $\Delta I_a = I_d - I_s \ll I_s$, and $\ln(I_d/I_s)$ can be approximated by $\Delta I_a / I_s$. If the two wavelengths are close to each other, then the difference of the effective optical path length and of the blood concentration change between the two wavelengths can be neglected ($I_1 \approx I_2$, $\Delta c_{a1} \approx \Delta c_{a2}$), and we can define the ratio

$$R_a = \frac{(\Delta I_a / I_s)_1}{(\Delta I_a / I_s)_2} \approx \frac{\varepsilon_{a1}}{\varepsilon_{a2}} \tag{5}$$

which depends on the oxygen saturation of the arterial blood, as will be discussed below.

Usually, in order to have a higher difference in light transmittance between the two wavelengths, commercial pulse oximeters choose one of the wavelengths in the infrared region, above the isosbestic wavelength (805 nm) and the other in the red region, where the difference in the extinction coefficient between oxygenated and deoxygenated blood is maximal. Then the red light scattering constant significantly differs from that of the infrared light (above 800 nm) resulting in significantly different optical path lengths for the two wavelengths. Furthermore, the difference in the absorption constant significantly affects the illuminated tissue region, especially in reflection pulse oximetry. Due to the difference between the optical properties of the red and infrared light the pulse oximeters need calibration, which can be either *in vivo* or *in vitro* calibration.[18–20]

It should be noted that the equations above are based on the assumption that the change in light transmission between systole and diastole is only due to change in blood volume. There is, however, substantial evidence that light transmission through blood also depends on the blood flow, probably due to changes in the blood cell orientation.[20,21] Though this effect may only have minimal influence on the blood flow through the arterial and arteriolar system, it significantly affects the light transmission properties for *in vitro* finger models. Hence the measurement of low value $SaO_2$ by means of pulse oximetry calibrated by *in vitro* models cannot provide reliable quantitative results.[21]

In a similar way to the measurement of $SaO_2$ by pulse oximetry, $SvO_2$ can be derived from transmission measurements of light in two wavelengths before and after an increase in venous blood volume. When the venous blood volume increases, the light transmitted through the tissue changes from $I_{max}$ to $I_{min}$ and the ratio $R_v$, where

$$R_v = \frac{\ln(I_{max} / I_{min})_1}{\ln(I_{max} / I_{min})_2} = \frac{\varepsilon_{v1}}{\varepsilon_{v2}} \tag{6}$$

depends on the oxygen saturation of the venous blood. $E_{v1}$ and $E_{v2}$ are the respective extinction coefficients in the venous blood. In order to obtain a high signal-to-noise ratio it is preferable to have a significant change in venous blood volume. If, however, $\Delta I_v$ is much less than $I_{min}$ then $R_v$ is given by

$$R_v = \frac{(\Delta I_v / I_{min})_1}{(\Delta I_v / I_{min})_2} = \frac{\varepsilon_{v1}}{\varepsilon_{v2}}. \tag{7}$$

The relationship between the ratio $R$ and the oxygen saturation $SO_2$ can be derived from the decomposition of the extinction coefficient $\varepsilon$ into its two components, the extinction coefficients for oxygenated blood $\varepsilon_o$ and for deoxygenated blood $\varepsilon_d$

$$\varepsilon = \varepsilon_o SO_2 + \varepsilon_d(1 - SO_2) = \varepsilon_d + SO_2(\varepsilon_o - \varepsilon_d). \quad (8)$$

Then[18,19]

$$R = \frac{\varepsilon_{d1} + SO_2(\varepsilon_{o1} - \varepsilon_{d1})}{\varepsilon_{d2} + SO_2(\varepsilon_{o2} - \varepsilon_{d2})} \quad (9)$$

and

$$SO_2 = \frac{\varepsilon_{d1} - R\varepsilon_{d2}}{R(\varepsilon_{o2} - \varepsilon_{d2}) + (\varepsilon_{d1} - \varepsilon_{o1})}. \quad (10)$$

In the current study, the transmission of light through the palm of the hand was used for the measurement of $SvO_2$. For these measurements two adjacent wavelengths in the infrared region, 767 and 811 nm, were chosen. This choice reduces the error of neglecting the difference in the scattering constant between the two wavelengths.[19] Furthermore, while the higher absorption of the red light in the tissue significantly reduces the light intensity transmitted through the hand, the higher transmittance of infrared light through the tissue enables a high signal-to-noise ratio.

## 3 Materials and Methods

### 3.1 Subjects and Examinations

Twenty healthy male subjects were examined. PPG in 767 and 811 nm was measured on the fingertip (index finger or thumb) and $SaO_2$ was derived from these measurements as described below. $SvO_2$ in the ulnar side of the hand or in the fingertip was then determined by measuring the light transmission in two wavelengths through the palm of the hand or the fingertip before and after the occlusion of veins in the ipsilateral forearm by means of a pressure cuff. The light transmission was measured at rest for about 30 s, then the cuff air pressure was raised to 25 mm Hg for about 30 seconds, then decreased. A pressure of 25 mm Hg occludes the veins but has no effect on the incoming arterial blood flow. The values of the light transmission in the finger or in the hand before and after the pressure application were used for the evaluation of $R_v$ in the fingertip or in the hand, respectively, using Eq. (6). The subjects were examined in the sitting position, with their hands at heart level.

### 3.2 Pulse Oximetry

The transmission PPG probe is schematically shown in Figure 1. Two infrared emitting diodes (HE7601SG and HE8111, Hitachi, Japan) emitting light at 767 and 811 nm, respectively, were installed at 120° angles to one another so that both illuminated the same skin surface. The spectral bandwidth (at 50% of the intensity level) for the two diodes was ±25 and ±30 nm, respectively. The current through the diodes was modulated at 3 and 30 kHz in order to measure their outputs separately. The light transmitted through the fingertip was detected by a PIN photodetector (S1223-01, Hamamatsu, Japan) and the detector signal was then demodulated, filtered by low-pass filter (0–1 Hz), and amplified. This signal was used for the determination of the baseline $I_s$ of the pulse [see Eq. (5)]. In a parallel channel the signal was high-pass filtered (0.2–40 Hz—time constant of five seconds) and the output was further amplified for the determination of the pulse amplitude [$\Delta I_a$ in Eq. (5)]. The PPG pulses in a period of about 20 s were used for the derivation of $R_a$ and consequently of $SaO_2$.

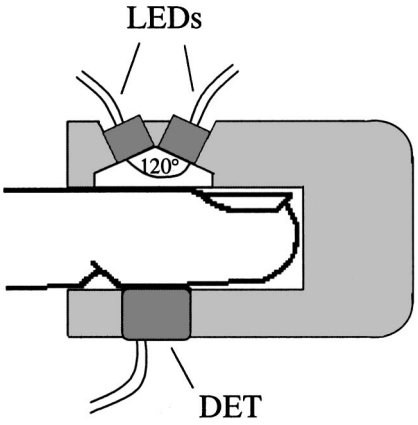


**Fig. 1** The two-wavelength PPG probe. The two LEDs (of emission spectra centered at 767 and 811 nm) are installed at 120° angles to each other. DET–PIN diode photodetector.

In order to check the significance of the wavelength selection, pulse oximetry was also measured at 635 and 937 nm using a probe identical to that of Figure 1. The PPG pulses in the fingertips of ten subjects were examined using both pairs of wavelengths, and the value of $SaO_2$ was derived for each pair of wavelengths, using Eq. (10). Three subjects were also examined by a commercial pulse oximeter (Nelcor Pulse Oximeter, Nelcor, USA).

### 3.3 Venous Blood Oxygenation

Venous blood oxygen saturation was measured in the fingertip and in the hand. The light transmission through the fingertip was measured by means of the PPG probe described above. The device for the measurement of light transmission through the palm of the hand is shown in Figure 2. The light emitting diodes were the same as those for PPG (767 and 811 nm). The photodetector was a photomultiplier tube [PMT, R5108, Hamamatsu, Japan] with a response of 22 mA/W for the 767 and 811 nm. The voltage on the PMT was 700–870 V, depending on the light transmission through the hand. The transmitted light was conveyed to the PMT by a fiber-optic bundle of 3 mm diameter.

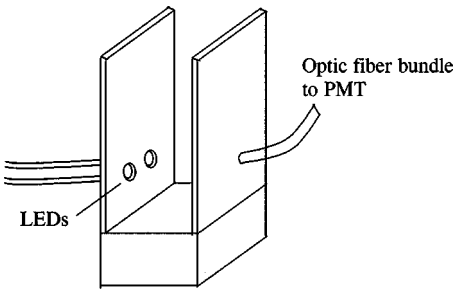


**Fig. 2** The device for the measurement of light transmission through the palm of the hand. The hand is installed between the two plates. The two LEDs (of emission spectra centered at 767 and 811 nm) illuminate the hand, and the optic fiber bundle conveys the transmitted light to the PMT.

$SvO_2$ was derived from the values of the light transmission before and after inducing an increase in venous blood volume [Eqs. (6) and (10)]. The increase in venous blood volume was achieved by applying 25 mm Hg on the forearm as described above.

### 3.4 Data Analysis

The output of the photodetectors was sampled at a rate of 500 samples per sec, digitized and stored in the computer memory for off-line analysis. The minima and maxima of the PPG signal were automatically identified using the algorithm which was previously described.[22] Then the amplitude (AM) and the baseline (BL) of the PPG signal were determined for each pulse. For each PPG examination the value of $R_a$ was derived from the mean values of AM/BL for two wavelengths using Eq. (5).

The mean light transmission before the venous occlusion was derived for a period of 3–5 s before starting the pressure application. The length of the period was chosen so that its light transmission fluctuations were minimal. The light transmission after the venous occlusion was determined for each wavelength in the region where the 811 curve stopped decreasing. From these values $R_v$ was calculated using Eq. (6).

$SaO_2$ and $SvO_2$ were calculated from the $R_a$ and $R_v$ values using Eq. (10) and the corresponding values of the extinction coefficients. For the light of the two light emitting diodes (LEDs), the extinction coefficients were derived from the absorption curves of oxygenated and deoxygenated blood, as published in four articles.[4,18,19,23] The effective extinction coefficient for each LED light (as provided by the manufacturer) was determined for the oxygenated and the deoxygenated hemoglobin using the spectrum of each LED light and the extinction coefficient curves. The mean value of the effective extinction coefficient as derived from these articles was substituted in Eq. (10) and the oxygen saturation in either the arterial or the venous blood was derived from the corresponding value of $R_a$ or $R_v$

$$\text{for 767 and 811 nm:}\quad SO_2 = \frac{1.484 - R}{0.018R + 0.733} \tag{11}$$

$$\text{and for 635 and 937 nm:}\quad SO_2 = \frac{3.97 - R}{0.36R + 3.38}. \tag{12}$$

Equations (11) and (12) were obtained by choosing the lower wavelength as $\lambda_1$ and substituting the corresponding values of the extinction coefficients in Eq. (10).

Figure 3 shows $SO_2$ as a function of $R$ for the pair of wavelengths 767 and 811 nm and for the pair of wavelengths 635 and 937 nm.

## 4 Results

The 767 and 811 nm light transmission curves through the fingertip and the hand before and after the application of 25 mm Hg cuff air pressure for one of the subjects are shown in Figures 4 and 5. Figures 4 and 5 present the low-pass filtered light transmission curves [direct-current (dc) component]. In the finger curve the PPG pulses are presented, and in the hand curves the blood volume oscillations in the respiratory rate

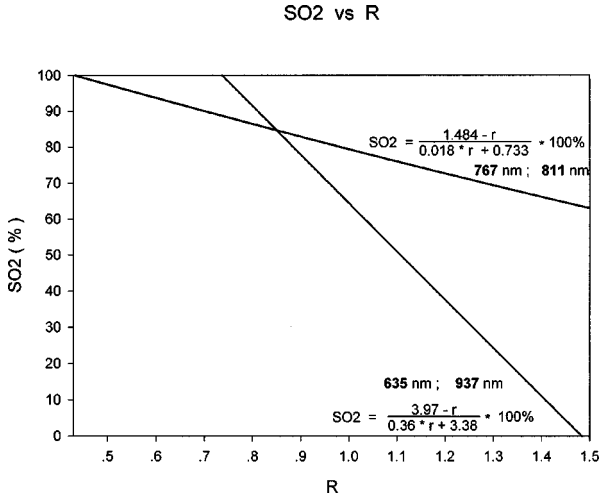


**Fig. 3** $SO_2$ as a function of $R$ for the two pairs of wavelengths. $SO_2$ decreases as $R$ increases.

can be seen. Both 767 and 811 nm light transmission curves show a swift decrease due to venous blood accumulation after the venous occlusion. The 811 nm light (which is close to the isosbestic wavelength) generally reached a plateau, or a minimum, after which the curve slowly increased. In several examinations the 767 nm curve continued to decrease even when the 811 nm stopped decreasing, but the decrease was moderate. The values of light transmission before and during the pressure application period were used for the assessment of $R_v$ and consequently of $SvO_2$, using Eqs. (6) and (11).

In some examinations the light transmission showed significant spontaneous fluctuations which resulted in inaccuracy in the determination of light transmission values before and during pressure application. The main type of fluctuations consisted of low frequency fluctuations in a 10–20 s period which are related to the activity of the autonomic nervous system.[22,24] In most of the examinations in which these fluc-

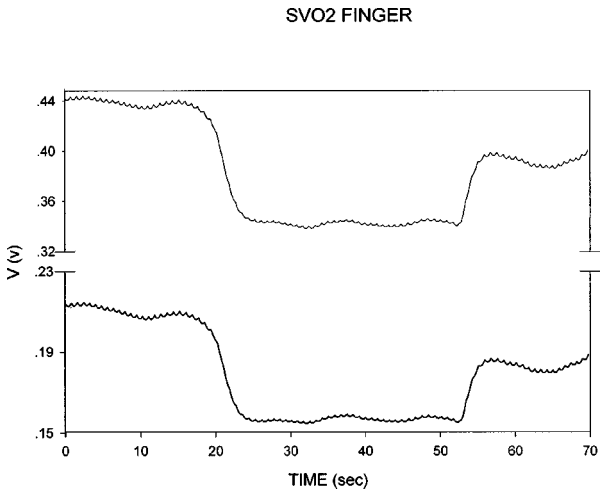


**Fig. 4** The curves of light transmission through the fingertip for the two wavelengths 767 (upper curve) and 811 nm. The decrease in the curves after 17 s is due to higher light absorption in the venous blood volume increase induced by the venous occlusion.

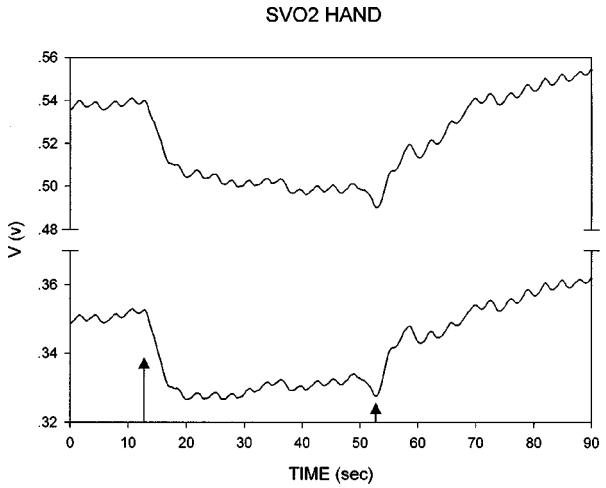


**Fig. 5** The curves of light transmission through the hand for the two wavelengths 767 (upper curve) and 811 nm. The two arrows indicate the period of venous occulsion.

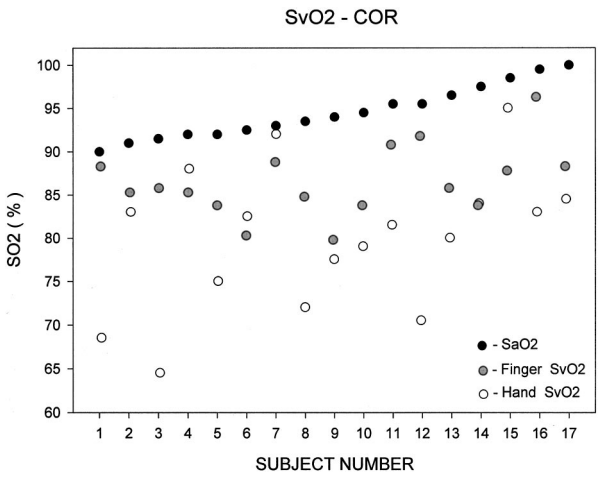


**Fig. 6** The values of $SaO_2$, fingertip $SvO_2$, and hand $SvO_2$ for each of the subjects. $SvO_2$ is lower than $SaO_2$.

tuations appeared it was possible to select a suitable region of relatively constant photodetector output, but for three subjects intense fluctuations did not enable appropriate analysis of the curves. These subjects' examinations were discarded from the study.

Each subject was examined twice, and the values of three parameters—$SaO_2$ (as measured by the fingertip PPG curves) and the fingertip and the hand $SvO_2$—were derived from the corresponding curves of the first and the second examinations. For each subject the values of $SaO_2$ in the two examinations were almost identical, (i.e., differed by 0%–2%) except for one subject, for whom values of 104% and 95% were obtained in the two examinations. The values of the finger $SvO_2$ in the two examinations differed by 0%–10% (mean 4.0%) except for the same subject above, for whom the values of $SvO_2$ were 103% and 89%, respectively. The values of the hand $SvO_2$ for 15 out of the 17 subjects differed by 1%–13% in the two examinations (mean 6.4%). For two subjects the values of hand $SvO_2$ in the two examinations differed by more than 30%: the subject above who showed values of 83% and 52% respectively and another subject who showed values of 75% and 43%. For these two subjects the higher value of $SvO_2$ was taken for further analysis, since the lower values of $SvO_2$ were exceptional (see Sec. 5). For the other 15 subjects the mean value of $SaO_2$ and $SvO_2$ for the two examinations was used for further analysis.

The mean value of $SaO_2$ was $94.5 \pm 3.0\%$. Three subjects, for whom the measured values of $SaO_2$ were 92%, 94%, and 96%, were also examined by means of a commercial pulse oximeter, which showed $SaO_2$ values of 97%–98%. The mean value of finger $SvO_2$ was $86.2 \pm 4.1\%$, and of hand $SvO_2$ was $80.0 \pm 8.2\%$. The value of $SaO_2$ for all the subjects was higher by 2%–15% than finger $SvO_2$ and by 1%–27% than hand $SvO_2$. For 11 subjects the finger $SvO_2$ was higher than hand $SvO_2$. The mean difference between finger $SvO_2$ and hand $SvO_2$ was $6.1 \pm 8.8\%$ which is statistically significant ($p < 0.01$ in paired student $t$-test). Figure 6 depicts the values of $SaO_2$, finger $SvO_2$, and hand $SvO_2$ for each of the subjects. The order of the data is according to the increasing value of $SaO_2$. The values of finger $SvO_2$ and hand $SvO_2$ as a function of $SaO_2$ are shown in Figure 7. Each pair of the three parameters did not show significant correlation.

The $SvO_2$ values were obtained from the time when the 811 nm curve stopped declining or first showed relative constancy—generally 5–10 s after the pressure application. Analysis of the curves taken at later periods provided lower values of $SvO_2$, indicating higher consumption of the tissue blood oxygen, as will be discussed later. The average value of $SvO_2$ at about 30 s after the start of the pressure application was $78.1 \pm 7.3\%$ for the finger and $61.0 \pm 12.4\%$ for the hand.

In ten subjects simultaneous examinations of $SaO_2$ were performed both in the 767 and 811 nm wavelengths and in the 635 and 937 nm wavelengths, using Eqs. (11) and (12), respectively. For the first pair of wavelengths the mean ($\pm$SD) value of $SaO_2$ was 94.2% ($\pm 3.2$). For the second pair the value of $SaO_2$ [as obtained from Eq. (12)] for each subject was lower than that for the first pair, and the mean ($\pm$SD) was 88.3% ($\pm 2.5$).

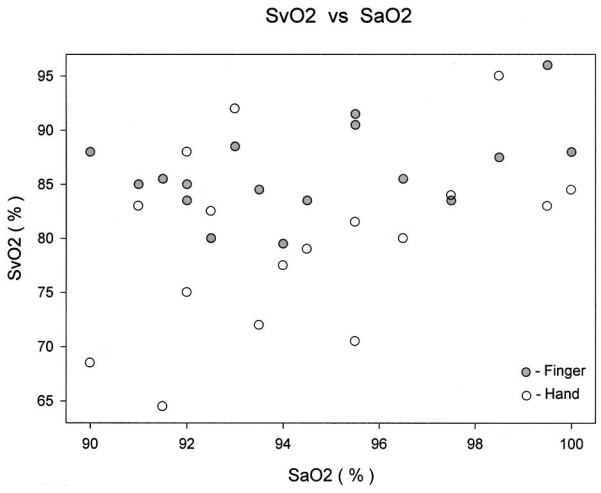


**Fig. 7** The values of fingertip $SvO_2$ and hand $SvO_2$ for each of the subjects. The correlation coefficient is 0.43 for both the finger $SvO_2$ and the hand $SvO_2$.

## 5 Discussion

Pulse oximeters are extensively used for $SaO_2$ measurement in order to assess the adequacy of the cardiopulmonary system, mainly during surgical operations. Since the arterial blood is distributed to all systemic organs from a single source—the left ventricle—the value of $SaO_2$ is the same in all the arteries, and is therefore independent of the arterial blood flow in the tissue under examination. The value of $SvO_2$ is lower than that of $SaO_2$ because of the oxygen consumption in the tissue. Reduction of blood flow while the demand for oxygen from the tissue remains unchanged results in reduced values of $SvO_2$. Hence the measurement of $SvO_2$ provides information on the tissue oxygen consumption or on the tissue blood flow.

In this study $SaO_2$ was measured by pulse oximetry and $SvO_2$ was measured by transmission oximetry of venous blood volume increment in the fingertip and the hand induced by venous occlusion. Both measurements were performed by light of two wavelengths in the near infrared region, at values of 767 and 811 nm. The small difference in the wavelength values assures similar scattering and absorption constants so that the assumption of equal effective path length for the two wavelengths is justified (see Appendix). The drawback of this pair of wavelengths is its low sensitivity due to the small difference in the absorption constant. In conventional pulse oximetry the two wavelengths are chosen to be of greater difference (say in the 650 and 900 nm regions) in order to obtain a greater difference in their PPG curves and consequent higher sensitivity in the measurement of $SaO_2$. For this choice of wavelengths however, the assumption of equal effective path lengths is not valid, and *in vivo* calibration is required for appropriate correspondence between the measured $R_a$ values and the patient's $SaO_2$ values. *In vitro* calibration by means of mechanical models of blood flow cannot provide accurate quantitative values of $SaO_2$ since the flow pattern in the tissue and in the model significantly differ, and the blood flow influences the light scattering by the blood cells due to the dependence of the blood cells' orientation on the blood flow.[20,21]

In our study, Eq. (11), which was obtained from the extinction coefficient values for 767 and 811 nm, was used for the assessment of $SaO_2$ and $SvO_2$ without additional calibration. The values obtained for $SaO_2$ were in the range of 90%–100% with a mean of 94.5%, somewhat lower but in good agreement with those expected for normal subjects, aside from a single examination of one of the subjects where an unexplained value of 104% for $SaO_2$ was obtained. Pulse oximetry measurements with light of 635 and 937 nm and Eq. (12) provided $SaO_2$ values which were lower than the $SaO_2$ values for healthy subjects. The low $SaO_2$ values are probably due to the higher effective optical path length for the red light ($l_1 < l_2$), which result in erroneous high values of $R$ and consequently low values of $SaO_2$ as can be seen in the Appendix. Quantitative assessment of the error due to the different path length for the 767 and 811 nm wavelengths is also given in the Appendix.

$SvO_2$ in the hand and in the fingertip were obtained from light transmission measurements in the two wavelengths before and during venous occlusion. In most examinations the 811 nm curve reached a constant value after about 10 s, while the 767 nm curve continued to decrease (see Figure 5). The explanation for this effect is that the transmission of the 811 nm light, due to its proximity to the isosbestic wavelength, mainly depends on the blood volume in the tissue, and stops decreasing when tissue blood volume reaches equilibrium, i.e., when the venous blood pressure reaches 25 mm Hg. At that point we expect the veins under the cuff to open, but blood flow in the limb will be small because of the high venous blood pressure. The transmitted light intensity for the 767 nm wavelength continues to decrease even when tissue blood volume remains constant because of the ongoing deoxygenation of the blood in the tissue due to lower blood flow and the higher absorption coefficient in the deoxygenated blood for the 767 nm wavelength. The value of $SvO_2$ in the fingertip and in the hand after 30 s of venous occlusion was significantly lower than its value after 10 s of occlusion, due to the lower blood flow.

The decrease in the value of $SvO_2$ after 30 s of occlusion indicates that the venous occlusion may decrease the $SvO_2$ after 10 s too. However, it seems that the venous occlusion affects the blood flow and $SvO_2$ only after a few seconds, so that its effect after 10 s is expected to be small. The effect of the change in the optical properties of the blood and tissue due to the occlusion can also be neglected.[12]

The value of $SvO_2$ in the fingertip is lower than $SaO_2$, but higher than $SvO_2$ in the hand, due to the relatively high amount of arterio-venous anastomoses (AVA) in the fingertip, which do not allow oxygen transfer to the tissue. The value of 80.0% for the hand $SvO_2$ may also be higher than the typical value of tissue $SvO_2$, which is about 75%[10,11,25] since the palm of the hand also contains AVA.

For two subjects significantly lower values of $SvO_2$ were obtained even after about 10 s of occlusion in one of the two examinations. These lower values of $SvO_2$ may occur if arterial blood volume decreases either due to the cuff pressure application or due to spontaneous fluctuations. It should be noted that in some examinations the light transmission curve did not immediately decrease after the application of the air pressure on the forearm, but showed a small increase in light transmission for a short time, then the typical decrease of larger magnitude shown in Figure 5. This small increase in light transmission indicates a small decrease in tissue blood volume, which can result either from back flow of arterial blood towards the region under the cuff, where the cuff air pressure results in lower transmural pressure on the blood vessels, or from a decrease in arterial and arteriolar diameter due to higher activity of the sympathetic nervous system. If so, the measurement of the decrease in light transmission in two wavelengths after venous occlusion can provide reliable quantitative information on $SvO_2$ only after the possible interference of arterial blood volume change is properly considered.

In this study, both $SaO_2$ and $SvO_2$ were derived from measurements of light transmission through the tissue using Eq. (11). The use of similar equation for the reflection mode is not straightforward, and is now under investigation. The use of transmission mode limits the use of the method for fingers, hands, and feet, while reflection measurements can be applied to other organs as well.

## 6 Conclusion

This study shows the possibility of measuring venous oxygen saturation by applying Eq. (11) to the values of light absorption in two close wavelengths before and after induction of venous blood volume increase by means of venous occlusion. The method is based on light transmission measurement, and is therefore applicable to $SvO_2$ measurements in the finger, hand, and foot. Since $SvO_2$ is related to tissue blood flow and oxygen consumption, the method provides information on the adequacy of the tissue blood supply to the limbs. Similarly, arterial blood oxygen saturation can be obtained from the PPG curves in these two wavelengths and Eq. (11), without the need for *in vitro* or *in vivo* calibration.

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## Appendix

Equation (5) is obtained from Eq. (4) by assuming small blood volume changes and a small difference between the two wavelengths in the effective optical path length and blood concentration change. If one only assumes small blood volume changes and a small difference of blood concentration changes, but permits a significant difference in the optical path between the two wavelengths, $\lambda_1$ and $\lambda_2$, then

$$R_a \approx \frac{\varepsilon_{a1}}{\varepsilon_{a2}} \frac{l_1}{l_2}.$$

Taking $\lambda_1$ as the lower wavelength, $l_1 > l_2$ due to the higher scattering for the lower wavelength. Then $R$ in Eqs. (9)–(11) should be replaced by $R(l_2/l_1)$ in order to obtain $SO_2$. By using the measured higher value of $R$ instead of the lower value of $R(l_2/l_1)$, the calculated value of $SO_2$ is lower than its real value. For the two wavelengths 767 and 811 nm, $(l_2/l_1)$ can be assessed from the data of Duncan et al.[26] for the forearm, $(l_2/l_1) = 0.97$.

Equation (11) shows that a decrease in $R$ results in an increase in $SO_2$. The relationship between the change in $R(\Delta R)$ and the change in $SO_2(\Delta SO_2)$ is given by $\Delta SO_2 = (dSO_2/dR)\Delta R$ where

$$dSO_2/dR = 0.760/(0.018R + 0.733)^2.$$

For $R = 0.75$ ($SO_2 = 98\%$) a decrease in $R$ by 3% results in an increase in $SO_2$ by

$$\Delta SO_2 = 1.36\,\Delta R = 1.36 \times 0.75 \times 0.03 = 0.03,$$

i.e., $SO_2$ should be increased by 3%.

For $R = 1.0$ ($SO_2 = 64\%$) an increase in $R$ by 3% results in an increase in $SO_2$ by 4%.

### References

1. J. S. Wyatt, M. Cope, D. T. Delpy, S. Wray, E. O. R. Reynolds, ''Quantification of cerebral oxygenation and haemodynamics in sick newborn infants by near infrared spectrophotometry,'' *Lancet* **8515 ii**, 1063–1066 (1986).
2. H. Heusmann, J. Kolzer, and G. Mitic, ''Characterization of the female breasts *in vivo* by time resolved and spectroscopic measurements in near infrared spectroscopy,'' *J. Biomed. Opt.* **1**, 425–434 (1996).
3. A. Cohen and N. Wadsworth, ''A light emitting diode skin reflectance oximeter,'' *Med. Biol. Eng. Comput.* **10**, 385–391 (1972).
4. Y. Mendelson, ''Transcutaneous blood gas measurement,'' in *Encyclopedia of Medical Devices and Instrumentation*, J. G. Webster, Ed. pp. 448–460, Wiley, New York (1988).
5. J. Farmer, ''Blood oxygen measurement,'' in *Design of Pulse Oximeters*, J. G. Webster, Ed. pp. 21–39, Institute of Physics Publishing, Bristol (1997).
6. J. W. Uzmann and E. H. Wood, ''Oximeter earpiece for measurement of systemic blood content and arterial pressure in the human ear,'' *Am. J. Physiol.* **163**, 756–757 (1950).
7. R. H. Taplin, H. G. Saville, and H. Paul, ''Improved earpiece for oximeters,'' *J. Aviation Med.* **24**, 70–72 (1953).
8. I. Yoshiya, Y. Shimady, and K. Tanake, ''Spectrophotometric monitoring of arterial oxygen saturation on the fingertip,'' *Med. Biol. Eng. Comput.* **18**, 27–32 (1980).
9. H. Iwasaki, K. Yoshizaki, and H. Koyano, ''Fiber-optic transmission catheter for regional venous oxygen saturation and blood flow,'' *J. Appl. Physiol.* **72**, 1616–1621 (1992).
10. C. W. Yoxall and A. M. Weindling, ''The measurement of peripheral venous oxyhemoglobin saturation in newborn infants by near infrared spectroscopy with venous occlusion,'' *Pediatr. Res.* **39**(6), 1103–1106 (1996).
11. C. W. Yoxall and A. M. Weindling, ''Measurement of venous oxyhemoglobin saturation in the adult human forearm by rear infrared spectroscopy with venous occlusion,'' *Med. Biol. Eng. Comput.* **35**, 331–336 (1997).
12. R. A. De Blasi, M. Ferrari, A. Natali, G. Conti, A. Mega, and A. Gaspareto, ''Noninvasive measurement of forearm blood flow and oxygen consumption by rear infrared spectroscopy,'' *J. Appl. Physiol.* **76**(3), 1388–1393 (1994).
13. L. Skov, O. Pryds, G. Greisen, and H. Lou, ''Estimation of cerebral venous saturation in newborn infants by near infrared spectroscopy,'' *Pediatr. Res.* **33**(1), 52–54 (1993).
14. Y. A. B. D. Wickramasinghe, L. N. Livera, S. A. Spencer, P. Rolfe, and M. S. Thorniley, ''Plethysmographic validation of near infrared spectroscopic monitoring of cerebral blood volume,'' *Arch. Dis. Child* **67**, 407–411 (1992).
15. C. W. Yoxall, A. M. Weindling, N. H. Dawani, and I. Peart, ''Measurement of cerebral venous oxyhemoglobin saturation in children by near-infrared spectroscopy and partial jugular venous occlusion,'' *Pediatr. Res.* **38**, 319–323 (1995).
16. C. E. Elwell, S. J. Matcher, L. Tyszczuk, J. H. Meek, and D. T. Delpy, ''Measurement of cerebral venous saturation in adults using near infrared spectroscopy,'' *Adv. Exp. Med. Biol.* **411**, 453–460 (1997).
17. T. Shiga, K. Yamamoto, K. Tanabe, Y. Nakase, and B. Chance, ''Study of an algorithm based on model experiments and diffusion theory for a portable tissue oximeter,'' *J. Biomed. Opt.* **2**(2), 154–161 (1997).
18. O. Wieben, ''Light absorbance in pulse oximetry,'' in *Design of Pulse Oximeters*, J. G. Webster, Ed. pp. 40–55, Institute of Physics Publishing, Bristol (1997).
19. P. D. Mannheimer, J. R. Casciani, M. E. Fein, and S. L. Nierlich, ''Wavelength selection for low-saturation pulse oximetry,'' *IEEE Trans. Biomed. Eng.* **44**(3), 148–158 (1997).
20. K. J. Reynolds, J. T. B. Moyle, L. B. Gale, M. K. Sykes, and C. E. W. Hahn, ''*In vitro* performance test system for pulse oximeters,'' *Med. Biol. Eng. Comput.* **30**, 629–635 (1992).
21. L. G. Lindberg and P. A. Oberg, ''Optical properties of blood in motion,'' *Opt. Eng.* **32**, 253–257 (1993).
22. M. Nitzan, A. Babchenko, and B. Khanokh, ''The very low frequency variability in the arterial blood pressure and in the blood volume pulse,'' *Med. Biol. Eng. Comput.* **37**, 54–58 (1999).
23. R. A. Laing, L. A. Danisch, and L. R. Young, ''The choroidal eye oximeter: An instrument for measuring oxygen saturation of choroidal blood in vivo,'' *IEEE Trans. Biomed. Eng.* **22**, 183–195 (1975).
24. M. Nitzan, S. Turivnenko, A. Milston, A. Babchenko, and Y. Mahler, ''Low frequency variability in the blood volume and in the blood

volume pulse measured by photoplethysmography,'' *J. Biomed. Opt.* **1**, 223–229 (1996).
25. A. C. Guyton, *Textbook of Medical Physiology*, 7th ed., p. 496, W. B. Saunders, Philadelphia (1982).
26. A. Duncan, J. H. Meek, M. Clemence, C. E. Elwell, L. Tyszczuk, M. Cope, and D. T. Delpy, ''Optical pathlength measurements on adult head, calf and forearm and the head of the newborn infant using phase resolved spectroscopy,'' *Phys. Med. Biol.* **40**, 295–304 (1995).