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# ARTERIAL BLOOD OXYGEN SATURATION DURING BLOOD PRESSURE CUFF-INDUCED HYPOPERFUSION

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## ABSTRACT

Pulse oximetry has been one of the most significant technological advances in clinical monitoring in the last two decades. Pulse oximetry is a non-invasive photometric technique that provides information about the arterial blood oxygen saturation (SpO<sub>2</sub>) and heart rate, and has widespread clinical applications. When peripheral perfusion is poor, as in states of hypovolaemia, hypothermia and vasoconstriction, oxygenation readings become unreliable or cease. The problem arises because conventional pulse oximetry sensors must be attached to the most peripheral parts of the body, such as finger, ear or toe, where pulsatile flow is most easily compromised. Pulse oximeters estimate arterial oxygen saturation by shining light at two different wavelengths, red and infrared, through vascular tissue. In this method the ac pulsatile photoplethysmographic (PPG) signal associated with cardiac contraction is assumed to be attributable solely to the arterial blood component. The amplitudes of the red and infrared ac PPG signals are sensitive to changes in arterial oxygen saturation because of differences in the light absorption of oxygenated and deoxygenated haemoglobin at these two wavelengths. From the ratios of these amplitudes, and the corresponding dc photoplethysmographic components, arterial blood oxygen saturation (SpO<sub>2</sub>) is estimated. Hence, the technique of pulse oximetry relies on the presence of adequate peripheral arterial pulsations, which are detected as photoplethysmographic (PPG) signals. The aim of this study was to investigate the effect of pressure cuff-induced hypoperfusion on photoplethysmographic signals and arterial blood oxygen saturation using a custom made finger blood oxygen saturation PPG/SpO<sub>2</sub> sensor and a commercial finger pulse oximeter. Blood oxygen saturation values from the custom oxygen saturation sensor and a commercial finger oxygen saturation sensor were recorded from 14 healthy volunteers at various induced brachial pressures. Both pulse oximeters showed gradual decrease of saturations during induced hypoperfusion which demonstrate the direct relation between blood volumes (PPG amplitudes), arterial vessel stenosis and blood oxygen saturation. The custom made pulse oximeter was found to be more sensitive to SpO<sub>2</sub> changes than the commercial pulse oximeter especially at high occluding pressures.

## 1. Introduction

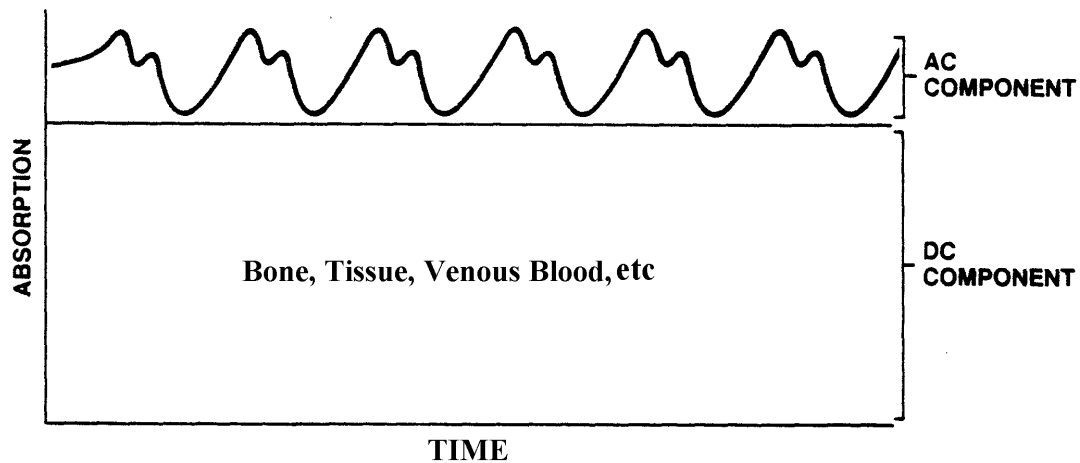
Photoplethysmography is a non-invasive optical technique widely used in the study and monitoring of the pulsations associated with changes in blood volume in a peripheral vascular bed. Whether the term “plethysmography”, is a misnomer is a matter of debate, yet the title has received general consent [1-5, 7].

The human body is normally assumed opaque to light transmission, but most soft tissues will transmit both visible and infrared radiation to some extent [8]. For example when a hand is held over the end of a torch in a dark room it can be seen that a small but perceptible amount of light is transmitted through the whole hand. This transmitted light has a definite red colour due to selective absorption by the blood in the hand. Furthermore, on the side where the torch is held on the skin surface there is a “halo” of back scattered light around the body of the torch. As the eye is essentially an integrating instrument, it is not possible to see the minute changes in the intensity of transmitted light which occur due to the blood pulse in the hand. If, instead of looking at this variable light level with the eye, a photoelectric detector were used, then it is comparatively easy to record these “pulsations” [1]. This is the principle on which photoplethysmography is based. The emitted light, which is made to transverse the skin, is reflected, absorbed and scattered in the tissue and blood. The modulated light level, which emerges, is measured using a suitable photodetector. It is possible for the hand to be directly transilluminated where the light source, usually in the region of 800 nm to 960 nm, is on one side of the skin and the detector on the other side. This method, also called transmission mode, is limited to areas such as the finger the ear lobe or the toe [5,6]. However, when light is directed down into the skin a proportion of this is backscattered so that it emerges from the skin adjacent to the light source. The light source and the photodetector can be positioned side by side. This method, also called the reflection mode, allows measurements on virtually any vascular skin area [5,6].

The intensity of the reflected and backscattered light which reaches the photodetector in either reflection or transmission mode is measured and the variations in the photodetector current are assumed to be related to blood volume changes underneath the probe [5,7]. These variations are electronically amplified and recorded as a voltage signal called the photoplethysmograph (PPG).

The photoplethysmographic signal (Figure 1) is divided into two components:

- i) A dc PPG component, a relatively constant voltage offset of which the magnitude is determined by the nature of the material through which the tissue passes (skin, cartilage, venous blood, etc.). The slowly changing dc component may be extracted using a low pass filter (typical bandwidth dc – 0.5 Hz) [8].
- ii) A pulsatile or ac PPG component synchronous with the heart rate is often assumed to be related to the arterial blood volume pulse. The ac PPG pulse shapes are indicative of vessel compliance and cardiac performance. The ac component, usually has an amplitude of 1-2% of the dc value and it may be extracted by a bandpass filter (typical bandwidth 0.5 Hz to 20 Hz) [8].



**Figure 1:** Photoplethysmographic (PPG) waveform as measured by transmission through tissue

Photoplethysmography is used in the estimation of arterial blood oxygen saturation ( $SpO_2$ ) by pulse oximetry. Pulse oximeters estimate arterial oxygen saturation by shining light at two different wavelengths, red and infrared, through vascular tissue. In this method the pulsatile photoplethysmographic (ac PPG) signal associated with cardiac contraction is assumed to be attributable solely to the arterial blood component. The amplitudes of the red and infrared ac PPG signals are sensitive to changes in arterial oxygen saturation because of differences in the light absorption of oxygenated and deoxygenated haemoglobin at these two wavelengths. From the ratios of these amplitudes, and the corresponding dc photoplethysmographic components, arterial blood oxygen saturation ( $SpO_2$ ) is estimated. Hence, the technique of pulse oximetry relies on the presence of adequate peripheral arterial pulsations, which are detected as photoplethysmographic (PPG) signals [6].

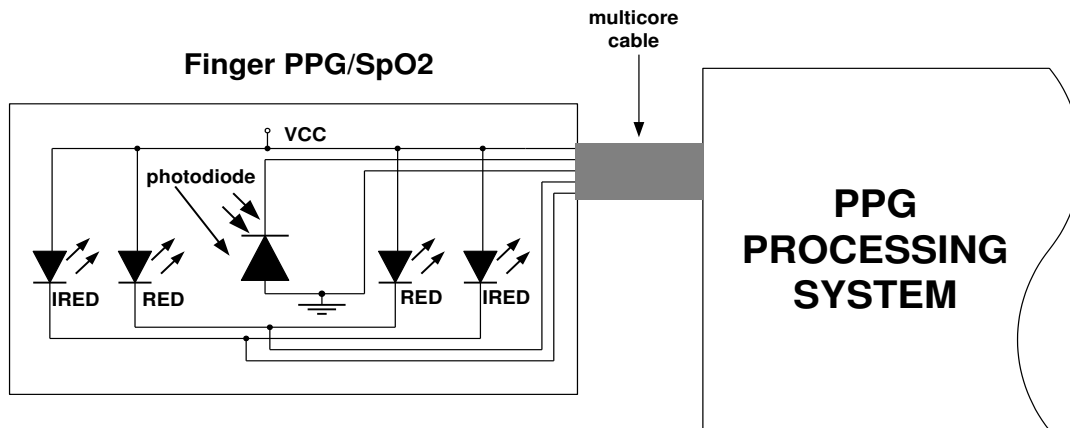
When peripheral perfusion is poor, as in states of hypovolaemia, hypothermia, vasoconstriction, low cardiac output and low mean arterial pressure, pulse oximeter readings become unreliable or cease altogether [9,10]. The oxygenation readings become unreliable in these circumstances because conventional pulse oximeter sensors are usually placed on the most peripheral parts of the body such as the finger, where pulsatile flow is most vulnerable, as it is compromised by diversion of blood flow to more vital organs. Hence, pulse oximetry becomes unreliable in a significant group of patients at just the time when the measurement of blood oxygen saturation would be clinically of most value. Newly developed pulse oximetry technologies such as Masimo SET were designed to display accurately blood oxygen saturation values during motion artefact or during periods of hypoperfusion. However, there are only a few reports on the accuracy of pulse oximeters during hypoperfusion in a clinical setting [11]. This pilot study will investigate in detail the morphology and amplitude of the PPG signal and its effect on pulse oximetry under controlled vasoconstrictive studies.

## 2. Methods

### 2.1 Instrumentation

A reflectance finger PPG/ $SpO_2$  probe was constructed utilising two surface mount infrared (IREDS) and two red emitting diodes and a surface mount silicon diode photodetector (Figure 2). The photodetector detected radiation back scattered by the tissue from both infrared and

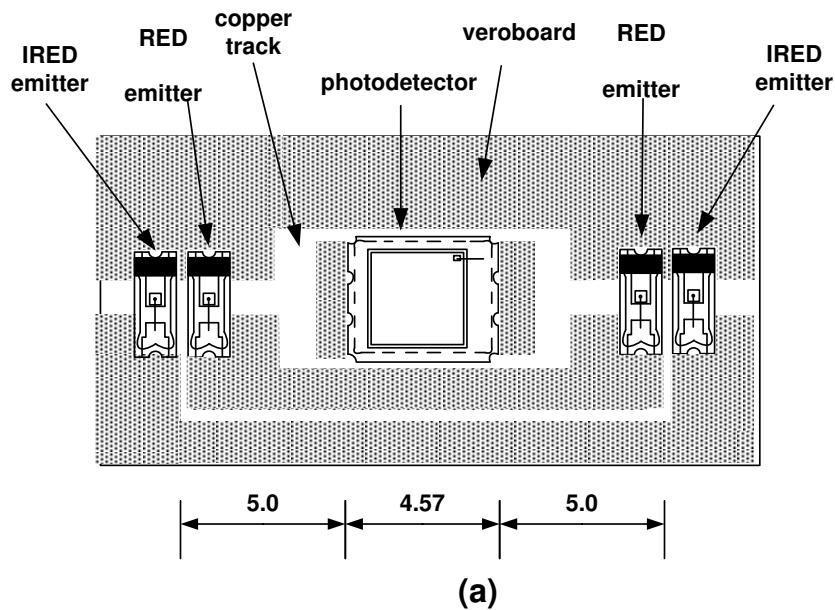
red emitters and gave an output current proportional to the detected radiation level. A screened multicore cable carried the power to the IREDS and REDs in the probe from the main PPG processing unit and also the detected PPG signals from the photodetector.



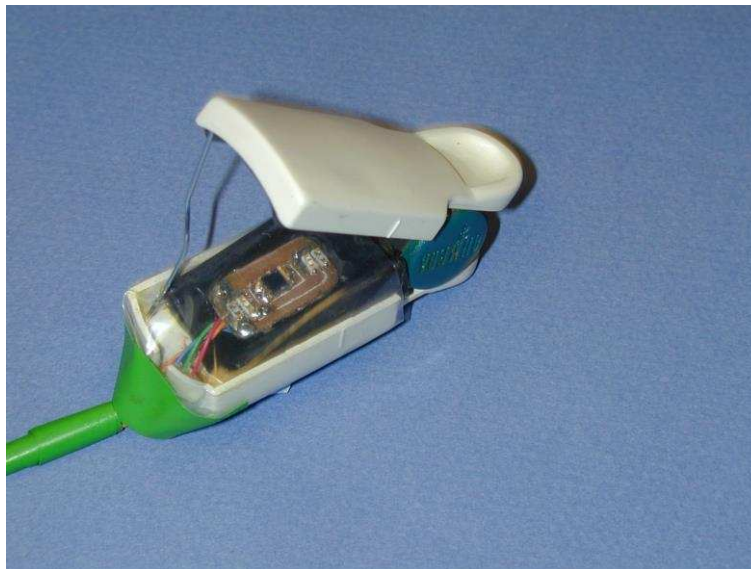
**Figure 2:** Block diagram of the Finger PPG/SpO<sub>2</sub> Probe connected to the PPG Processing System

**Optical Components:** The infrared and red emitters used were ceramic chip surface mount types (dimensions of each: 3.2 mm x 1.27 mm) with peak emission wavelengths at 880 nm and 655 nm, respectively (ELCOS GmbH). The photodetector was a surface mount silicon PhotoPinDiode (dimensions: 4.57 mm x 3.81 mm) which had the positive side on the front and the negative side on a ceramic contact base (ELCOS GmbH).

**Mechanical Construction of the Finger PPG Probe:** The photodetector was mounted between the red and infrared emitters to detect radiation back scattered by the tissue from both infrared and red emitters and gave an output current proportional to the detected radiation level. The distance between the emitters and the photodetector was 5 mm (Figure 3a). The emitter and photodiode chips were mounted on the copper side (Figure 3a) of an epoxy glass copper clad single sided eurocard (dimensions: 20 mm x 10 mm x 1.6 mm). Figure 3b shows a close-up photograph of the complete design of the reflectance finger probe.



\* All measurements are in mm



**Figure 3:** (a) Top view of the custom made finger PPG/SpO<sub>2</sub> probe showing the layout of the surface mount components (IRED and RED emitters and photodetector) mounted on a veroboard; (b) Photograph of the Reflectance finger Probe

An electrically isolated, time-multiplexed PPG processing system [12, 13] was used to detect and pre-process simultaneously the red and infrared ac and dc PPG output signals. Blood oxygen saturation values were also obtained using a commercial transmittance finger pulse oximeter (Diascope 2 VISMO; S&W Medico Teknik, Albertslund, Denmark). Lead II ECG signals were also recorded using a commercial ECG machine (Diascope 2 VISMO; S&W Medico Teknik, Albertslund, Denmark). PPG traces (obtained at red and infrared wavelengths) from the custom made finger pulse oximeter, SpO<sub>2</sub> traces from the commercial pulse oximeter and ECG signals were digitised at a sampling rate of 100 Hz by a 16-bit data acquisition card (National Instruments Corporation, Austin, Texas). The signals were further analysed by the Virtual Instrument (VI) implemented in LabView [12]. All acquired

signals were also saved in spreadsheet format for further post processing analysis. The digitised signals were analysed offline in Matlab 6.5 using the available filter design and signal processing toolboxes.

## **2.2 Measurement**

The institutional Ethics Committee of City University approved this study, and all subjects gave written consent for participation prior to the study. Fourteen healthy male volunteers, mean age,  $\pm$  SD ( $28 \pm 5.2$ ) who had not been taking any regular medication and were free of cardiovascular or chronic pulmonary diseases or other significant medical problems participated in this study.

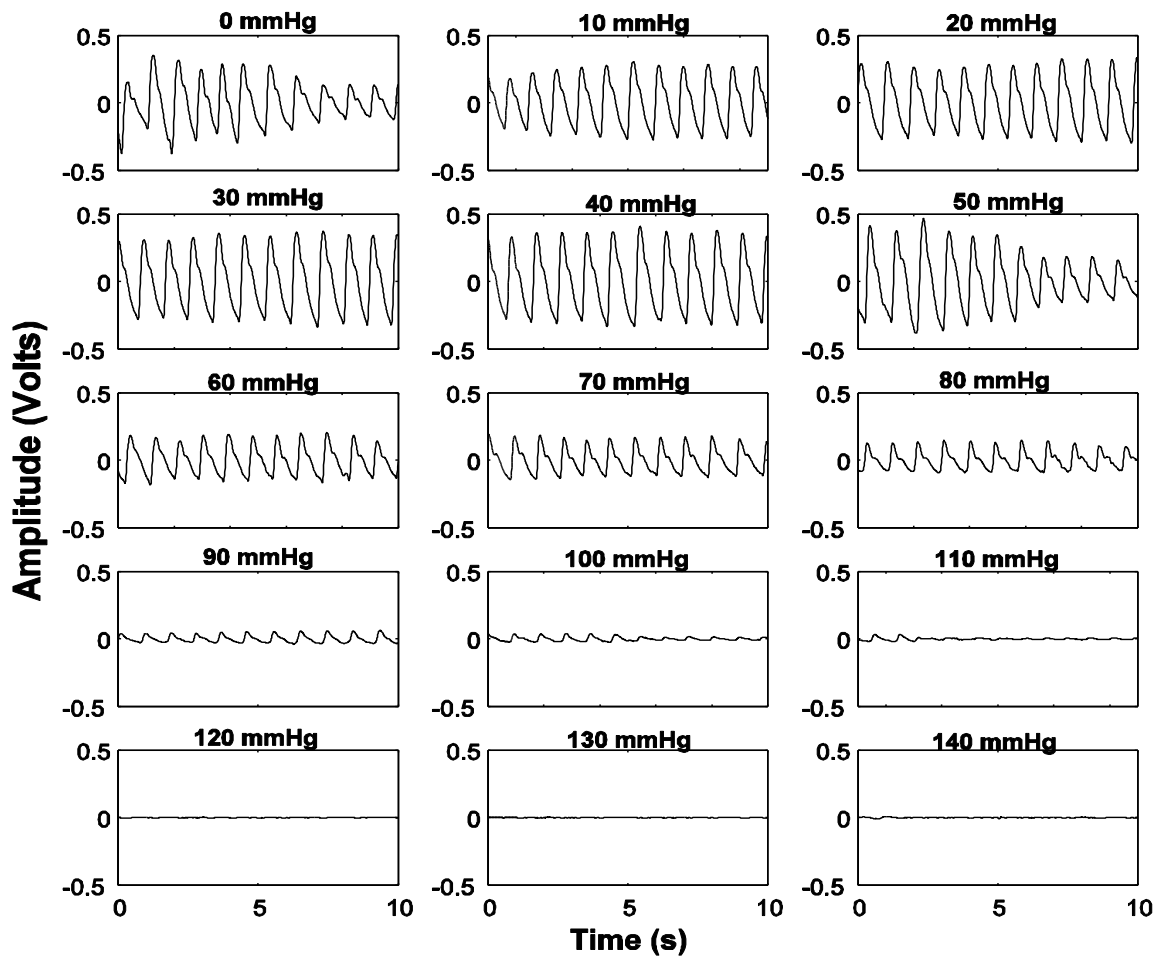
All measurements were performed in a control lab facility. Volunteers were told to rest comfortably and quietly in the supine position in an examination table for three minutes to obtain a stable haemodynamic period. Left and right arm blood pressures using a sphygmomanometer were taken prior to the signal acquisition. The cuff of the sphygmomanometer was then placed on the left arm at the level of the brachial artery. The custom made reflectance finger PPG/SpO<sub>2</sub> probe was placed on the index finger of the left hand and the commercial transmittance finger pulse oximeter was placed on the ring finger of the same hand. The volunteer was also connected to an ECG machine. Hypoperfusion was induced by gradually occluding the brachial artery using the sphygmomanometer at increments of 10 mmHg (10-15 seconds per pressure increment). During the gradual hypoperfusion process all parameters (ECG, custom made PPG/SpO<sub>2</sub> probe, commercial SpO<sub>2</sub> probe) were monitored and recorded continuously. In the event the volunteer felt uncomfortable, the process was stopped.

## **2.3 Data Analysis and Statistics**

The amplitudes of the finger ac PPG signals (red and infrared) for all fourteen volunteers were measured during the hypoperfusion process, and the means and standard deviations (SD) calculated. A Kruskal-Wallis One Way Analysis of Variance (ANOVA) was performed to see if there was any significant difference between the mean PPG amplitudes at all induced pressures. A value of  $p < 0.05$  was considered statistically significant. The SpO<sub>2</sub> values between the two pulse oximeters during the period of hypoperfusion were also compared.

## **3. Results**

Measurable finger ac PPG traces at both wavelengths were obtained in all volunteers in all pressures taken prior to complete arterial occlusion where the finger PPG signals ceased due to no blood flow to the finger. Figure 4 depicts typical ac infrared PPG traces, at all pressure increments, from one volunteer.

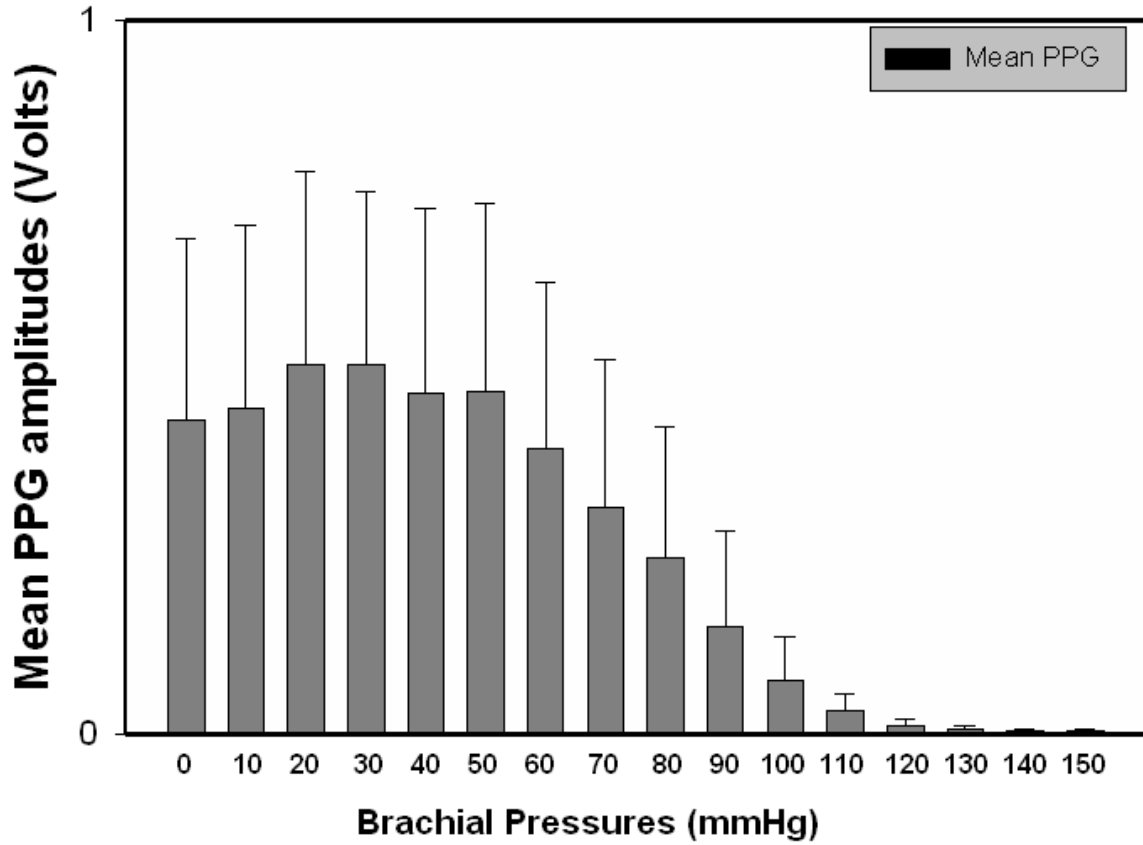


**Figure 4:** Infrared ac PPG signals at various pressure increments (0 to 140mmHg)

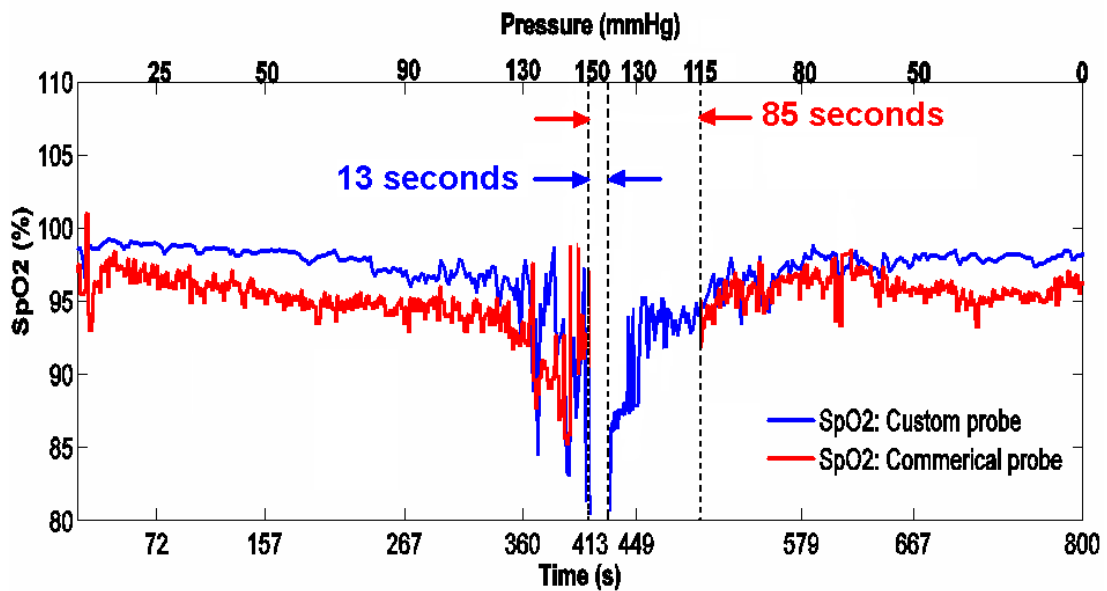
Figure 5 gives the mean  $\pm$  SD of the ac PPG amplitudes at the different pressure increments. To see if there was any significant difference between the mean PPG amplitudes at all induced pressures, a Kruskal-Wallis One Way Analysis of Variance on Ranks was performed. The results of the test show that there are statistically significant differences between the ac PPGs in the low pressures (0 to 80 mmHg) than those in the upper pressures (90 to 150 mmHg) at both wavelengths.

The SpO<sub>2</sub> values from both pulse oximeters were observed during the hypoperfused period. Figure 6 clearly demonstrates the behaviour of the two pulse oximeters during induced hypoperfusion. As it was expected the SpO<sub>2</sub> values decreased gradually as the cuff pressure increased. With the systematic occlusion of the brachial artery the volume of blood reaching the finger was decreased and that was obvious from the changes in the amplitude of the ac PPG signal from the custom made finger probe. The custom SpO<sub>2</sub> probe was found to be much more sensitive to changes in SpO<sub>2</sub> than the commercial finger pulse oximeter and this is clearly visible in Figure 6. This is because most commercial pulse oximeters include time averaging to maintain a signal well after the peak pressure in the cuff has been reached. Also, commercial pulse oximeters such as the one used in this study include time averaging in their algorithms to minimise the influence of transit noise on data or low amplitude pulsations.





**Figure 5:** Mean ( $\pm$  SD) ac PPG amplitudes at different brachial occlusion pressures



**Figure 6:** Blood oxygen saturation traces during hypoperfusion. Gradual hypoperfusion started from 0 to 140 mmHg (time 0 to 400 seconds). After full occlusion the cuff was released gradually down to 0 mmHg (time 420 to 800 seconds).

#### **4. Discussion and Conclusion**

Finger PPG signals with large amplitudes and high signal-to-noise ratios were measured in the majority of induced pressures prior to complete occlusion of the brachial artery in all volunteers. During hypoperfusion the amplitude of the PPG signals were decreased gradually to the point that were not visible at all on the screen of the computer. The mean PPGs at low pressures (0 to 80 mmHg) found to be statistically significant with the mean PPGs at the upper pressures (90 to 150 mmHg) at both wavelengths.

The decrease in the amplitude of the PPG signals correlated well with the decrease in blood oxygen saturation. This is in full agreement with the physiological phenomenon that suggests that during arterial vessel stenosis the volume of blood decreases with a direct effect on SpO<sub>2</sub> values measured at a vascular site downstream from the stenosis. The custom finger pulse oximetry was found to be more sensitive to SpO<sub>2</sub> changes during induced hypoperfusion when compared with the commercial pulse oximetry. Additional clinical studies, in a group of patients with peripheral vascular disease, are suggested to investigate such a phenomenon further.

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