Investigation of oesophageal photoplethysmographic signals and blood oxygen saturation measurements in cardiothoracic surgery patients

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Abstract

Pulse oximeter probes attached to the finger may fail to estimate blood oxygen saturation (SpO_2) in patients with compromised peripheral perfusion (e.g. hypothermic cardiopulmonary bypass surgery). The measurement of SpO_2 from a central organ such as the oesophagus is suggested as an alternative to overcome this problem. A reflectance oesophageal pulse oximeter probe and a processing system implemented in *LabVIEW* were developed. The system was evaluated in clinical measurements on 50 cardiothoracic surgery patients. Oesophageal photoplethysmographic (PPG) signals with large amplitudes and high signal-to-noise ratios were measured from various depths within the oesophagus from all the cardiothoracic patients. The oesophageal PPG amplitudes from these patients were in good agreement with previous oesophageal PPG amplitude measurements from healthy anaesthetized patients. The oesophageal pulse oximeter SpO_2 results agreed well with the estimated arterial oxygen saturation (SaO_2) values inferred from the oxygen tension obtained by blood gas analysis. The mean ($\pm$ SD) of the differences between the oesophageal pulse oximeter SpO_2 readings and those from blood gas analysis was $0.02 \pm 0.88\%$. Also, the oesophageal pulse oximeter was found to be reliable and accurate in five cases of poor peripheral perfusion when a commercial finger pulse oximeter probe failed to estimate oxygen saturation values for at least 10 min. These results suggest that the arterial blood circulation to the oesophagus is less subject to vasoconstriction and decreased PPG amplitudes than are the peripheral sites used for pulse oximetry such as the finger. It is concluded that oesophageal SpO_2 monitoring may be of clinical value.

Keywords: photoplethysmography (PPG), oesophagus, blood oxygen saturation, cardiac surgery

1. Introduction

Photoplethysmography is a non-invasive electro-optical technique widely used in the study and monitoring of the pulsations associated with changes in blood volume in a peripheral vascular bed (Challoner 1979, Roberts 1982, Dorlas and Nijboer 1985, Higgins and Fronek 1986, Lindberg and Oberg 1991).

Photoplethysmography is based on the absorption properties of vascular tissue when it is transilluminated by light. The emitted light, which is made to traverse the skin, is reflected, absorbed and scattered in the tissues and blood. The modulated light which emerges, is measured using a suitable photodetector. It is possible for the tissue to be directly transilluminated where the light source, usually in the region of 800 nm to 960 nm, is on one side of the tissue and the detector on the other side. This mode, also called the *transmission mode*, is limited to areas such as the finger, ear lobe or toe (Nijboer *et al* 1981, Mendelson and Ochs 1988). However, when light is directed down into the skin a proportion of the light 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 mode, also called the *reflection mode*, allows measurements on virtually any skin area (Nijboer *et al* 1981, Mendelson and Ochs 1988).

The intensity of the 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 (Nijboer *et al* 1981, Roberts 1982). These variations are amplified and recorded as a voltage signal called the photoplethysmograph (PPG).

The PPG signal is divided into two components: a dc PPG component, a relatively constant voltage whose magnitude is determined by the nature of the material through which the light passes (skin, cartilage, venous blood, etc) and a pulsatile or ac PPG component synchronous with the heart rate which is 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% to 2% of the dc value (Webster 1997).

Photoplethysmography is used in the estimation of arterial oxygen saturation by pulse oximetry (SpO_2). Pulse oximeters estimate arterial oxygen saturation non-invasively by shining light at two different wavelengths, red and near infrared, through vascular tissue. The pulsatile photoplethysmographic (ac PPG) signal associated with cardiac contraction, described above, 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 (Webster 1997). From the ratios of these amplitudes and the corresponding dc PPG components, arterial blood oxygen saturation is estimated. Hence, the technique of pulse oximetry relies on the presence of adequate peripheral arterial pulsations, which are detected as PPG signals (Mendelson and Ochs 1988). 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. The oxygenation readings become unreliable in these circumstances because conventional pulse oximeter transmission sensors are usually placed on the most peripheral

parts of the body such as the finger, ear lobe or toe, where pulsatile flow is most vulnerable, as it is compromised by diversion of blood flow to more vital organs. Reflection pulse oximeter sensors, which have been introduced relatively recently, enable the measurement of oxygen saturation in more central parts of the body such as the forehead and nose, however these sites have proved disappointing as they give no improvement in poorly perfused patients (Rosenberg and Pedersen 1990, Clayton *et al* 1991).

Hence, pulse oximetry becomes unreliable in a significant group of patients at just the time when the measurement of blood oxygen saturation (SpO_2) would be clinically of most value. In particular, it can fail in patients undergoing prolonged procedures such as cardiac, vascular, reconstructive or neuro-surgery. There is, therefore, a need to find a means of solving this frustrating and serious clinical problem. Clinical studies on healthy anaesthetized patients using an earlier prototype system comprising an oesophageal PPG probe and a PPG processing system have shown that measurable PPG signals with large amplitudes and good signal-to-noise ratio at two wavelengths (red and infrared) could be detected from the entire length of the oesophagus (Kyriacou *et al* 1999, 2001). This paper describes the development of a new PPG and SpO_2 processing system, which was used for the investigation and comparison of oesophageal and finger PPGs and SpO_2s in patients undergoing high-risk operations, such as hypothermic cardiothoracic bypass surgery, in whom conventional pulse oximetry might fail due to poor peripheral circulation. This study on cardiac patients also provided data with which to address a fundamental question posed at the beginning of the investigation: are the detected oesophageal signals due to pulsatile blood flow in the arteries or are they partly or wholly a cardiac movement artefact?

2. Methods

2.1. Instrumentation and software

A reflectance oesophageal PPG probe was constructed utilizing miniaturized opto-electronic devices, two red (660 nm) and two infrared (880 nm) emitters and a photodetector (Kyriacou *et al* 1999). The probe was designed to fit into a transparent oesophageal stomach tube (French gauge 20). A finger reflectance PPG probe, optically and electronically identical to the oesophageal PPG probe, was also constructed to facilitate comparisons between the two sites (oesophagus and finger). An electrically isolated, time-multiplexed PPG processing system was developed to detect and pre-process simultaneously the red and infrared ac and dc PPG output signals. The processing system also incorporated a 3-lead ECG channel which was used as a timing reference for the oesophageal and finger PPG signals. Oesophageal and finger time-multiplexed ac and dc PPG traces (obtained at red and infrared wavelengths) together with ECG traces, were digitized by a 16-bit data acquisition card (National Instruments Corporation, Austin, TX). The digitized PPG and ECG signals were further analysed by a *Virtual Instrument* implemented in *LabVIEW* (National Instruments Corporation, Austin, TX). Oesophageal and finger PPG signals and ECG traces were displayed simultaneously on a laptop computer. The design of the *Virtual Instrument* incorporated algorithms allowing the online estimation of oesophageal and finger SpO_2 using the ratio of ratios method (Webster 1997). A general block diagram of the processing system is shown in figure 1.

2.2. Patients and measurements

Local research ethics committee approval was obtained prior to commencing the study of 50 adult patients (41 males and 9 females) aged 26 to 81 years undergoing elective thoracic

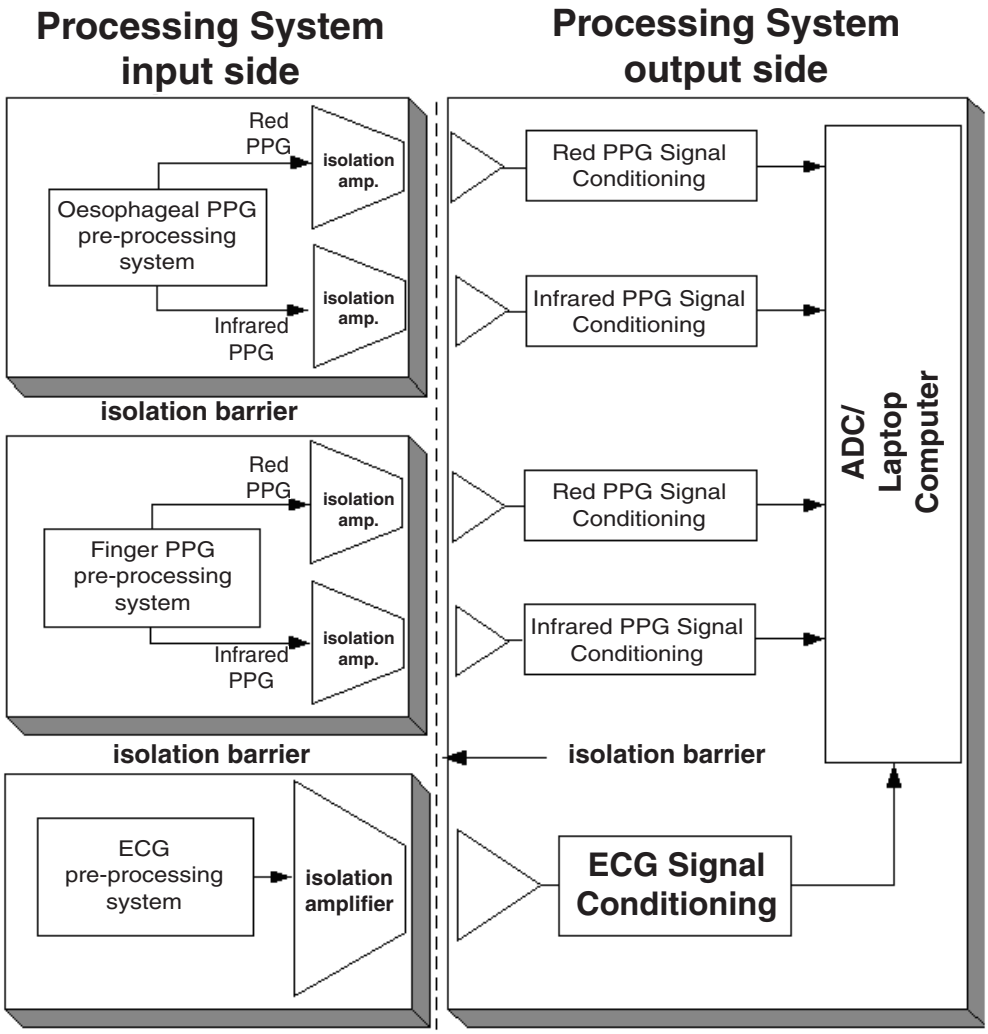


Figure 1. Block diagram of the processing system.

surgery. Anaesthesia was induced with midazolam and etomidate followed by a dose of rocuronium or pancuronium. Fentanyl was used for analgesia. The trachea was intubated and the lungs were mechanically ventilated. Heart rate, systolic and diastolic blood pressures were monitored continuously. Central temperature was measured from the nasopharynx and peripheral temperature from the left shoulder tip. The reflectance custom-made finger probe was placed on the index finger of the patient. A commercially available transmission type pulse oximeter probe (Marquette Tram 200A; Marquette Electronics, Milwaukee, WI) was also used on an adjacent finger to record finger SpO_2. The oesophageal PPG probe was inserted into a sealed 20 French gauge stomach tube. The tube, lubricated with aqueous gel, was then inserted through the mouth into the oesophagus under direct vision. The stomach tube was advanced into the oesophagus until the end of the probe inside the tube was 30 cm from the lips. After placement of the monitoring lines and the establishment of cardiovascular stability, PPG signals were observed at various depths in the oesophagus as the probe was withdrawn,

until the site which gave the best signal (high signal-to-noise ratio and small ventilator artefact) was determined. At this time, oesophageal ac PPG signals were collected for all 50 patients. These ac PPG amplitudes were later measured manually on printouts from *LabVIEW*, and the means and standard deviations (SD) calculated. PPG traces and SpO_2 values from the oesophagus and finger, along with ECG traces, were recorded simultaneously. Monitoring in theatre was intermittent. Oesophageal, finger and ECG data were collected for at least 10 to 15 min prior to skin incision. After sternotomy, signals were recorded for approximately 15 min. Monitoring continued for 10 to 15 min prior to bypass. PPG signals were recorded until cessation of cardiac activity on bypass on the heart–lung machine, and when no pulsatile signals were seen on the screen of the laptop. The monitoring started again approximately 10 min before coming off bypass and continued for another 30 min until stable cardiac activity was established. The recording of signals in theatre continued during the closure of the chest for approximately 15 to 20 min. The last monitoring period took place in the intensive care unit where the patient was monitored continuously for approximately 30 to 60 min. During this period the patients were still peripherally cold and therefore potentially at risk of peripheral pulse oximeter failure.

In order to investigate the possible effect of cardiac movement on the PPG signals, observations were made during cross-clamping of the aorta when pulsatile blood flow ceased despite ongoing cardiac movement. Signals were also recorded from two patients with no cardiac activity whilst a pulsatile flow cardiopulmonary bypass machine was in use.

During the above recording periods, samples of arterial blood were drawn into 2 ml heparinized syringes and analysed immediately by an Instrumentation Laboratories IL BG-1400 blood gas analyser (BGA) (Instrumentation Laboratories, Lexington, MA, USA).

2.3. Data analysis and statistics

Patients were accepted into the PPG amplitude analysis if measurable PPGs were present at both wavelengths in the oesophagus. Also, patients were only accepted into the SpO_2 statistical analysis if for every blood oxygen saturation value obtained by blood gas analysis, there were simultaneous SpO_2 values from the oesophageal, custom finger and commercial finger pulse oximeters. Oesophageal ac PPG signals were collected for all 50 patients during a period of cardiovascular stability and after the placement of monitoring lines. Linear regression analysis was used to compare the blood oxygen saturation results from the oesophageal pulse oximeter with arterial blood oxygen saturation (SaO_2) values obtained by blood gas analysis. The limits of agreement between the oesophageal and blood gas analysis results were calculated using the *between-method differences* analysis (Altman and Bland 1983).

3. Results

3.1. Results from the investigation of PPG signals in cardiac patients

Measurable PPG traces at red and infrared wavelengths were obtained in the oesophagus in all 50 patients. Figure 2 depicts typical traces from one patient undergoing cardiopulmonary bypass surgery during the various monitoring periods as described above (probe depth 17 cm from the lips). Figure 2(a) shows oesophageal and finger ac PPGs, obtained at both wavelengths and ECG signals recorded prior to skin incision. The signals in figure 2(b) were recorded just before sternotomy. In figure 2(c) the signals were recorded after the chest was open. Figure 2(d) shows the transition from before bypass to being on cardiopulmonary bypass. When the heart–lung machine was switched on (indicated in the figure as ‘on bypass’) the

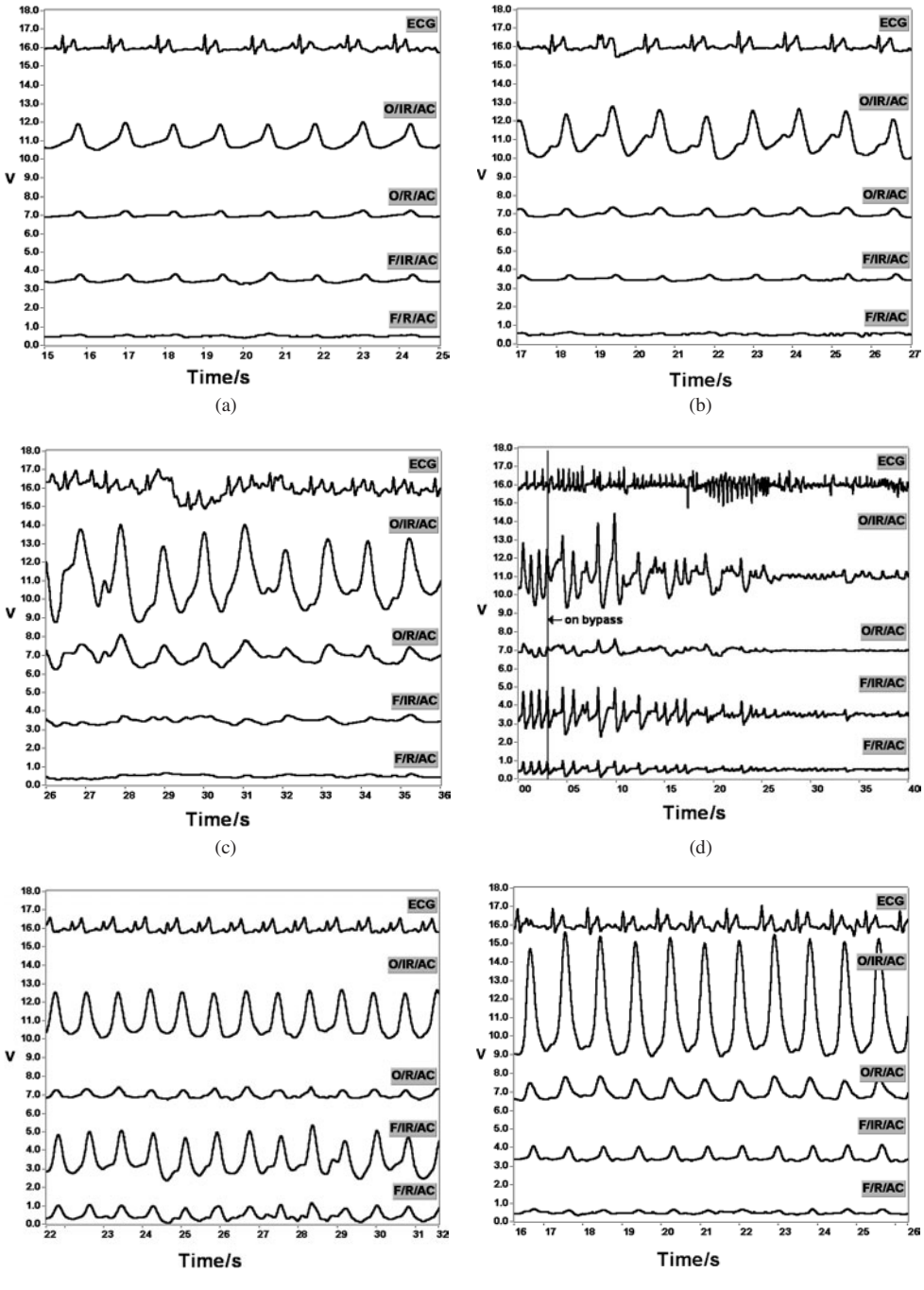


Figure 2. Oesophageal, finger and ECG traces obtained from an anaesthetized patient undergoing cardiopulmonary bypass surgery (probe depth 17 cm from lips): (a) prior to skin incision; (b) in operating theatre before sternotomy; (c) after sternotomy; (d) during bypass transition (before bypass, on bypass); (e) closing the chest; (f) in intensive care unit. O/IR/AC: oesophageal infrared ac PPG, O/R/AC: oesophageal red ac PPG, F/IR/AC: finger infrared ac PPG, F/R/AC: finger red ac PPG.

Table 1. Mean ± SD of ac peak-to-peak PPG amplitudes (mV) at two wavelengths measured in the upper, mid and lower oesophagus prior to skin incision.

	Mean ac PPG amplitudes		
	Upper oesophagus (14 cm to 17 cm)	Mid oesophagus (18 cm to 22 cm)	Lower oesophagus (27 cm to 28 cm)
Infrared (880 nm)	177 ± 94 mV	532 ± 316 mV	358 ± 84 mV
Red (655 nm)	69 ± 37 mV	222 ± 125 mV	183 ± 73 mV
Number of patients (*n*)	27	19	4

pulsatile PPG signals disappeared within the next 20 to 25 s. The ECG trace on bypass shows a variable high frequency activity as expected. Figures 2(e) and (f) show PPG and ECG signals after bypass, during closing of the chest and post-operatively in the intensive care unit, respectively.

The oesophageal PPG signals recorded from all patients (before and after bypass) were of high signal-to-noise ratio and large amplitudes. The chosen oesophageal monitoring depths ranged from 14 cm to 28 cm, measured from the upper lip (mean ± SD: 17.8 ± 3.3 cm).

The optimal oesophageal monitoring depth for each patient was considered to be the depth at which oesophageal PPGs with good signal-to-noise ratio and acceptable ventilator artefact (synchronous modulation in the form of a sinusoidal baseline shift in time with the approximately 5 s period of the ventilatory cycle) could be obtained (Kyriacou *et al* 1999, 2001). The magnitude of the ventilator artefact at the depth range of 14 cm to 28 cm was of the order of 10% to 40% of the oesophageal PPG peak-to-peak amplitude. It was originally thought that with a ventilator artefact of that order it would be impossible to estimate continually and accurately oesophageal blood oxygen saturation values unless the ventilator was switched off temporarily. However, it was found that the algorithm used to estimate SpO_2 functioned reliably despite this level of ventilator artefact.

Table 1 gives the mean ± SD of the ac PPG amplitudes at both wavelengths at the different oesophageal monitoring depths for the 50 patients. The amplitudes at the monitoring depths in table 1 are grouped into three ranges: the upper oesophageal depths (14 cm to 17 cm), the mid oesophageal depths (18 cm to 22 cm) and the lower oesophageal depths (27 cm and 28 cm). The ac PPGs in the mid and lower oesophagus (depths of 18 cm or greater) had larger mean amplitudes at both wavelengths than those in the upper oesophagus (14 cm to 17 cm). These quantitative results were within the ranges of those obtained in an earlier PPG amplitude study at five oesophageal depths in healthy anaesthetized patients (Kyriacou *et al* 2001). The estimated error in these manual amplitude measurements is approximately ±3%. As may be seen from the SDs in table 1, there was considerable variability between patients in the PPG signal amplitudes at the various oesophageal depths.

3.2. Results from an investigation to determine the contribution of cardiac movement artefact to the oesophageal PPG signals

The study of the thoracic patients, especially the patients undergoing cardiac surgery ($n = 47$), where the heartbeat ceases and the blood is circulated by the heart–lung machine, confirmed that the oesophageal PPG signals were due to arterial pulsatile blood and not cardiac movement artefact.

3.2.1. Investigation of oesophageal PPG signals during aortic cross-clamping. In order to establish the patient's circulation on bypass, the venous blood returning to the right side of the heart is diverted to the heart–lung machine and returned to the aorta beyond a cross-clamp. This clamp is required to prevent retrograde flow to the coronary arteries (important for coronary artery bypass grafting) and regurgitant flow to the left ventricle (important for open heart surgery). On clamping the aorta, all pulsatile blood flow from the heart to the aorta ceases, even if the heart is still beating. Indeed, cardiac movement does persist temporarily after aortic cross-clamping, as was confirmed by direct observation of the heart and the associated electrical activity recorded on the ECG (see figure 3(a)). For most cases, the heart–lung machine was of the continuous flow type such that the circulation was not pulsatile in the arterial system. Immediately on cross-clamping, the pulsatile oesophageal PPG signals terminated, and the PPG traces recorded on the screen of the laptop became flat despite the continuing cardiac movement (see figure 3(b)). This is strong evidence that the oesophageal PPG signals are not cardiac movement artefacts. This observation was common to all the patients undergoing continuous flow cardiopulmonary bypass surgery.

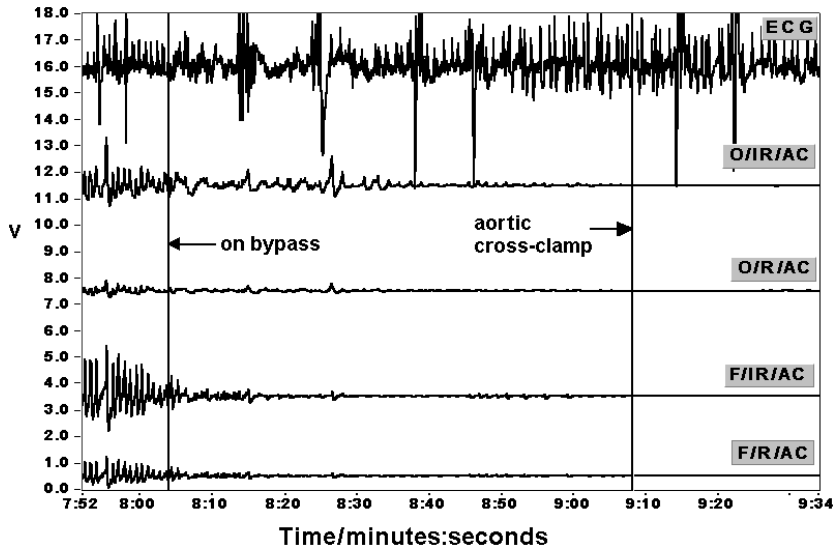
3.2.2. Investigation of oesophageal PPG signals during pulsatile flow bypass. The second investigation was performed on two patients undergoing cardiopulmonary bypass surgery, with the additional modality of pulsatile flow. In the pulsatile flow mode the machine imitates the heart by pulsing the blood in the arterial system. Both of the patients went on bypass using the continuous blood flow mode. Figure 4 illustrates the transition between continuous flow and pulsatile flow of the heart–lung machine (the blood flow modes can be changed during the operation). During the continuous flow bypass (after aortic cross-clamping) there was no heart activity and no ECG or pulsatile PPG signals (see figure 4).

When the machine was switched to pulsatile flow mode at a pulsing rate of 60 pulses per minute, pulsatile PPGs (oesophageal and finger) appeared on the screen of the laptop (see figure 4). That again confirmed that the morphology of the oesophageal PPG signals was due to the pulsatile property of the arterial blood coming from the pulsatile flow heart–lung machine and not due to cardiac movement (since the heart was completely stopped, note the flat ECG trace in figure 4).

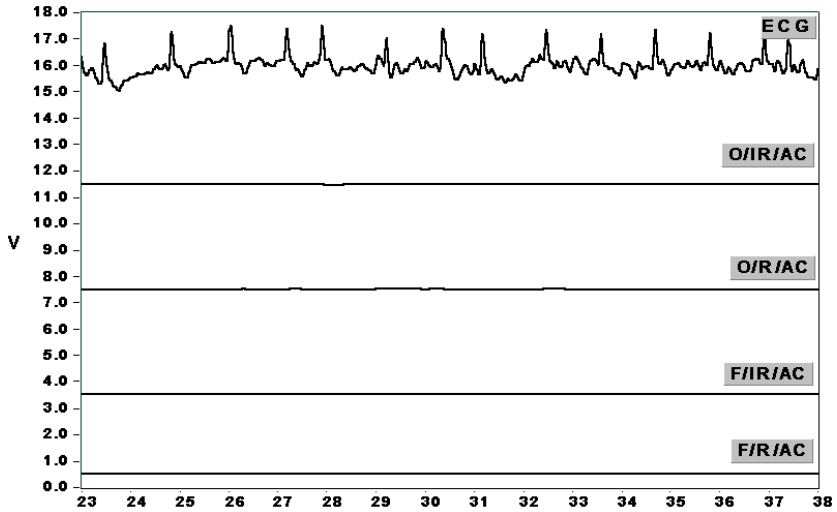
3.2.3. Evidence from the quantitative estimations of oesophageal SpO_2. The accuracy of the oesophageal SpO_2 values measured in 49 of the 50 patients (blood oxygen measurements were not made in one patient) was another indication that the oesophageal PPGs were not the result of cardiac movement but were due to arterial blood pulsations. The SpO_2 values obtained from the oesophagus are discussed in detail in the following section.

3.3. Comparisons of blood oxygen saturation measurements from oesophageal pulse oximetry with values from blood gas analysis

A total of 155 sets of data points from 49 patients were used for the regression analysis. Two to five arterial blood samples were collected from each patient (one before and one post-bypass in all patients), with further samples in procedures of longer duration, or in the event of the commercial pulse oximeter indicating hypoxaemia. A plot of SpO_2 readings obtained from the reflectance oesophageal pulse oximeter against values of SaO_2 from blood gas analysis is shown in figure 5. The equation of the best fit linear regression line was: (oesophageal SpO_2) = $12.32 + 0.875SaO_2$; $r^2 = 0.74$; standard error of estimate (SEE) = 0.86%; $p < 0.001$.



(a)



(b)

Figure 3. Oesophageal and finger PPG signals during aortic cross-clamping: (a) illustrates the on-bypass and aortic cross-clamp monitoring times; (b) after aortic cross-clamping, the ECG shows clearly heart activity but there are no pulsatile oesophageal or finger PPG signals. O/IR/AC: oesophageal infrared ac PPG, O/R/AC: oesophageal red ac PPG, F/IR/AC: finger infrared ac PPG, F/R/AC: finger red ac PPG.

The mean ($\pm$ SD) of the differences between the oesophageal pulse oximeter SpO_2 readings and those from blood gas analysis was $0.02 \pm 0.88\%$. The limits of agreement (mean difference $\pm$ 2SD) were 1.8% to –1.8% (Altman and Bland 1983). The estimated error in the calculated value of oesophageal SpO_2 due to ventilator artefact was less than $\pm 1.5\%$.

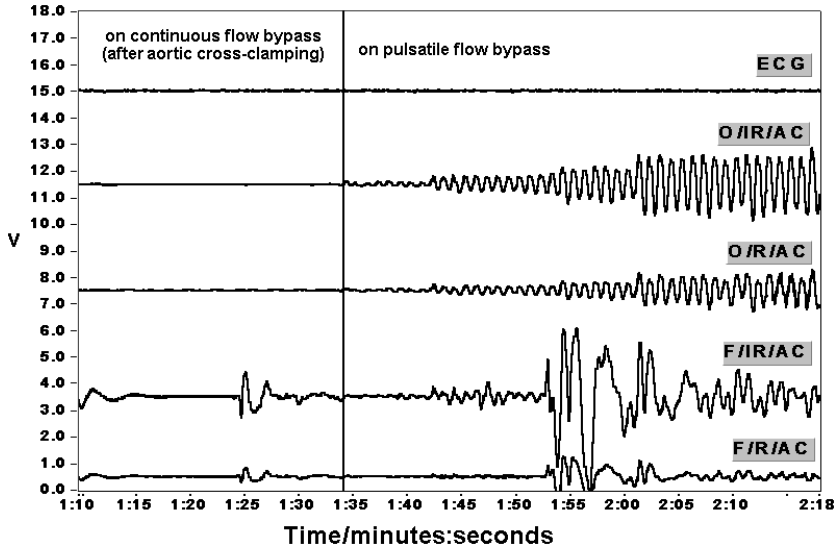


Figure 4. PPG signals during cardiopulmonary bypass surgery with the heart–lung machine switched from continuous flow bypass to pulsatile flow bypass (measurements performed after aortic cross-clamping). O/IR/AC: oesophageal infrared ac PPG, O/R/AC: oesophageal red ac PPG, F/IR/AC: finger infrared ac PPG, F/R/AC: finger red ac PPG.

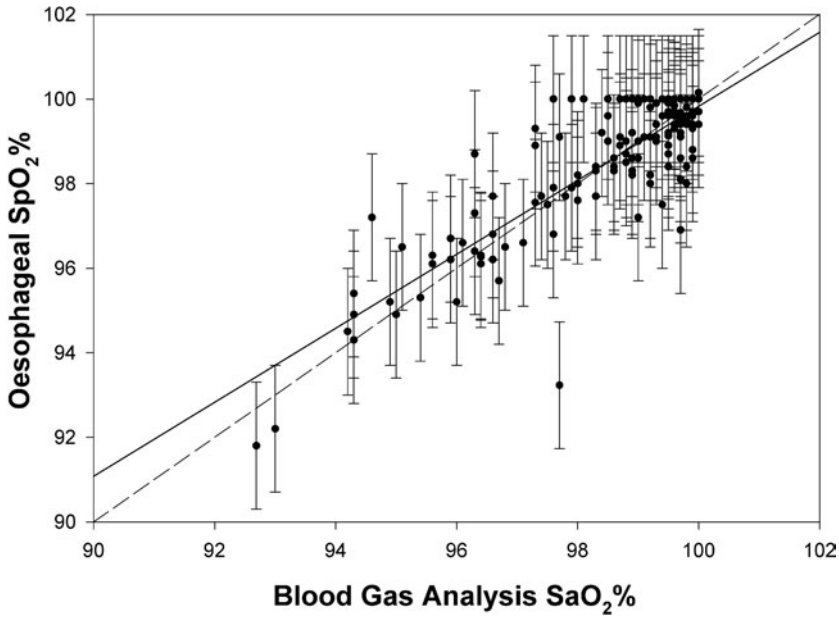


Figure 5. Plot of SpO_2 measurements obtained from the oesophageal probe against SaO_2 from the BGA in 49 patients. The solid line represents the best fit linear regression line. (Oesophageal SpO_2) = 12.32 + 0.875SaO_2; $r^2 = 0.74$; SEE = 0.86%; $n = 155$; $p < 0.001$. The dashed line represents identity. The error bars represent oesophageal SpO_2 error of $\pm 1.5\%$.

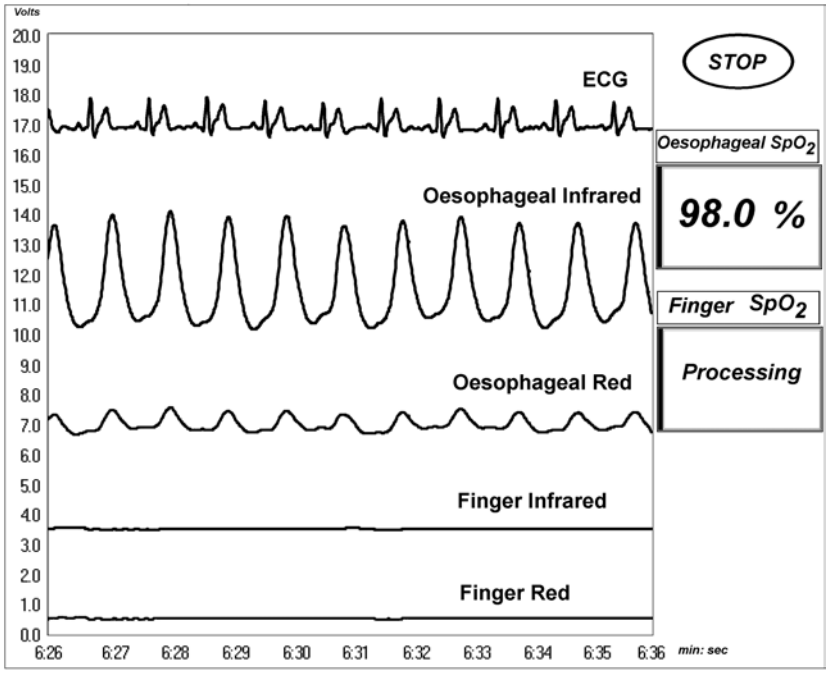


Figure 6. Typical PPG traces obtained from a cardiopulmonary bypass patient during times of finger pulse oximeter probe failure.

3.4. Patients in whom peripheral pulse oximetry failed

Five of the 49 patients (10.2%) had periods of at least 10 min (continuous) duration when both finger pulse oximeters (commercial and custom-made) failed to record pulsatile PPG signals and to estimate SpO_2 values. The oesophageal pulse oximeter operated successfully during the periods that the finger probes failed. Figure 6 shows typical PPG traces obtained from the oesophageal and the custom-made finger probes during peripheral (finger) pulse oximetry failure. The oesophageal PPG traces obtained at both wavelengths were of high signal-to-noise ratio and large amplitude. An oesophageal SpO_2 was estimated at all times without any difficulty.

The five patients in whom the peripheral pulse oximetry failure occurred were all cardiac patients undergoing cardiothoracic bypass surgical procedures. In four of the patients the finger pulse oximeters failed post-operatively (within the first half hour after completion of the surgery) in the intensive care unit and in the fifth patient the failure occurred in the operating theatre before the patient went on bypass. The oesophageal monitoring depths for the five patients ranged from 15 cm to 22 cm, measured from the upper lip (15 cm (two patients), 16 cm (one patient), 17 cm (one patient), 22 cm (one patient)).

Table 2 gives the mean of the ac PPG amplitudes at both wavelengths at the different oesophageal monitoring depths for the five patients. The measured PPG amplitudes were separated into two groups according to the monitoring depths, the upper oesophageal depths (14 cm to 17 cm) and the mid oesophageal depths (18 cm to 22 cm).

During the time of the finger probe failure blood samples (a total of seven measurements for all five patients) were collected and blood gas analysis was performed. Oesophageal pulse oximetry SpO_2 values were also recorded at the same time as blood sampling. The mean

Table 2. Mean ± SD of ac peak-to-peak PPG amplitudes (mV) at two wavelengths measured in the upper and mid oesophagus of five patients in whom peripheral pulse oximetry failed.

	Mean ac PPG amplitudes	
	Upper oesophagus (14 cm to 17cm)	Mid oesophagus (18 cm to 22 cm)
Infrared (880 nm)	182 ± 120.4 mV	477 mV
Red (655 nm)	65 ± 27.3 mV	273 mV
Number of patients (*n*)	4	1

(± SD) of the differences between the SaO_2 values from blood gas analysis and SpO_2 from the oesophageal pulse oximeter was 0.0 ± 0.48%.

4. Discussion and conclusion

Oesophageal PPG signals with large amplitudes and high signal-to-noise ratios were measured at various depths within the oesophagus (ranging from the upper to the deep oesophagus) from all 50 cardiothoracic patients. The oesophageal PPG amplitudes were within the ranges previously measured from non-cardiac surgery patients (Kyriacou *et al* 2001). The upper to mid oesophagus proved to be the most appropriate depth for measuring blood oxygen saturation (see table 1). The main reason was that at these depths the magnitude of the ventilator artefact was less than 30% of the oesophageal PPG peak-to-peak amplitude, and did not have a significant effect on the estimation of oesophageal SpO_2.

The study on cardiac patients resolved the uncertainty over the contribution (partial or complete) of cardiac movement artefact to the oesophageal PPG signals. The presence or amount of this artefact had been in doubt since the beginning of the study. The investigation of oesophageal PPG signals during aortic cross-clamping showed that pulsatile oesophageal PPGs ceased although there was still cardiac activity (see figure 3). Also, the investigation of oesophageal PPGs during pulsatile flow bypass showed that pulsatile oesophageal PPG signals of arterial origin existed when the heart was completely stopped (see figure 4). However, as measurements were made on only two patients with pulsatile flow bypass further studies are needed to confirm this conclusion. The results of these two investigations, along with the good agreement between the oesophageal SpO_2 values and those from blood gas analysis (figure 5), showed that the oesophageal PPG signals are probably due entirely to pulsatile blood flow in arteries. If the oesophageal PPGs were merely artefact signals caused by the mechanical movement of the heart then the likelihood of obtaining saturation values that were in agreement with the BGA would be very small.

In addition, the oesophageal pulse oximeter was found to be reliable and accurate (as calibrated with blood gas analysis) in cases of poor peripheral perfusion (five patients) where both finger pulse oximeters failed to estimate oxygen saturation values for at least 10 min. Of the 49 cardiothoracic patients in the SpO_2 statistical analysis 10.2% had a finger pulse oximetry failure, which is in agreement with previously reported measurements (Reich *et al* 1996). Although the percentage failure rate in this study is only approximately 10%, in terms of absolute numbers this would represent a significant clinical problem, and a reliable means of monitoring throughout such a failure would be of real value. The mean oesophageal PPG amplitudes obtained from the five patients when the finger pulse oximetry failed (see table 2) were of the same order of magnitude as the mean PPG amplitudes in healthy anaesthetized

patients (Kyriacou *et al* 2001) and cardiothoracic patients (see table 1). These results on the five patients from whom reliable oesophageal SpO_2 values were obtained while finger pulse oximetry failed, suggest that the arterial blood circulation to the oesophagus may be less subject to vasoconstriction and decreased PPG amplitudes than are the peripheral sites such as the finger. Only five patients exhibited this phenomenon in this study and, therefore, more work is needed to confirm these findings. This novel monitoring site, the oesophagus, may also be of value in patients who have burns or other serious injuries where the oesophagus may be the only available site for a pulse oximetry probe.

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