

Kyriacou, P. A., Powell, S., Langford, R. M. & Jones, D. P. (2002). Esophageal pulse oximetry utilizing reflectance photoplethysmography. *IEEE Transactions on Biomedical Engineering*, 49(11), pp. 1360-1368. doi: 10.1109/TBME.2002.804584



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**Original citation:** Kyriacou, P. A., Powell, S., Langford, R. M. & Jones, D. P. (2002). Esophageal pulse oximetry utilizing reflectance photoplethysmography. *IEEE Transactions on Biomedical Engineering*, 49(11), pp. 1360-1368. doi: 10.1109/TBME.2002.804584

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# Esophageal Pulse Oximetry Utilizing Reflectance Photoplethysmography

Panayiotis A. Kyriacou\*, *Member IEEE*, Sarah Powell, Richard M. Langford, and Deric P. Jones, *Member IEEE*

**Abstract**—Peripheral perfusion is often poor and barely pulsatile in patients undergoing prolonged major surgery. Hence, the arterial blood oxygen saturation (SpO<sub>2</sub>) readings from commercial finger pulse oximeters can become unreliable or cease when they are most needed. To overcome this limitation, the esophagus has been investigated as an alternative measurement site, as perfusion may be preferentially preserved centrally. A reflectance esophageal pulse oximeter probe, and a processing system implemented in *LabVIEW* were developed. The system was evaluated in clinical measurements on 49 cardiothoracic surgery patients. The SpO<sub>2</sub> values from the esophagus were in good agreement with arterial blood oxygen saturation (SaO<sub>2</sub>) values obtained from blood gas analysis and CO-oximetry. The means ( $\pm$ SD) of the differences between the esophageal SpO<sub>2</sub> and SaO<sub>2</sub> results from blood gas analysis and CO-oximetry were  $0.02 \pm 0.88\%$  and  $-0.73 \pm 0.72\%$ , respectively. In five (10.2%) of the patients, the finger pulse oximeter failed for at least 10 min while the esophageal SpO<sub>2</sub> readings remained reliable. The results confirm that the esophagus may be used as an alternative monitoring site for pulse oximetry even in patients with compromised peripheral perfusion.

**Index Terms**—Esophagus, pulse oximetry.

## I. INTRODUCTION

**P**ULSE oximeters are noninvasive, easy to use, and readily available. Pulse oximetry provides information about the arterial blood oxygen saturation and heart rate, and has widespread clinical applications. The use of pulse oximetry has been described in many settings: hospital, outpatient, domiciliary use and in veterinary clinics. In the early 1990s, pulse oximetry became a mandated international standard for monitoring during anesthesia following the publication in 1986 of the Harvard minimum standards for monitoring. Kelleher [1] reviewed 220 references in a review published in 1989. In a follow-up review in 1992, Severinghaus and Kelleher [2] found more than 500 new reports between 1989 and October 1991. Nearly 2000 further reports have been published since October 1991.

Although generally reliable, pulse oximeters do fail and many of their limitations, both physiological (abnormal hemoglobins, other absorbants, anemia, skin pigmentation) and

technical (signal artifact, electromagnetic interference) have been described [3]. Pulse oximeters require adequate peripheral perfusion to give accurate results, and they have been reported to fail in patients with compromised peripheral perfusion [4]–[6]. Pulse oximetry is a pulse dependent technique, and any significant reduction in the amplitude of the pulsatile component of the photoplethysmographic (PPG) signal detected by the oximeter can lead to inaccurate values for blood oxygen saturation (SpO<sub>2</sub>) or complete failure. When peripheral perfusion is poor, as in states of hypovolemia, hypothermia, and vasoconstriction, oxygenation readings become unreliable or cease [7]–[9]. Such clinical situations occur, for example, after prolonged operations, especially hypothermic cardiopulmonary bypass surgery. The problem arises because conventional transmission pulse oximetry sensors must be attached to peripheral parts of the body, such as a finger, ear or toe, where pulsatile flow is most easily compromised. Measurements at sites other than the finger or ear, such as the forehead and nose, give no improvement in patients with poor peripheral perfusion [10], [11]. Thus, SpO<sub>2</sub> readings may be unobtainable at just the time when they would be most valuable.

To avoid the difficulties associated with conventional measurements of arterial blood oxygen saturation during conditions of poor peripheral perfusion and pulsation, it has been proposed that the upper esophagus be used as a measurement site. Atlee and Bratanow [12] presented results of blood oxygen saturation measurements obtained at the cricopharyngeal muscle in the upper esophagus ( $14 \pm 1$  cm from the incisors) using a “transoesophageal” probe where the optical components of the oximetry sensor were incorporated into a traditional anesthesia esophageal stethoscope. The results showed that the “transoesophageal” probe underestimated or overestimated SpO<sub>2</sub> values depending on the geometry of the sensor [13], [14]. In another clinical study, Prielipp *et al.* [15] compared this “transoesophageal” probe with two conventional finger pulse oximeter probes. They concluded that the “transoesophageal” probe did not perform well as the standard finger oximetry monitors.

The investigation of PPG signals within the whole depth of the esophagus has been suggested [16] in an attempt to overcome the limitations of measurements at the cricopharyngeal muscle. The use of a more central monitoring site should increase the likelihood of obtaining reliable oxygen saturation readings even during periods of compromised peripheral perfusion. Earlier preliminary studies on healthy anesthetized patients categorized ASA 1 (American Standard of Anesthesiology; scale 1–5, with five the most critically ill patient) using a prototype system showed that measurable PPG

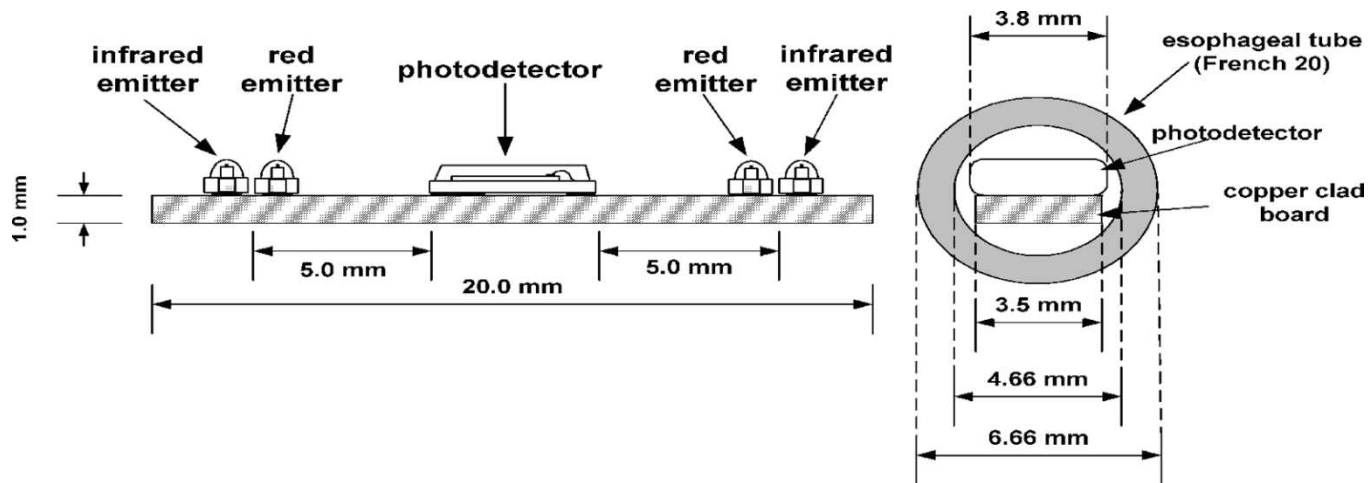


Fig. 1. Side and cross sectional views of the esophageal pulse oximetry probe. In the cross-sectional view, the probe is shown inserted in the esophageal tube.

signals with large amplitudes and good signal-to-noise ratio (SNR) at two wavelengths [red (R) and infrared (IR)] could be detected from the entire length of the esophagus [17], [16], [18]. In the present study, a new esophageal pulse oximetry processing system which allows the online estimation of esophageal  $SpO_2$  was developed to investigate patients (ASA 2, 3, 4) undergoing high-risk operations, such as hypothermic cardiopulmonary bypass surgery. Cardiopulmonary bypass patients have an arterial cannula inserted routinely, thereby facilitating rigorous validation of the esophageal  $SpO_2$  measurements with CO-oximetry and blood gas analysis. There appears to have been no previous report of measurements of  $SpO_2$  in the esophagus below the cricopharyngeous muscle.

## II. METHODS

### A. Esophageal Pulse Oximeter Probe

A reflectance esophageal pulse oximeter probe was constructed. This new probe comprised two (IR) and two (R) surface mount emitters and a surface mount photodetector (Fig. 1). The photodetector detected radiation back scattered by the tissue from both IR and R emitters and gave an output current proportional to the detected radiation intensity. A six-core cable carried the power to the IR and R emitters in the probe from the main photoplethysmography (PPG) processing unit and also the PPG signal currents from the photodetector (Fig. 1).

1) *Optical Components:* The IR and R emitters used were ceramic chip surface mount types (dimensions of each: 3.2 mm  $\times$  1.27 mm) with peak emission wavelengths at 880 and 655 nm, respectively (ELCOS GmbH). The photodetector was a surface mount silicon PhotoPinDiode on a ceramic contact base (dimensions: 4.57 mm  $\times$  3.81 mm) (ELCOS GmbH).

2) *Mechanical Construction of the Esophageal Pulse Oximetry Probe:* The photodetector was mounted between the R and IR emitters to detect radiation intensity back scattered by the tissue (Fig. 1). The emitter and photodiode chips were mounted on the copper side of an epoxy glass copper clad single sided eurocard (dimensions: 20 mm  $\times$  3.5 mm  $\times$  1.0

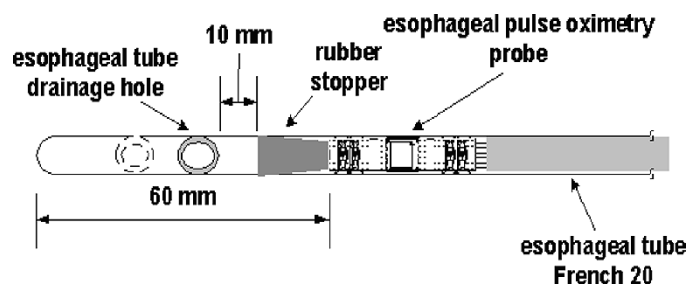


Fig. 2. Final position of the rubber stopper in the esophageal tube with the esophageal pulse oximeter probe *in situ*.

mm). Low-temperature (200 °C to 250 °C) soldering was used for connecting the surface mount components to the copper tracks of the board. Insulated wrapping wire (30 AWG) was used for the interconnection of the optical components.

The esophageal probe with attached cable was designed to fit into a plastic transparent disposable stomach/esophageal tube (Figs. 1 and 2) (Pennine Healthcare, Derby, U.K.). The esophageal tube used was a size 20 French gauge (external diameter: 6.66 mm; internal diameter: 4.66 mm; length: 780 mm, without X-ray detectable line) mainly used for gastric lavage (washout) or other gastric surgical procedures. The tube had a closed end with two side drainage holes. The esophageal tube was sealed at the bottom to prevent gastric juices or esophageal mucous reaching the electronic and optical components of the probe during clinical measurements. The sealing of the tube was achieved using a solid rubber stopper (dimensions: upper diameter: 6 mm; lower diameter: 4 mm; length: 14 mm) (Fisher Scientific, Loughborough, U.K.). The rubber stopper was pushed from the upper drainage hole of the tube toward the top of the tube (Fig. 2), until the bottom end of the rubber stopper was situated approximately 1 cm above the upper drainage hole of the tube. The upper diameter of the rubber stopper is greater than the internal diameter of the tube (diameter difference: 1.34 mm), therefore, when pushed into the tube a tight contact with the plastic was achieved, which kept it firmly in position. In its final position, the end of the esophageal probe was approximately 60 mm from the distal end of the stomach tube (Fig. 2).

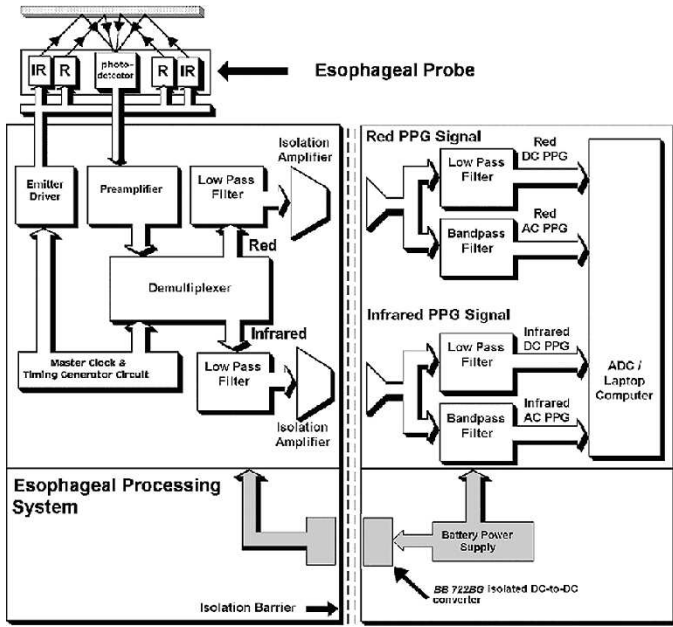


Fig. 3. Detailed block diagram of the esophageal processing system.

### B. Processing System Hardware

A processing system was constructed to preprocess, record and display esophageal PPG signals on a laptop personal computer (Fig. 3). To minimize the risk to the patient from the electrical hazard associated with the accidental or unintended mains power up of the laptop computer, the input (patient side) and output (monitoring side) circuits of the system were isolated. The input side (patient side) of the processing system was electrically isolated from the output side (monitoring side) using two Burr-Brown ISO122 analog isolation amplifiers and a Burr-Brown 722BG isolated dc-to-dc converter. Esophageal time multiplexed ac and dc PPG traces (obtained at R and IR wavelengths) were processed by the system and digitized by a 16-bit data acquisition card. A detailed block diagram of the processing system is shown in Fig. 3.

1) *Input Side Circuitry of the Processing System:* The master clock and timing generator circuit (Fig. 3) of the processing system comprised a crystal oscillator, a frequency divider, and a ring shift counter. It was used to generate the timing signals for switching (ON/OFF) the R and IR emitters. These timing control signals were also used for synchronizing the demultiplexer that separated the mixed (R and IR) PPG signals at the output of the preamplifier circuit into R and IR PPG signals. The emitters were driven by a pair of identical constant current sources one for each wavelength. Analog switches (MC14053) were used to time multiplex the R and IR emitters at 75 Hz (each emitter is switched on every 13.33 ms for an interval of 3.33 ms). The constant current circuits provided a total of 40 mA of pulse current to each pair of emitters in parallel. The R and IR emitters were never on at the same time, although during part of the emitter switching cycle they were both off to allow the photodetector to detect ambient light. The duty cycle was 25%. The R emitters were on for the first quarter cycle, then all emitters were off for the second quarter cycle. The IR emitters were on for the third

quarter cycle, and all were again off for the final quarter cycle. The effect of ambient light could have been estimated during the off periods and used to correct the results, although it was deemed unnecessary as the esophageal pulse oximetry probe was in “total darkness,” in the collapsed tube of the esophagus.

The preamplifier circuit (Fig. 3) consisted of a transimpedance (current-to-voltage) amplifier and an inverting amplifier. The transimpedance amplifier converted the photodiode current into a mixed signal voltage containing R and IR PPGs. The negative going output signal of the transimpedance amplifier was then inverted by a unity gain inverting amplifier. The mixed PPG signal from the output of the inverting amplifier was then fed into a demultiplexer (MC14052) to separate the PPG signals into two independent channels (R and IR). The inputs to the demultiplexer were the inverting amplifier output and the control timing signals from the master clock and timing generator circuit.

In order to eliminate the high-frequency (75 Hz) switching noise from the demultiplexer, caused by the timing control signals, the R and IR PPG signals were each low-pass filtered. The cutoff frequency was set at 40.2 Hz. The R and IR PPG signals were then passed through the isolation barrier of the analog isolation amplifier to the output side of the processing system.

2) *Isolation and Output Side Circuitry of the Processing System:* Two analog isolation amplifiers were used to pass the esophageal PPG signals (R and IR) from the input side to the output side of the processing system (Fig. 3). The isolation amplifiers used were Burr-Brown ISO122.

The IR and R PPG signals each consisted of a large amplitude dc PPG component and a small amplitude ac PPG component (approximately 1% to 2% of the dc component). These signals were split into independent channels (IR ac and dc and R ac and dc) using active filters (Fig. 3). The ac PPG components were extracted using two identical bandpass filters, one for the IR and one for the R wavelength. The bandpass filters used consisted of a first order high-pass filter, to block the dc PPG component, and a Butterworth two-pole low-pass active filter which attenuated high frequencies. The frequency response was 0.48–20 Hz (at –3 dB) with a gain of 1.58 in the passband. The dc PPG components for the R and IR wavelengths were extracted from the combined ac and dc PPG signals at the output of the isolation amplifiers using two identical unity gain two-pole, active low pass Butterworth filters with a cutoff frequency at 0.15 Hz (Fig. 3).

### C. Power Supplies for Processing System

The processing system used two heavy duty (+12 V) sealed lead–acid rechargeable (Yuasa NP2.1-12, 1.76 Ah) batteries [dimensions (each): 78 mm × 34 mm × 67 mm; weight (each): 0.90 kg]. The ±12-V output from the batteries was connected to the output side of the processing system (Fig. 3).

The power was distributed to the isolated input side of the processing system using a dual isolated dc-to-dc converter (Burr-Brown 722BG) (Fig. 3). The 722BG converted a single +12-V dc input to a pair of bipolar output voltages (±12 V) of the same magnitude as the input voltage. The converter was capable of providing a total maximum output current of 100 mA (per output) at rated voltage. The two bipolar output supplies of the

converter were connected in parallel for higher output current ( $2 \times 100$  mA).

#### D. Mechanical Construction of the Processing System

The printed circuit board (PCB) for the processing system and the two sealed lead–acid batteries were enclosed in a Eurocard black case (dimensions W, H, D: 247 mm  $\times$  102 mm  $\times$  220 mm). The esophageal pulse oximetry probe plugged into a six-pin DIN socket on the front panel. The four analog output channels (esophageal R and IR ac and dc PPGs) were taken from a 68-pin vertically mounted male connector on the back panel via a ribbon cable to a PCMCIA analog-to-digital card.

#### E. Software for Control and Analysis

All four PPG output signals were digitized by a 16-bit data acquisition card (DAQCard-AI-16XE-50, National Instruments Corporation, Austin, TX). The computer used for data acquisition, processing, displaying and data storage was a *Dell Latitude CPi D266XT* laptop personal computer.

The digitized PPG signals were analyzed by a *Virtual Instrument (VI)* implemented in *LabVIEW* (National Instruments Corporation, Austin, TX). This *VI* read the esophageal PPG data, converted them into a spreadsheet format and saved them into a file specified by the user and displayed the signals in real time on the screen of the laptop computer. Algorithms were also developed in the *VI* for the online estimation of esophageal SpO<sub>2</sub>.

1) *Analog Signal Acquisition:* The PPG signals were sampled at a rate of 100 samples per second for each of the four channels. The analog filters described in Section II-B with a maximum upper cutoff frequency of 20 Hz acted as anti-aliasing filters.

2) *Calculation of Ratio (R) and Estimation of Esophageal SpO<sub>2</sub>:* The algorithm used to estimate esophageal SpO<sub>2</sub> calculated the ratio (*R*) of the quotients of the ac and dc PPG amplitudes at the R (655 nm) and IR (880 nm) wavelengths

$$R(\text{ratio}) = \frac{ac_{655}/dc_{655}}{ac_{880}/dc_{880}}. \quad (1)$$

The ratio (*R*) was then used to compute the arterial oxygen saturation (SpO<sub>2</sub>) using [19]

$$\text{SpO}_2 = 110 - 25R. \quad (2)$$

This equation is a linear approximation to an empirical calibration curve established by measurements on a large group of healthy volunteers with arterial blood oxygen saturation (SaO<sub>2</sub>) values generally greater than 70% [19].

The *Virtual Instrument* algorithm calculated the amplitude of the pulsatile ac PPG signal at each of the two wavelengths by finding the difference between the maximum peak ( $ac_{\text{max}}$ ) and the minimum trough ( $ac_{\text{min}}$ ) of approximately two consecutive PPG cycles (over 2 s). The maximum dc PPG components for both wavelengths were also found and the ratio *R* calculated. A running mean over five consecutive ratio values was taken and this mean value of *R* was displayed on the front panel of the *VI*. The mean ratio value was then used in (2) to calculate SpO<sub>2</sub>. The value of SpO<sub>2</sub> was updated every 2 s and displayed on the front panel of the *VI*.

TABLE I  
PATIENT DETAILS

Number of Patients	Type of operation
35	Coronary Artery Bypass Graft (CABG) (continuous flow) (open chest)
2	CABG (pulsatile flow) (open chest)
7	Heart Valve Replacement (open chest)
2	Heart Valve Replacement + CABG (open chest)
1	Excision of Cervical Rib (closed chest)
1	Thoracoscopy (closed chest)
1	Thoracotomy (open chest)

#### F. Thermal Safety Tests

The emitters are thermally insulated from the tissue by the 1-mm-thick plastic wall of the esophageal tube and the operating current of the emitters is relatively low. However, temperature tests both *in vitro* and *in vivo*, previously reported, were conducted to confirm that temperature rises in the esophagus at the outside wall of the esophageal tube adjacent to the probe would not be of clinical significance [17].

### III. PATIENTS AND MEASUREMENTS

Local research ethics committee approval was obtained prior to commencing the study of ASA 2, 3, and 4 anesthetized patients. Forty-nine patients (40 male and 9 female) aged 26–81 years undergoing elective cardiothoracic surgery were recruited to the study. Table I summarizes the number of patients per type of operation. Patients with esophageal pathology or previous surgery to the esophagus or stomach were excluded.

The trachea was intubated and the lungs were mechanically ventilated using a Blease Manley MP3 ventilator. Ventilation mode was, therefore, intermittent positive pressure ventilation by a pressure generator ventilator. Intravenous access was established with a 14-gauge cannula in a forearm vein. A 20-gauge arterial cannula was placed in a radial artery to allow continuous blood pressure monitoring during induction of anesthesia. The right internal jugular vein was cannulated using a triple lumen cannula to allow central venous pressure monitoring. Central temperature was measured from the nasopharynx and peripheral temperature from the left shoulder tip. The esophageal pulse oximetry probe was inserted into a sealed 20 French gauge stomach tube, which was lubricated with aqueous gel. The tube was then inserted through the mouth into the esophagus under direct vision. The stomach tube was advanced into the esophagus until the end of the probe itself was 30 cm from the lips, as indicated by a 30-cm mark previously positioned on the plastic esophageal tube prior to insertion in the esophagus.

The probe was withdrawn slowly, and PPG signals were observed at various depths of the esophagus to determine the optimal measuring site at which reliable signals with high SNR were obtained. The probe was then left at this depth for the duration of surgery and PPG traces and SpO<sub>2</sub> values were recorded simultaneously. During the esophageal measurements, values of blood oxygen saturation from a commercial transmission type finger pulse oximeter (Marquette, Tram 200A; Marquette Electronics, Milwaukee, WI) were also recorded.

Esophageal pulse oximetry monitoring was temporarily stopped during transportation of the patient from the anesthetic induction room into the operating room. Monitoring in theater was intermittent. Esophageal data were collected before skin incision for a period of 15 min, after sternotomy for 15 min, prior to bypass for 15 min, after bypass for 45 min and postoperatively in the intensive care unit for up to 60 min.

During the above recording periods samples (approximately 1 ml) of arterial blood were drawn into 2-ml heparinised syringes and analyzed immediately by an Instrumentation Laboratories IL 482 CO-oximeter or an Instrumentation Laboratories IL BG-1400 Blood Gas Analyzer (BGA) (Instrumentation Laboratories, Lexington, MA) depending on availability in the clinical workplace. Calibration of the CO-oximeter was performed according to the manufacturer's instructions at weekly intervals. This calibration was a one-point calibration at a level of THb = 14.9 g/dL using IL CalDye. The blood gas analyzer executes a one-point calibration cycle every 30 min and after every sample. A two-point calibration is executed every 4 h. The PCO<sub>2</sub> and PO<sub>2</sub> electrodes are calibrated using certified gas standard mixtures. The pH electrode is calibrated using standardized reagents. In addition, three quality-control standard samples are measured daily to check the automated calibrations.

#### A. Data Analysis and Statistics

Patients were only accepted into the statistical analysis if for every esophageal SpO<sub>2</sub> value there was a corresponding CO-oximetry or blood gas analysis estimate of arterial blood oxygen saturation. The limits of agreement between the esophageal SpO<sub>2</sub> results and those from CO-oximetry and blood gas analysis, were calculated using the *between-method differences* analysis outlined by Altman and Bland [20], [21]. Bland and Altman suggest that the best way to compare two methods is to plot the difference between the methods against their mean. The level of agreement can be summarized by calculating the bias, estimated by the mean difference ( $d$ ), and the standard deviation of the differences ( $s$ ). Provided differences within  $d \pm 2s$  (limits of agreement) are not clinically significant, the two methods can be used interchangeably.

Linear Regression analysis was used to compare the blood oxygen saturation results from CO-oximetry and blood gas analysis with the R/R ratio  $R$  measured by the esophageal pulse oximeter.

### IV. RESULTS

#### A. Results From the Thermal Safety Tests

The rise in temperature at the outside surface of the esophageal tube in the *in vitro* tests was no more than 0.2 °C for both the R and IR emitters. In the *in vivo* tests, the rise in temperature at the outside surface of the esophageal tube was less than 0.7 °C for the R emitter and 0.6 °C for the IR emitter [17].

#### B. Results From the Investigation of Esophageal PPG Signals in Cardiac Patients

It was found possible to record good quality PPG signals from the esophagus in all patients. The measured effective SNR

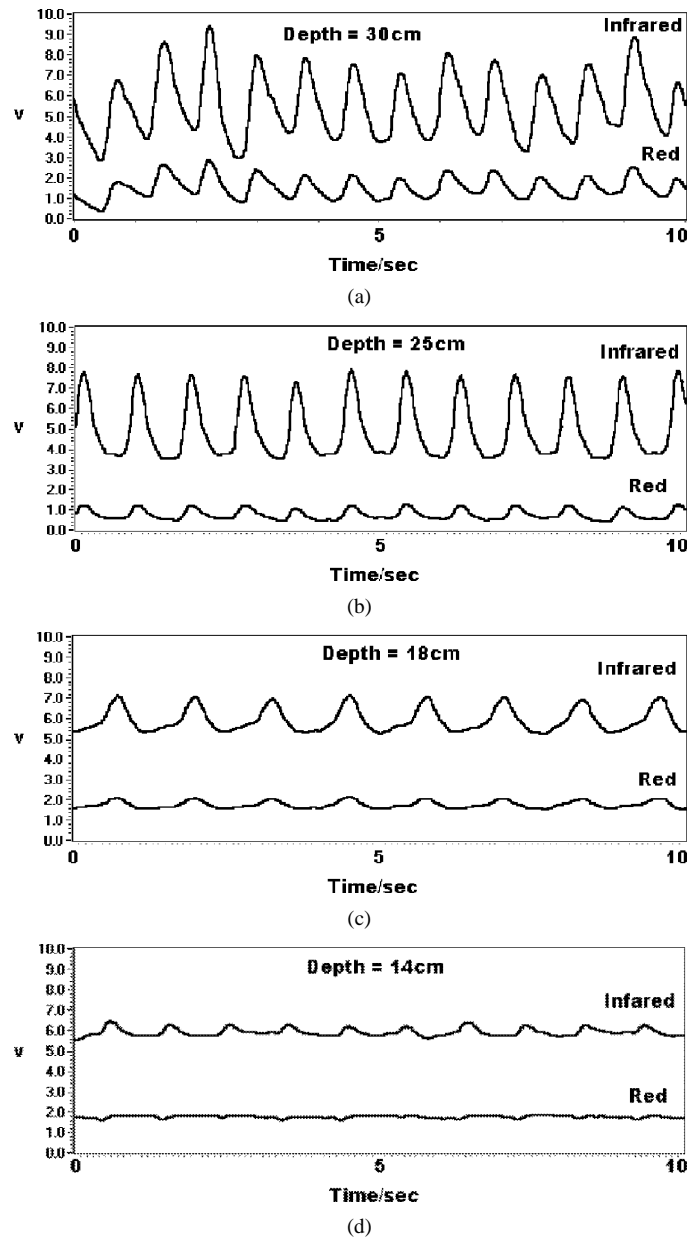


Fig. 4. Esophageal PPG traces at various esophageal depths [(a) 30, (b) 25, (c) 18, and (d) 14 cm] obtained from an anesthetized patient undergoing cardiopulmonary bypass surgery.

was always better than 40 dB at the output of the system. The amplitudes of the signals varied with depth in the esophagus. Fig. 4 depicts typical PPG traces from a CABG patient at various esophageal depths. As can be seen from Fig. 4 the amplitudes of the PPG signals in the upper esophagus (14 and 18 cm) are smaller than those in the lower esophagus (25 and 30 cm). This was the trend in all patients.

#### C. Comparisons of Blood Oxygen Saturation Measurements From the Esophageal Pulse Oximeter With Those From Blood Gas Analysis

A total of 155 pairs of blood oxygen saturation measurements were used for the *between-method differences* analysis [20], [21]. An average ( $\pm$ SD) of 3.5 ( $\pm$ 1.5) blood samples were collected from each patient.

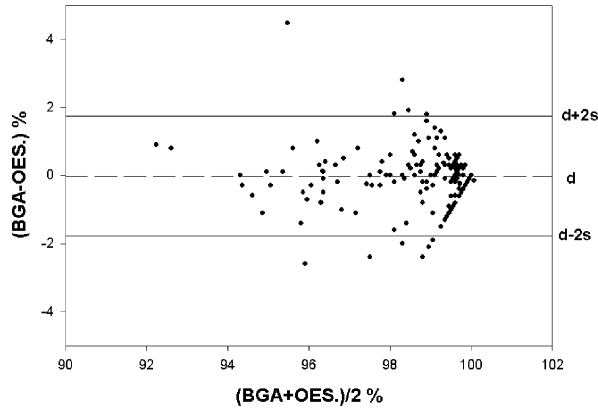


Fig. 5. The difference between blood oxygen saturation values from the blood gas analyzer and SpO<sub>2</sub> readings obtained from the reflectance esophageal pulse oximeter plotted against their mean for 49 patients.

A plot of the difference between blood oxygen saturation values from the blood gas analyzer and SpO<sub>2</sub> readings obtained from the reflectance esophageal pulse oximeter against their mean is shown in Fig. 5. The bias, estimated by the mean difference ( $d$ ), and the standard deviation of the differences ( $s$ ) give the degree of agreement between the two methods. The mean difference ( $d$ ) (blood gas analysis minus esophageal pulse oximeter) was  $-0.02\%$  and the standard deviation ( $s$ ) was  $0.9\%$ . The limits of agreement ( $d \pm 2s$ ) were

$$d - 2s = -0.02 - (2 * 0.9) = -1.8\%$$

$$d + 2s = -0.02 + (2 * 0.9) = 1.8\%.$$

A plot of oxygen saturation values from blood gas analysis against the  $R$  as measured by the esophageal pulse oximeter is shown in Fig. 6. The equation of the best fit linear regression line is:  $\text{SpO}_2 = 108.2 - 21.1R$ ;  $r^2 = 0.74$ ; standard error of the estimate (SEE) =  $0.84$ ;  $p < 0.001$ . The empirical calibration equation [see (2), Section II-E2] [19] used for the estimation of esophageal SpO<sub>2</sub> from measured values of  $R$  is given by the dashed line in Fig. 6.

#### D. Comparisons of Blood Oxygen Saturation Measurements From the Esophageal Pulse Oximeter With Those From CO-Oximetry

Although not available routinely for the majority of patients, CO-oximetry measurements were made on 17 patients. A total of 36 pairs of blood oxygen saturation measurements were used for a *between-method differences* analysis [20], [21]. An average ( $\pm$ SD) of  $2.1 (\pm 1.2)$  blood samples were collected from each patient. Fig. 7 shows a comparison of blood oxygen saturation values measured by the CO-oximeter and the esophageal pulse oximeter probe. The mean difference ( $d$ ) was  $-0.7\%$  and the standard deviation ( $s$ ) was  $0.7\%$ . The limits of agreement were

$$d - 2s = -0.7 - (2 * 0.7) = -2.1\%$$

$$d + 2s = -0.7 + (2 * 0.7) = 0.7\%.$$

A plot of the oxygen saturation values from the CO-oximeter against  $R$  as measured by the esophageal pulse oximeter is shown in Fig. 8. The equation of the best fit linear regression

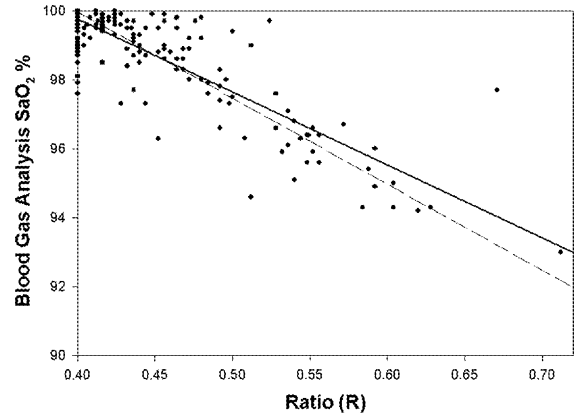


Fig. 6. Plot of SaO<sub>2</sub> from blood gas analysis against the  $R$  measured by the esophageal pulse oximeter for 49 patients. The solid line represents the best fit linear regression line:  $\text{SpO}_2 = 108.2 - 21.1R$ ;  $r^2 = 0.74$ ; SEE =  $0.84$ ;  $n = 155$ ;  $p < 0.001$ . The dashed line represents the empirical calibration equation  $\text{SpO}_2 = 110 - 25R$  used for the estimation of esophageal SpO<sub>2</sub>.

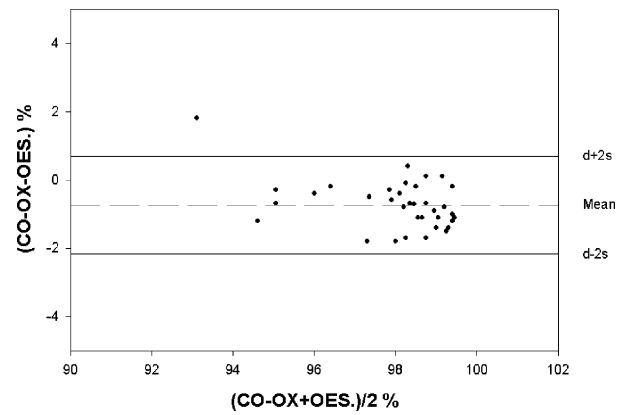


Fig. 7. The difference between blood oxygen saturation values from the CO-oximeter and SpO<sub>2</sub> readings obtained from the reflectance esophageal pulse oximeter plotted against their mean for 17 patients.

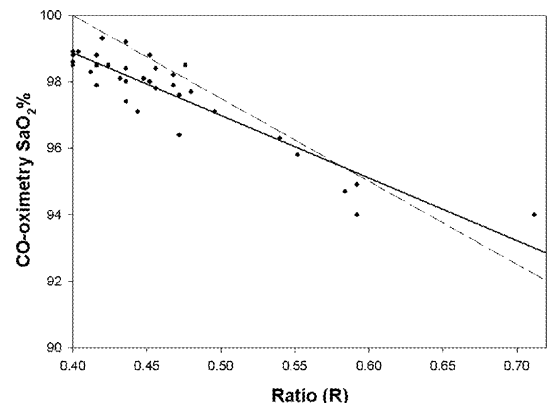


Fig. 8. Plot of SaO<sub>2</sub> from the CO-oximetry against the  $R$  measured by the esophageal pulse oximeter for 17 patients. The solid line represents the best fit linear regression line:  $\text{SpO}_2 = 106.4 - 18.8R$ ;  $r^2 = 0.83$ ; SEE =  $0.58$ ;  $n = 36$ ;  $p < 0.001$ . The dashed line represents the empirical calibration equation  $\text{SpO}_2 = 110 - 25R$  used for the estimation of esophageal SpO<sub>2</sub>.

line was  $\text{SpO}_2 = 106.4 - 18.8R$ ;  $r^2 = 0.83$ ; SEE =  $0.58$ ;  $n = 36$ ;  $p < 0.001$  (solid line in Fig. 8). The dashed line in Fig. 8 represents the empirical calibration equation [19] used for the estimation of esophageal SpO<sub>2</sub>.

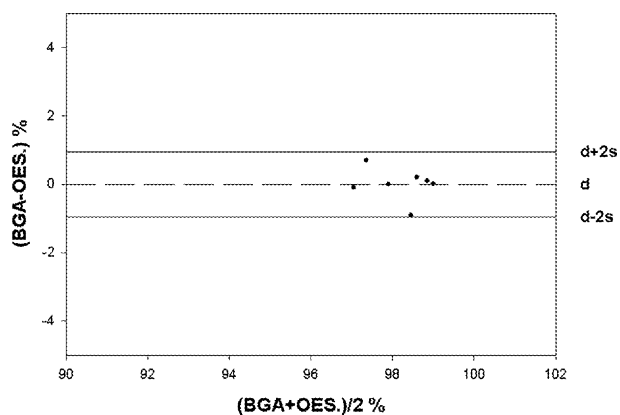


Fig. 9. The difference between blood oxygen saturation values from the blood gas analyzer and SpO<sub>2</sub> readings obtained from the reflectance esophageal pulse oximeter plotted against their mean for five patients when peripheral pulse oximetry failed.

#### E. Patients in Whom Peripheral Pulse Oximetry Failed: Results of the Esophageal Blood Oxygen Saturation Measurements

Of the 49 patients included in the study, five (10.2%) had periods of at least 10 min during which the commercial finger pulse oximeter failed continuously to record pulsatile PPG signals and, thus, failed to estimate SpO<sub>2</sub> values. The esophageal pulse oximeter operated successfully during all the periods when the commercial pulse oximeter failed. The esophageal PPG traces obtained at both wavelengths were of large amplitudes and had similar SNRs to those of patients in whom there was no failure. An esophageal SpO<sub>2</sub> was estimated every 2 s with high reliability.

The five patients in whom peripheral pulse oximetry failure occurred were all cardiac patients undergoing cardiothoracic bypass surgical procedures. In four of the patients, the finger pulse oximeters failed postoperatively (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 room before the patient went on bypass.

During the period of commercial finger pulse oximeter failure, blood samples (a total of seven samples for the five patients) were collected and blood gas analysis was performed. Esophageal pulse oximetry values were also recorded at the same time as the blood sampling.

Fig. 9 shows a comparison of blood oxygen saturation values measured by the blood gas analyzer and the esophageal pulse oximeter probe using a Bland and Altman analysis [20], [21]. The mean difference ( $d$ ) was 0.0% and the standard deviation ( $s$ ) was 0.5%. The limits of agreement were

$$d - 2s = 0.0 - (2 * 0.5) = -1.0\%$$

$$d + 2s = 0.0 + (2 * 0.5) = 1.0\%.$$

#### V. DISCUSSION AND CONCLUSION

The aim of this study was to develop and evaluate an esophageal reflectance pulse oximeter, which would allow accurate and reliable measurements of oxygen saturation in patients with compromised peripheral circulation. The hypothesis

was that blood flow to this central site may be preferentially preserved. A miniature opto-electronic reflectance esophageal oximeter probe, small enough to fit into a conventional stomach tube (French gauge 20) was developed. A novel isolated processing system, based on a *LabVIEW* Virtual Instrument, was also designed to display esophageal PPGs and give an online estimation of esophageal SpO<sub>2</sub> values.

In none of the temperature tests, both *in vitro* and *in vivo* previously reported, was a temperature rise in excess of 0.7 °C detected at the outside wall of the esophageal tube [17]. It is concluded that there is negligible risk of thermal injury to the esophagus when using this probe, as this temperature rise would not be expected to result in tissue damage.

The results show that the esophageal probe can record good quality R and IR ac and dc PPG signals from any depth within the esophagus. The PPG amplitudes are generally higher in the lower esophagus, probably because the chief blood supply of the thoracic esophagus is either direct from the aorta or from branches of the bronchial arteries. The cervical esophagus is supplied mainly by branches of the inferior thyroid artery, which may lead to reduced pulsatility, compared with that in the lower esophagus supplied by the aorta. The present probe allows optimization of the signal by variation of the monitoring depth and is, therefore, more versatile than the “transoesophageal” probe described by Atlee and Bratanow and Prielipp *et al.* [12], [15] which could only be positioned in the upper esophagus at the cricopharyngeous muscle. Moreover, our experience indicates that the PPG amplitudes are likely to be smaller near the cricopharyngeous muscle than they are at greater depths. In any particular patient, the optimal depth for esophageal monitoring may also depend on the presence of abnormality, such as hiatus hernia with esophageal dilatation and reflux of gastric contents. In this study, patients with known esophageal disease, including reflux, were excluded. In any case, the majority of our measurements were derived from above the gastro-esophageal sphincter region and, hence, above the level of a hiatus hernia and dilatation.

In general, the esophageal PPG signals were adversely affected by electrocautery. However electrocautery artifacts were also present in the commercial pulse oximeter connected to the patient’s finger. Similar problems have been reported by other investigators [12]. The electronic components of the probe are completely electrically insulated from the surrounding tissues by the 1-mm-thick plastic wall of the stomach tube. There is no evidence that the probe is damaged by electrocautery or defibrillation (internal or external) as the PPG signal recovered immediately afterwards in all cases and continued to function satisfactorily in multiple uses on subsequent patients. No attempt was made to eliminate the electrocautery artifact in these initial measurements. Further investigations and development will be required for artifact elimination, for example by radio-frequency filtering or shielding [15].

The SpO<sub>2</sub> results from the esophageal pulse oximeter are in good agreement with blood oxygen saturation values obtained from blood gas analysis and CO-oximetry. The limits of agreement from the Bland and Altman analysis are  $\pm 1.8\%$  for the blood gas analysis results and  $-0.7\%$  and  $+2.1\%$  for the CO-oximetry results. In general, clinical practice limits of



agreement of less than  $\pm 3\%$  are considered acceptable [19]. It can reasonably be argued, therefore, that the estimation of blood oxygen saturation by the methods of esophageal pulse oximetry, blood gas analysis and CO-oximetry could be used interchangeably in cardiothoracic surgery patients. Fig. 5 illustrates that the differences between blood oxygen saturation values from the blood gas analyzer and SpO<sub>2</sub> readings obtained from the reflectance esophageal pulse oximeter differ by no more than 3%, except in one measurement, where the difference is +4.5%. There appears to be no technical or physiological reason for the observed deviation, and the PPG signals were acceptable. Notwithstanding, the deviation is only 4.5%, which is less than the 10% observed deviation between two commercial devices reported by Prielipp *et al.* [15]. The algorithm that estimates esophageal SpO<sub>2</sub> in our system was designed to saturate at 100% as in commercial pulse oximeters. This explains the linear relationship (see Fig. 5) between the differences and the mean at mean values between 99% and 100% saturation.

The best fit lines to the plots of blood gas analyzer (Fig. 6) and the CO-oximeter SaO<sub>2</sub> results (Fig. 8) against the *R* ratios from the esophageal pulse oximeter are SpO<sub>2</sub> = 106.4 – 18.8*R* and SpO<sub>2</sub> = 108.2 – 21.1*R*, respectively. These equations are in reasonable agreement with one another, although there were more blood gas analyzer results (*n* = 155) than CO-oximeter values (*n* = 36). The empirical calibration equation used in the esophageal pulse oximeter is SpO<sub>2</sub> = 110 – 25*R*. The slope and intercept of the empirical calibration line is higher than the corresponding values for both best fit lines. The difference between the best fit lines and the empirical calibration line may be due to the fact that the calibration equation is based on probe wavelengths of 660 and 940 nm [19] instead of 655 and 880 nm as used in the present probe. The accumulation of points in these two figures (Figs. 6 and 8) at 0.4 results from the fact that the algorithm estimating SpO<sub>2</sub> is constrained to limit the maximum saturation values to 100%.

Prielipp *et al.* [15] in their measurements at the cricopharyngeal muscle in the esophagus concluded that a “transoesophageal” oximetry probe was inferior to conventional finger pulse oximeters. However, our preliminary measurements below the cricopharyngeal muscle suggest that measurements at these lower sites may permit more accurate values of blood oxygen saturation to be made.

The esophageal pulse oximeter was found to be reliable and accurate in those cases of poor peripheral perfusion when the commercial finger pulse oximeter failed for at least 10 min. Five (10.2%) of the 49 cardiothoracic patients studied, had a finger pulse oximetry failure, which is in agreement with the previously reported incidence of failure [6]. 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.

These results show that in cardiothoracic patients the arterial blood circulation to the esophagus is less subject to vasoconstriction and decreased PPG amplitudes than the finger. Therefore, the human esophagus can be used as an alternative SpO<sub>2</sub> monitoring site during surgery and in intensive care. Moreover,

these results suggest that the esophagus will continue to provide reliable SpO<sub>2</sub> values even when peripheral pulse oximeters fail. Only five patients showed peripheral failure in this study and, therefore, more work is needed to confirm these findings both in cardiothoracic and other patients.

In summary, a new and clinically useful minimally invasive esophageal pulse oximetry system has been successfully developed and evaluated. It shows promise for the reliable monitoring of SpO<sub>2</sub> in patients undergoing prolonged surgical procedures.

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**Panayiotis A. Kyriacou** (M'98) was born in Famagusta, Cyprus, and received the B.E.Sc. degree in electrical engineering from the University of Western Ontario, London, ON, Canada, in 1994, and the M.Sc. and Ph.D. degrees in medical electronics and physics from St. Bartholomew's Medical College, University of London, London, U.K., in 1995 and 2001, respectively.

He is currently a Lecturer in Medical Electronics and Physics at Queen Mary, University of London. He has been engaged in research into the application

of electronic and physical techniques to solve practical problems in surgery and anaesthesia. His research interests are in the developing of noninvasive techniques for blood gas measurements, electro-optical sensors, microprocessor-based medical instrumentation, computer-implemented virtual medical instruments, physiological monitoring, biosignal processing, and the study of light interaction with biological media.



**Richard M. Langford** was Appointed Consultant Anesthetist and Director of Academic Anesthetics Unit, St. Bartholomew's Hospital, London, U.K., in 1991. From 1998–1999, he was Chairman of the Anesthetics Department. He has held regional postgraduate training posts since 1994, and has been Royal College of Anesthetists Regional Advisor, North Thames East, since 1999. He has been a partner in "IBIS" (1997–2000) and in "IMPROVE" (1996–1998)—two technology-based European Commission Biomedicine Projects: Acute and

chronic pain studies, peri-operative endocrine changes, toxicology of smoke inhalation injury; and cyanide measurement. He has established the Barts and The Royal London High Fidelity Medical Simulation Centre for undergraduate and postgraduate, multiprofessional healthcare training, opened in 2000 by Princess Anne.

His research interests include: opto-electrical haemodynamic patient-monitoring methods, depth of anaesthesia and cerebral monitoring during surgery and intensive care.



**Deric P. Jones** (M'01) was born in Beaufort, Monmouthshire, South Wales and received the degrees of B.Sc. in physics and the Ph.D. in metal physics from Imperial College, University of London, U.K. He joined the Medical School of St. Bartholomew's Hospital in 1976 where he became a Reader in Medical Electronics and Physics. He has been in the Department of Engineering, Queen Mary, University of London since 2000. His research interests include the application of opto-electronic techniques in anaesthesia and surgery, and physiological measurements in orthodontics and ophthalmology.

**Sarah Powell** is a Specialist Registrar in Anesthetics at The Bart's and the London School of Anesthesia, London, U.K.