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Pulseless Oximetry
A Preliminary Evaluation

Thomas K. Aldrich, MD; Pragya Gupta, MD; Sean P. Stoy, MD; Anthony Carlese, DO; and Daniel J. Goldstein, MD

BACKGROUND: Pulse oximetry fails when pulsations are weak or absent, common in patients with continuous flow left ventricular assist devices (LVADs). We developed a method to measure arterial oxygenation (Sao_2) noninvasively in pulseless patients with LVADs.

METHODS: The technique involves 5- to 10-s occlusions of radial and ulnar arteries on one hand. A fingertip is transilluminated alternately with light-emitting diodes emitting 660 nm (red) and 905 nm (infrared). During the approximately 1 s after release of occlusion, changing attenuance of each wavelength is measured and their red/infrared arterial blood attenuance ratio (R/IR) calculated. We studied five normal subjects breathing hyperoxic, normoxic, or hypoxic gas mixtures to establish a calibration curve, using standard pulse oximetry as the gold standard. We also studied seven pulseless patients with LVADs (two studied twice) at clinically determined oxygenation.

RESULTS: Normal subject data showed close correlation of oxygen saturation by pulse oximetry (Spo_2) with R/IR, ($Spo_2 = 111 - [26.7 \times R/IR]$; $R^2 = 0.975$). For patients with LVADs, predicted Sao_2 (from the calibration curve) tended to underestimate measured Sao_2 (from arterial blood) by a clinically insignificant 1.1 ± 1.6 percentage points (mean $\pm$ SD), maximum 3.4 percentage points.

CONCLUSIONS: Preliminary results in a small number of patients demonstrate that pulseless oximetry can be used to estimate arterial saturation with acceptable accuracy. A noninvasive oximeter that does not rely on pulsatile flow would be a valuable advance in assessing oxygenation in patients with LVADs, for whom the only current option is arterial puncture, which is painful, risks arterial injury, and only provides a snapshot evaluation of oxygenation.

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ABBREVIATIONS: AC = alternating current; DC = direct current; LVAD = left ventricular assist device; R/IR = red/infrared arterial blood attenuance ratio; Sao_2 = arterial oxygenation; Spo_2 = oxygen saturation by pulse oximetry

AFFILIATIONS: From the Pulmonary Medicine Division (Drs Aldrich, Gupta, and Stoy), the Critical Care Medicine Division (Dr Carlese), and the Department of Cardiothoracic Surgery (Dr Goldstein), Montefiore Medical Center and Albert Einstein College of Medicine, Bronx, NY.

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CORRESPONDENCE TO: Thomas K. Aldrich, MD, Pulmonary Medicine Division, Montefiore Medical Center, 111 E 210th St, Bronx, NY 10467; e-mail: taldrich@montefiore.org

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Pulse oximetry has become indispensable in all areas of medicine.[1] It estimates arterial oxygenation (Sao_2) by calculating the ratio of pulsatile changes in light attenuance at two wavelengths, selected for their differing absorption by oxyhemoglobin and reduced hemoglobins.[2,3] Pulse oximetry fails when pulse volume is too small to achieve acceptable signal to noise ratios, as in peripheral vascular disease[4] or in patients on continuous flow left ventricular assist devices (LVADs),[5] both of which are increasing in prevalence.[6,7] For such patients, if arterial oxygenation must be assessed, the only option is arterial puncture for arterial blood gas measurement, which is painful, carries a small risk of arterial injury, and provides only a "snapshot" assessment of oxygenation. Therefore, a noninvasive oximeter that does not require pulsatile flow would have a valuable role in assessment of such patients. We report preliminary evaluation of the accuracy of such a technique in patients with LVADs.

Materials and Methods

Rationale

An approach to measuring arterial saturation noninvasively in the absence of pulsatile flow would be to transiently occlude arterial supply (but not venous outflow) of a hand and compare changes in attenuance of two wavelengths through a finger when arterial occlusion is released. The only substantial change in attenuance would due to inflow of arterial blood, so comparison of prerelease to postrelease attenuance of two appropriately selected wavelengths should correlate with arterial saturation.

Technique

Using a Dolphin Voyager pulse oximeter (Dolphin Medical Inc), which allows for downloading of high-resolution (16-bit) photoplethysmographic data, one fingertip was alternately transilluminated at 27.5 Hz by two light-emitting diodes, emitting approximately 660 nm and 905 nm. Photoplethysmographic data were collected before, during, and after 5- to 10-s manual occlusions of radial and ulnar arteries. An example of the resulting data, from a normal subject, is shown in Figure 1A. Naturally occurring pulsations can be observed, followed at the 7-s point, by a gradual rise in transmission due to outflow of venous blood, during a 10-s arterial occlusion. At release of occlusion, at the 17-s time point, transmission rapidly decreases, because of arterial inflow.

For the 1 to 2 s after release of occlusion, raw light levels at each wavelength at each time point are expressed as fractions of their maximum, yielding relative transmittance (T) levels, demonstrating the decrease caused by influx of arterial blood (Fig 1B, top two tracings). Arterial blood attenuance (the sum of absorbance and scatter) is calculated for each wavelength at each time point as $A = \log(1/T)$ (Fig 1B, bottom two tracings). Arterial blood attenuance of 660 nm is then plotted against arterial blood attenuance of 905 nm, yielding a straight line (Fig 1C), the slope of which represents the average red/infrared arterial blood attenuance ratio (R/IR).

Subjects

The study was approved by the Einstein institutional review board protocol #12-10-359. Informed consent was obtained from all subjects.

Five normal subjects, two women and three men, two white and three Asian, with ages ranging from 29 to 63 years, were studied while they breathed room air, supplemental oxygen by nasal cannula, or supplemental nitrogen by nasal cannula, monitored by standard pulse oximetry. No side effects of breathing the hyperoxic or hypoxic mixtures were noted. For each subject at each level of oxygenation, median R/IR data were calculated and plotted against the oxygen saturation by pulse oximetry (Spo_2) measured by the standard pulse oximeter. A calibration curve was generated by linear regression (Fig 2).

Seven pulseless patients with LVADs, two women and five men, three black, one white, and three Asian, with ages ranging from 45 to 76 years, with arterial lines in place, were studied (two on two occasions) at their clinically determined levels of oxygenation. R/IR was calculated as described previously and compared with Sao_2 measured from arterial blood collected from the arterial line at the same time as the R/IR measurement.

Results

No discomfort or adverse effects were noted. Figure 2 shows the calibration curve generated from normal subjects' data, medians of at least three replicates: estimated Sao_2 (%) $= 111 - (26.7 \times \text{R/IR})$; $R^2 = 0.975$. Data for the normal subjects (No. 1-5) were highly reproducible and follow the expected inverse relationship between Spo_2 and R/IR.

Table 1 lists selected clinical data for the nine studies in seven patients with LVADs. Figure 3 shows one example of raw data from a patient with an LVAD (No. 2). The median of at least three R/IR changing attenuance ratios was calculated for each study. Estimated Sao_2 was calculated from the median R/IR, using the calibration curve derived in normal subjects.

Figure 4 shows results of the nine studies in patients with LVADs. Data tended to fall slightly above the calibration curve calculated for the normal subjects, so in the patients with LVADs, R/IR tended to underestimate Sao_2 by an average of 1.1 ± 1.6 percentage points (mean $\pm$ SD), with the largest difference 3.4 percentage points.

Discussion

We demonstrate that a comparison of changes in attenuance of light at two different wavelengths, transmitted through a fingertip, brought about by release of momentary arterial occlusion, can be used to estimate arterial saturation with reasonable accuracy, without reliance on pulsatile flow. Our experiments in patients with LVADs were performed in subjects with no pulse or insufficient pulse to result in reliable pulse oximetry readings, but they had normal or near-normal cardiac output and normal or near-normal blood flow into digits. They had no fingertip pulses, not because of impaired blood flow into