Continuous Venous Oximetry in Surgical Patients

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A prospective study was performed to evaluate the efficacy of continuous venous oximetry to supplement traditional hemodynamic monitoring in 39 critically ill surgical patients. There was no statistically significant difference in SvO_2 between the continuous *in vivo* values and *in vitro* values (0.694 ± 0.095 *vs.* 0.698 ± 0.108). There was no statistically significant correlation between continuously measured SvO_2 and PaO_2 ($r = 0.09$, $p > 0.5$), SaO_2 ($r = 0.08$, $p > 0.5$), or oxygen consumption ($r = 0.46$, $p > 0.5$). There was a slight but statistically significant correlation between continuously measured SvO_2 and cardiac output ($r = 0.40$, $p < 0.025$) and oxygen delivery ($r = 0.49$, $p < 0.005$). There was a highly significant correlation between continuously measured SvO_2 and oxygen utilization coefficient ($r = -0.96$, $p < 0.001$). Continuously measured SvO_2 is a reliable predictor of SvO_2 measured intermittently by *in vitro* methods. In critically ill surgical patients, SvO_2 does not correlate highly with the individual determinants of oxygen transport but rather correlates with the oxygen utilization coefficient and therefore reflects the overall balance between oxygen consumption and delivery.

CONTINUOUS *in vivo* venous oximetry has been suggested to be a reliable indicator of "combined cardiorespiratory function."[1] Recent clinical reports using continuous SvO_2 measurements have validated these measurements in a variety of patient populations.[2-4] The purpose of this report is to evaluate the correlation between continuously measured SvO_2 and standard *in vitro* measurements of SvO_2 and to compare continuously measured SvO_2 with the oxygen transport parameters of arterial oxygen saturation, cardiac output, oxygen delivery, oxygen consumption, and oxygen utilization coefficient in a group of critically ill patients who have undergone major surgical procedures.

This study was funded in part by NIH/NHLBI Public Health Service Research Training Award 5-F32-HL06508-02CLN.

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Submitted for publication: October 19, 1985.

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Methods

Thirty-nine patients admitted to the Surgical Intensive Care Unit at the University of Miami/Jackson Memorial Medical Center who had sustained trauma or undergone a major noncardiac surgical procedure and required hemodynamic monitoring were studied. The primary surgical diagnosis, initial SvO_2, and indication for hemodynamic monitoring are shown in Table 1. All patients had a Shaw Opticath flow-directed pulmonary catheter (Oximetrix Corporation, Moutain View, CA) inserted percutaneously. The devices were standardized by the *in vitro* method prior to insertion of the catheter in 25 patients and by the *in vivo* calibration method in 13 patients. Venous oxygen saturation (SvO_2) was also measured *in vitro* using an American Optical Unistat cooximeter (American Optical Corporation, Buffalo, NY). Arterial and venous gas tensions and pH were measured on a Corning 175 automated blood gas analyzer (Corning Medical and Scientific Co., Atlanta, GA). The data reported are mean values and standard errors obtained from the first complete hemodynamic profile and simultaneous arterial and venous gas analysis in each patient. Derived cardiopulmonary parameters were calculated using standard equations.[5]

$$CaO_2 = (SaO_2 \times Hb \times 1.34) + (PaO_2 \times 0.0031)$$

$$CvO_2 = (SvO_2 \times Hb \times 1.34) + (PvO_2 \times 0.0031)$$

$$C(a\text{-}v)O_2 = CaO_2 - CvO_2$$

$$DO_2 = CO \times 10 \times CaO_2$$

$$VO_2 = CO \times 10 \times C(a\text{-}v)O_2$$

$$OUC = VO_2/DO_2$$

TABLE 1. *Diagnoses*

#	Diagnosis	SvO_2	Other
1	Pancreatic abscess	0.81	Septic shock
2	Hepatic Abscess	0.61	Pneumonia
3	GSW	0.75	Hypertension, oliguria
4	CA oral cavity	0.59	CHF, shock
5	CA colon	0.70	CAD
6	Cecal volvulus	0.70	ARF
7	Aortic aneurysm	0.32	Cardiomyopathy
8	Cervical CA	0.55	Oliguria
9	Variceal bleeding	0.68	CAD
10	Prostatic CA	0.67	CAD
11	Aortic thrombosis	0.66	Shock, oliguria
12	Recurrent basal cell CA	0.67	CAD
13	Variceal bleeding	0.65	Oliguria
14	Multiple trauma	0.49	Elevated ICP
15	Renal artery stenosis	0.70	Hypertension
16	Multiple trauma	0.51	
17	Obstructive jaundice	0.66	CAD, hypertension
18	Acute pancreatitis	0.77	Shock
19	Colon perforation	0.82	
20	Bowel obstruction	0.64	Oliguria
21	Vascular occlusion	0.80	CAD, hypertension
22	Multiple trauma	0.70	Elevated ICP
23	CA colon	0.70	CAD, shock
24	CA Colon	0.75	Oliguria, hypertension
25	GSW to Aorta	0.68	Oliguria, shock
26	CA uterus	0.74	Postop hemorrhage
27	SW Chest	0.68	Oliguria
28	CA esophagus	0.62	
29	Exploratory laprotomy	0.61	Shock, ARF
30	GSW	0.83	Shock
31	Multiple trauma	0.45	Elevated ICP
32	Acute pancreatitis	0.73	
33	CA esophagus	0.67	Hypotension
34	Multiple trauma	0.58	ARF
35	Cecal volvulus	0.71	ARF
36	Cholangitis	0.73	ARF
37	CA cervix	0.60	Oliguria
38	Acute abdomen	0.70	ARF
39	Pancreatic abscess	0.82	ARF

Admission diagnosis, initial SvO_2 value, and indication for monitoring are shown. (GSW = gun shot wound, ARF = acute respiratory failure, CA = carcinoma, CHF = congestive heart failure, CAD = coronary artery disease, ICP = intracranial pressure, SW = stab wound.)

(CaO_2 = arterial oxygen content [ml O_2/dl], SaO_2 = arterial oxygen saturation [fraction], Hb = hemoglobin concentration [g/dl], PaO_2 = arterial oygen tension [mmHg], CvO_2 = venous oxygen content [ml O_2/dl], SvO_2 = mixed venous oxygen saturation [fraction], PvO_2 = mixed venous oxygen tension [mmHg], $C(a\text{-}v)O_2$ = arterial-venous oxygen content difference [ml O_2/dl], DO_2 = oxygen delivery [ml/min], CO = cardiac output [l/min], VO_2 = oxygen consumption [ml/min], OUC = oxygen utilization coefficient [fraction].)

All patients had cardiac output measurements by the thermodilution technique. The measurements were made in triplicate and the mean value of the measurements recorded. The calibration of the *in vivo* oximeter was checked whenever there was a significant change in the SvO_2 reading from the baseline value, the device was disconnected for transport of patients, or there was a clinical suspicion that the value may be in error for any reason. If the *in vivo* and *in vitro* values varied by more than 0.02, the *in vivo* device was recalibrated. The device was recalibrated at least once in each 24-hour period.

Data were evaluated using Student's t-test and linear regression analysis. P values less than 0.05 were considered statistically significant.

Results

There was no statistically significant difference between initial SvO_2 values obtained by continuous *in vivo* measurements and those obtained from *in vitro* cooximetry (0.694 ± 0.095 *vs.* 0.698 ± 0.108, $p > 0.5$). There was a highly significant correlation between values obtained by these two methods over a wide range of SvO_2 values (0.42 to 0.83) ($r = 0.92$, $p < 0.001$) (Fig. 1, Table 2). At the time of the first calibration check, there was no significant difference in the SvO_2 values obtained by the two techniques (0.750 ± 0.017 *vs.* 0.738 ± 0.014, $p > 0.05$). Also at the time of recalibration the same high degree of correlation existed between values obtained by the two techniques ($r = 0.91$, $p < 0.001$).

There was no statistically significant correlation found between continuously measured SvO_2 and PaO_2 ($r = 0.09$, $p > 0.5$), SaO_2 ($r = 0.08$, $p > 0.5$) (Fig. 2), oxygen consumption ($r = 0.46$, $p > 0.5$), or arterial venous oxygen content difference ($r = -0.25$, $p > 0.5$). There was a slight but statistically significant correlation between continuously measured SvO_2 and cardiac output ($r = 0.40$, $p < 0.025$) and oxygen delivery ($r = 0.49$, $p < 0.005$). There was a highly significant statistical correlation between continuously measured SvO_2 and the oxygen utilization ratio ($r = -0.96$, $p < 0.001$) (Fig. 3).

Discussion

The content of oxygen in mixed venous blood is determined by the variables of the Fick equation. The Fick

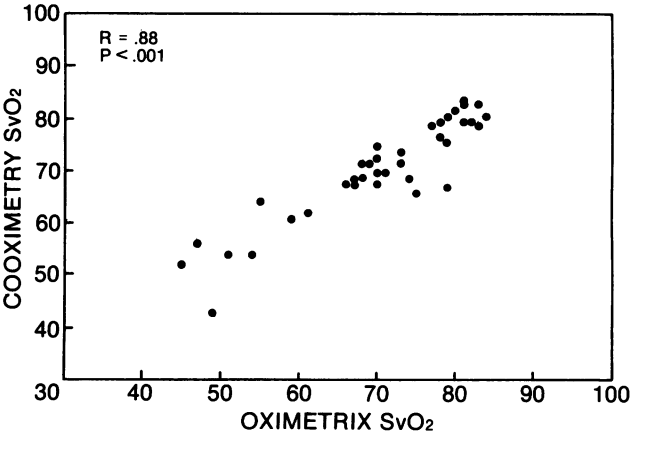


FIG. 1. The relationship between SvO_2 measured *in vivo* using a fiber optic pulmonary artery catheter and *in vitro* using a bench oximeter.

equation relates cardiac output, tissue oxygen consumption, and the arterial-venous oxygen content difference:

$$VO_2 = C(a\text{-}v)O_2 \times CO \times 10.$$

When the terms of the Fick equation are rearranged, it may be seen that the determinants of SvO_2 are the components of oxygen delivery and oxygen consumption:

$$VO_2/(CO \times 10) = C(a\text{-}v)O_2$$

$$VO_2/(CO \times 10) = CaO_2 - CvO_2$$

$$VO_2/(CO \times 10) - CaO_2 = -CvO_2$$

$$CvO_2 = CaO_2 - [VO_2/(CO \times 10)]$$

$$CvO_2/CaO_2 = 1 - [VO_2/(CO \times 10 \times CaO_2)].$$

(If $SaO_2 = 1.0$ then $SvO_2 = CvO_2/CaO_2$.)

$$SvO_2 = 1 - [VO_2/(CO \times 10 \times CaO_2)]$$

$$SvO_2 = 1 - VO_2/DO_2.$$

(See methods section for abbreviations.)

An increase in oxygen consumption or decrease in cardiac output, hemoglobin concentration, or arterial oxygen saturation will produce a decrease in SvO_2, provided there is no change in the other components of the equation.

Normally, venous oxygen saturation is maintained within a narrow range. Decreases in SvO_2 occur frequently in patients with cardiopulmonary complications. The changes in SvO_2 often precede changes in blood pressure, heart rate, or pulmonary artery occlusion pressure.[6] The degree of decrease may correlate with the magnitude of functional cardiopulmonary impairment[7] and decreases in SvO_2 that do not improve with therapy are associated with a poor prognosis.[8] Severe venous hypoxemia has been associated with lactic acidosis and a high mortality in critically ill patients.[9]

Instrumentation for the continuous monitoring of oxygen saturation has been available since the 1970's. The incorporation of fiberoptics into a flow-directed pulmonary artery catheter and improvements in catheter design and handling characteristics became available in the early 1980's.[10] The system that is currently available has been shown to be accurate[1] and clinically useful.[11]

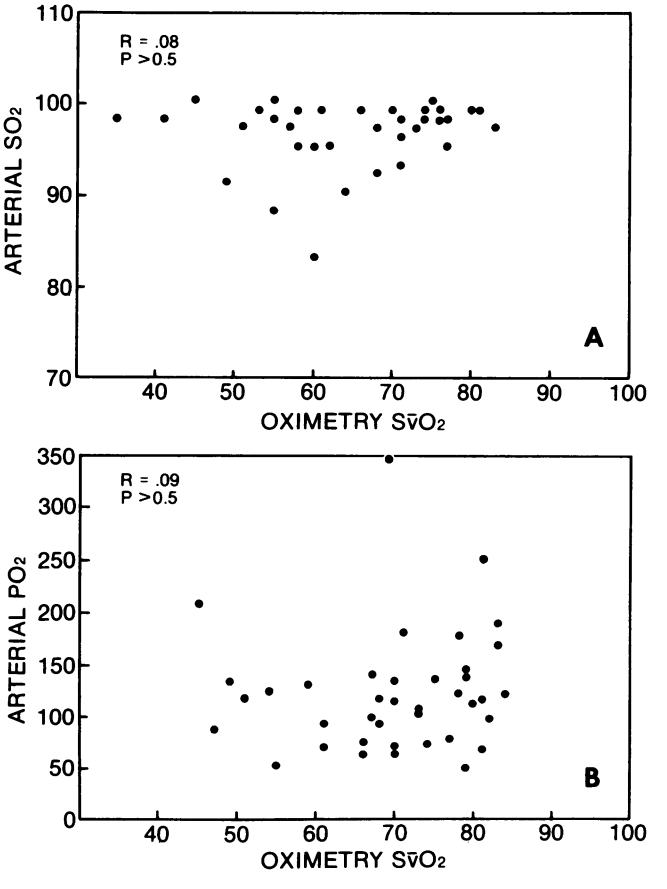
While continuously measured SvO_2 may correlate with hemodynamic changes (cardiac output) in some groups of patients,[3] the nonsteady nature of critical illness[12] has taught us not to expect that arterial oxygen saturation, hemoglobin concentration, or oxygen consumption will remain stable, and therefore changes in SvO_2 will not necessarily reflect changes in cardiac output in these patients. Data from this study indicate that, although there is some statistical correlation between SvO_2 and both cardiac output and oxygen delivery, the correlation coefficients are so small that the use of SvO_2 as a predictor of cardiac output is unreliable. Similarly, correlations could not be established between SvO_2 and arterial oxygenation, oxygen consumption, or arterial-venous oxygen content difference as independent variables. The high degree of inverse correlation between SvO_2 and oxygen utilization ratio makes this relationship clinically useful.

TABLE 2. *Distribution of Initial SvO_2 Values*

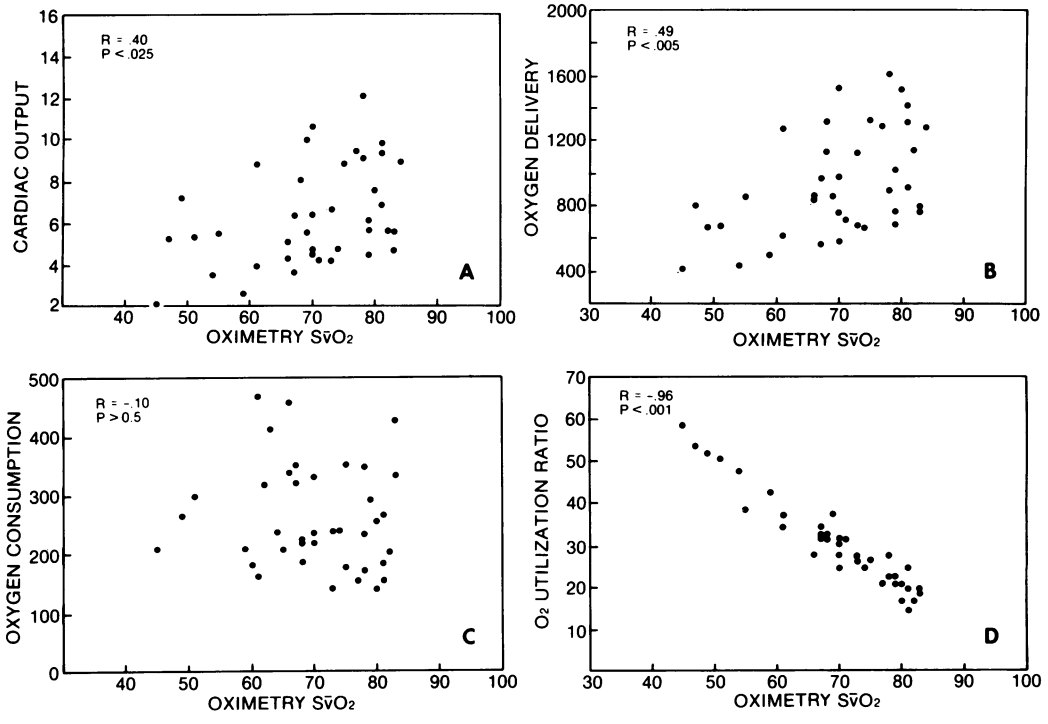
	Initial SvO_2				
	<50	50–59	60–69	70–80	>80
Number of patients	3	4	14	14	4

The number of patients within each range of initial SvO_2 value is shown.

The goal of many interventions in critically ill patients is to insure that oxygen delivery to the tissue meets or exceeds the oxygen demand of that tissue.[13] Our clinical ability to monitor this relationship is severely lacking. While we can measure total body oxygen uptake by the patient with reasonable reliability, oxygen uptake is equal to oxygen consumption only in the steady state. To make



FIGS. 2A and B. The relationship between SvO_2, PaO_2 (*A*), and SaO_2 (*B*).



FIGS. 3A–D. The relationship between SvO_2, cardiac output (*A*), oxygen delivery (*B*), oxygen consumption (*C*), and oxygen utilization ratio (*D*).

matters worse, oxygen consumed by the patient is not necessarily equal to the oxygen demand by the tissues of the patient. Normally, oxygen consumption increases when oxygen demand increases. Oxygen consumption may increase through an increase in the extraction of oxygen from arterial blood as it traverses the capillary bed (*i.e.*, an increase in $C(a\text{-}v)O_2$) or through an increase in blood flow (*i.e.*, cardiac output). Both of these factors may increase by approximately threefold in normal subjects, allowing a ninefold increase in oxygen consumption to meet the oxygen demand of the tissue. Critically ill patients may not be capable of increasing cardiac output spontaneously and therefore may have a markedly diminished "safety factor" in regard to increasing oxygen consumption. When oxygen demand exceeds oxygen consumption, anaerobic metabolism ensues and lactic acidosis results.[14]

An additional complicating factor is the fact that oxygen consumed by the various tissues differs, and it is not possible at this time to measure clinically oxygen demand or consumption of individual organs. For example, at rest myocardial oxygen extraction is near maximal while renal oxygen extraction is very low. At times of stress when myocardial oxygen demand increases, oxygen consumption can increase only by increases in myocardial blood flow. During this same period of stress, renal blood flow may actually decrease dramatically and renal oxygen consumption may be maintained by increased oxygen extraction in the renal capillary bed. Mixed venous blood represents a "flow-weighted average" of the blood returning from all perfused tissues. That is to say, the magnitude of the effect of oxygen extraction by any organ on SvO_2 is proportional to the blood flow to that organ so that low-consumption, high-flow organs (kidneys) have a greater effect on SvO_2 than do high-consumption, low-flow organs (myocardium).

Since our goals are not necessarily to provide the highest oxygen delivery but rather to bring into balance the relationship between oxygen consumption and oxygen delivery, it seems apparent that continuously measured SvO_2 is at this time the best indicator clinically available to assure that this goal has been attained.

Continuous venous oximetry in critically ill surgical patients serves three major purposes. First, a low or rapidly decreasing SvO_2 indicates an imbalance between oxygen consumption and oxygen delivery that requires further investigation of the determinants of these parameters. A low or falling SvO_2 does not tell us which therapy is appropriate in a given situation but rather tells us that more information is needed to assess the problem. The falling SvO_2 may indicate a decrease in hemoglobin concentration, arterial oxygen content, or cardiac output, or an increase in tissue oxygen consumption. When a low or decreasing SvO2 is encountered, the clinician may obtain an arterial blood gas analysis, hemoglobin value, and hemodynamic assessment of the patient to select the most appropriate intervention that may restore the balance between oxygen consumption and delivery. This function has been described by Watson[15] as the "early warning system" of cardiorespiratory imbalance.

The second purpose of continuous venous oximetry in

surgical patients is to provide assurance that, in fact, the patient's oxygen transport is in balance. This function has been described by Civetta as "safety in no numbers."[16] A stable mixed venous saturation indicates that either the determinants of oxygen transport balance are unchanged or that two factors are changing in equal and opposite directions. The safety in no numbers allows clinicians not to make measurements of arterial and venous blood gases on a frequent basis and not to make hemodynamic measurements on a frequent basis in patients who do not require such costly and time-consuming interventions.

The third purpose of continuous venous oximetry is to improve the efficiency of the delivery of care to critically ill patients. Typically, in an intensive care unit when a cardiorespiratory crisis occurs, initial measurements of hemodynamics and gas exchange parameters are made and an intervention is initiated. Following the intervention and a period for stabilization, the cardiorespiratory profiles are repeated and the intervention (vasoactive or inotropic drug infusion, FiO_2, positive end-expiratory pressure, etc.) is modified or another intervention is instituted. The cardiorespiratory profile is repeated, and this cycle repeats itself until the crisis is resolved. The time required for this orderly approach to a cardiorespiratory crisis in terms of time for the measurement, stabilization period, return of laboratory results, and institution of the next intervention limits the clinician to a maximum of three to four intervention changes per hour. By using continuously measured SvO_2 as an indicator of the balance between oxygen supply and demand, the frequency of the interventions, once the major problem is identified by a complete cardiorespiratory profile, may be increased. It must be emphasized, however, that continuous measurement of SvO_2 does not replace a full cardiorespiratory profile to indicate which intervention is more likely to restore the balance between oxygen supply and demand. Thus, continuously measured SvO_2 may improve the efficiency of the delivery of critical care to patients who are most stable by decreasing the frequency of other measurements and to those who are least stable by allowing an increased number of interventions between complete assessments. The result of this approach to the monitoring of critically ill patients is to reduce the number of measurements in those patients who are most stable and optimize the timing of measurements in those who are least stable.

Acknowledgments

The authors express their appreciation to the fellows, residents, nurses, and respiratory therapists in the SICU at Jackson Memorial Medical Center whose dedication to patient care made this study possible. Special thanks go to Hans Anderson, RRT, and David D'Angostino, RRT, who aided in data collection for this study.

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