Comparison of Central-Venous to Mixed-Venous Oxygen Saturation During Changes in Oxygen Supply/Demand*

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Because central venous O₂ saturation (superior vena cava, ScvO₂) can be monitored with less patient risk than mixed venous O₂ saturation (pulmonary artery, SvO₂), we examined the correlations between SvO₂ and ScvO₂ over a broad range of cardiorespiratory conditions, including hypoxia, hemorrhage, and resuscitation in anesthetized dogs. The correlation coefficient (r) between SvO₂ and ScvO₂ in 179 simultaneously drawn blood samples from 22 dogs was 0.97. In another nine dogs, the two sites were continuously and simultaneously monitored with fiberoptic catheters; r was 0.96 with a mean difference of 3.7±2.9 percent (SD) saturation. In each dog the changes in ScvO₂ closely paralleled the changes in SvO₂. Although absolute values of ScvO₂ are not sufficiently identical to SvO₂ to calculate O₂ uptake or pulmonary shunt precisely, close tracking of changes in the two sites across a wide range of hemodynamic conditions warrant further consideration of ScvO₂ for patient monitoring of trends in O₂ supply/demand.

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The recent addition of fiberoptic sensors in pulmonary artery (PA) catheters has made possible the continuous monitoring of mixed venous oxygen saturation (SvO₂). This can be helpful in patient management, because changes in SvO₂ indicate changes in the balance of O₂ supply and demand and thereby provide an index of tissue oxygenation, even without the determination of cardiac output or calculation of actual values for O₂ delivery and consumption. However, the use of PA catheters is not without risk to the patient, and the risk/benefit ratio is currently a matter for vigorous debate. Central venous catheters (superior vena cava), on the other hand, are less costly, have less risk of complications, and are used in the critically ill more routinely than PA catheters. Central venous O₂ saturation (ScvO₂) monitoring has been advocated as a simple method of assessing changes in cardiac function in patients with MI. Others, however, have questioned the usefulness of ScvO₂ measurements, particularly in shock, because ScvO₂ tended to diverge from SvO₂.

To test the hypothesis that ScvO₂ parallels SvO₂ sufficiently well over wide ranges of altered O₂ delivery to monitor cardiorespiratory status in critically ill patients, we made simultaneous measurements of ScvO₂ and SvO₂ in anesthetized dogs while decreasing O₂ delivery by lowering inspired O₂ fraction and by hemorrhage. In addition, we used specially made fiberoptic catheters to monitor both variables continuously and to observe on-line the rapidity with which each variable changed with alterations of O₂ delivery.

METHODS

Thirty-eight mongrel dogs of either sex (av wt, 18±2 kg) were anesthetized with pentobarbital sodium (30 mg/kg IV), with supplemental doses given as needed. They were intubated and ventilated with a Harvard respirator at ten breaths/min and a tidal volume sufficient to maintain arterial PCO₂ at 35 to 40 mm Hg. The animals were paralyzed with succinylcholine chloride. Systemic and PA pressures, heart rate, and ECG were continuously recorded.

In the first series of experiments (n = 24) double-lumen PA catheters were custom made from plastic tubing of 1.2- and 3.0-mm outside diameters. The distal tip of the catheter was floated into a pulmonary artery by observing the pressure wave form. The proximal orifice was 14 to 16.5 cm from the tip, and its location in the superior vena cava was verified at autopsy. In two cases in which the proximal orifice was in the right atrium, the data were discarded. Simultaneous blood samples were drawn anaerobically and slowly, and immediately analyzed for O₂ saturation with an instrumentation Laboratory 282 CO-oximeter.

In the second series of 14 dogs, mixed and central venous O₂ saturations were continuously monitored with fiberoptic catheters introduced through the right external jugular vein. The PA catheter was a Swan Ganz oximetry TD catheter (7.5 F, American Edwards Lab). After inflating the balloon with 1.0 ml of air, we placed the catheter tip in a pulmonary artery by observing the pressure wave form until a typical wedge tracing appeared. A custom-made catheter (4F, Baxter Edwards) which used the same fiberoptics as the Edwards PA catheter, was advanced into the external jugular vein between 18 and 21 cm, depending on the size of the dog. The placement of the catheter tip in the superior vena cava was verified at autopsy. In one dog the tip was in the right atrium, and we were unable to advance the second catheter into the vena cava of four other dogs. Data from these animals were excluded from analysis.

The fiberoptic catheters were each connected to a separate model Sat-1 oximeter/cardiac output computer (American Edwards), which displayed a digital output and a continuous graph of saturation over

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time. With this system, $O_2$ saturation values were obtained by two-wavelength reflectance spectrophotometry and updated at 2-s intervals. Data from both catheters were transferred via RS-232 interfaces to a Compaq computer for display and storage. After the catheters were in place, in vivo calibration was completed according to the manufacturer's instructions using the co-oximeter as the reference. At 30-min intervals, mixed and central venous blood samples were drawn for measurement of $O_2$ saturation, and the catheters were recalibrated to the CO-oximeter value if the difference between in vivo and in vitro saturation was greater than 5.
Table 1 — Mean Values and Correlations for Mixed and Central Venous Fiberoptic Catheters under Various Conditions *

| Condition | SvO₂   | ScvO₂  | r     | n | |SvO₂-ScvO₂| |
|-----------|--------|--------|-------|---|-----------------|
| Total     | 53 ± 16| 52 ± 15| 0.96  | 29851| 3.7 ± 2.9 |
| Control   | 59 ± 14| 57 ± 15| 0.98  | 14167| 2.8 ± 2.0 |
| Hemorrhage| 33 ± 14| 37 ± 12| 0.94  | 1490 | 6.0 ± 3.1 |
| Resuscitation | 51 ± 12| 50 ± 11| 0.91  | 9838 | 4.2 ± 2.7 |
| Hypoxia   | 27 ± 12| 31 ± 10| 0.78  | 1897 | 5.9 ± 5.9 |
| Hypoxia   | 63 ± 12| 61 ± 14| 0.96  | 1896 | 3.5 ± 2.6 |

*Values are mean ± SD; all correlations (r) significant at p < .001; SvO₂, mixed venous O₂ saturation; ScvO₂, central venous O₂ saturation; n, number of comparisons; |SvO₂-ScvO₂|, mean difference.

percent. Data were used for analysis only when there had been no alarms indicative of inappropriate light reflection.

The animals were studied in a normoxic control condition and after a variety of cardiorespiratory perturbations. Hypoxia was induced by ventilation with 9 percent O₂ in N₂, and hyperoxia with 100 percent O₂. Some animals were bled, up to 40 ml/kg, and resuscitated either with volume replacement of RBCs and dextran, or by replacing 15 percent of the shed blood volume with hypertonic saline solution (7.5 percent NaCl in hydroxyethylstarch). In other dogs, dexametoxamide (Fisons Ltd) an experimental β₂ agonist, was infused at 4 μg/kg -min -1 to stimulate cardiac output.

Mixed and central venous O₂ saturation values were compared by linear regression and Pearson product-moment correlations. Data are reported as means ± SD.

RESULTS

In vitro O₂ Saturation Measurements

The correlation between SvO₂ and ScvO₂ for the first series of experiments obtained from intermittent simultaneous blood sampling is shown in Figure 1. The individual correlation coefficients between the two sites for each of 22 dogs ranged from 0.92 to 0.99. The various experimental interventions, from hypoxia to severe hypoxia, produced a range in venous O₂ saturation from greater than 80 percent to less than 10 percent. When changes in O₂ saturation between consecutive sampling periods were plotted for the two sampling sites, the correlation was similarly high (Fig 2). We also computed the mean difference between the catheter sites using the absolute value of (SvO₂-ScvO₂), ignoring the sign of the difference because there was no consistent unidirectional bias between the two sites. The mean difference in O₂ saturation between the two sampling locations for all comparisons in this series was 4.3 ± 3.6 percent.

In vivo O₂ Saturation Measurements

In the nine dogs from which we obtained data in the second series of experiments, the observation period of continuous in vivo O₂ saturations ranged from 1 to 4 h. The correlation coefficient between mixed and central venous saturation for all comparisons combined was 0.95 (Table 1). When calculated for each dog individually, the correlation coefficient across all conditions was >0.96 in all but two animals, in which it was 0.85 and 0.65. The time course curve for SvO₂ and ScvO₂ across varying conditions is shown in Figures 3 and 4 for two animals representing the high correlation while Figure 5 depicts the dog with the lowest correlation. The largest difference observed in the entire set of experiments is shown during the hypoxic period in Figure 5. Qualitatively, changes in ScvO₂ paralleled changes in SvO₂ and any time lag between the two was always less than one minute.

![Figure 3](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21595/ on 04/03/2017)
FIGURE 4. Time course of mixed and central venous O₂ saturation during different experimental perturbations of the animal. HTS = hypertonic saline solution (7.5%).

The comparison between $S\text{V}O_2$ and $S\text{cvO}_2$ during the different experimental conditions is summarized in Table 1. The correlation between the two was highest during control conditions and hyperoxia, slightly lower during hemorrhage and resuscitation, and lowest during hypoxia. The mean difference between $S\text{V}O_2$ and $S\text{cvO}_2$ ranged from 3 to 6 percent saturation for the different conditions, with the greatest differences observed during hypoxia and hemorrhage when saturations were lowest. Table 2 lists the relative frequency of the differences between $S\text{V}O_2$ and $S\text{cvO}_2$ for all comparisons combined. In 77 percent of the comparisons the difference between the two was less than 5 percent saturation.

FIGURE 5. Time course of mixed and central venous O₂ saturation in the animal that showed the lowest correlation between the two parameters. HTS = hypertonic saline solution (7.5%); Pentobarb = sodium pentobarbital.
Table 2 — Frequency of Differences Between Mixed and Central Venous \(O_2\) Saturation Expressed in Magnitude of Difference*

| \(|\text{SvO}_2-\text{ScvO}_2\)| | \(n\) | Relative Frequency, % |
|---|---|---|
| Diff<3% | 15883 | 53.8 |
| 3%<Diff<5% | 6928 | 23.5 |
| 5%<Diff<10% | 6029 | 20.4 |
| 10%<Diff | 691 | 2.3 |

*\(\text{SvO}_2-\text{ScvO}_2\)_d absolute value of difference between mixed and central venous \(O_2\) saturation; \(n\), number of comparisons within given range.

**DISCUSSION**

The \(O_2\) saturation values for the pulmonary artery and superior vena cava were generally in close accordance throughout all the experimental conditions of this study. Furthermore, during abrupt changes in the oxygen supply to demand ratio, changes in \(\text{ScvO}_2\) matched changes in \(\text{SvO}_2\) almost immediately. Hypoxia caused the greatest divergence of values with an average difference of −6 percent saturation and a worse-case difference of −20 percent. \(\text{ScvO}_2\) was expected to be slightly lower than \(\text{SvO}_2\) during steady-state conditions, due to a relatively large contribution of highly saturated venous renal effluent to the inferior vena cava. Our data in Table 1 are in general agreement with this expectation. In nonshock patients12,14 and healthy volunteers16 similar differences between mixed and central venous saturations have been reported. During hypoxia and hemorrhagic shock, a redistribution of blood flow away from renal and splanchic beds to the heart and brain would tend to reverse this difference.17,18 Such redistribution is consistent with the somewhat higher \(\text{ScvO}_2\) saturations we observed in both hemorrhage and hypoxia (Table 1). Similar relative increases in \(\text{ScvO}_2\) have been shown in clinical situations in which regional blood flow and/or oxygen consumption are affected such as shock,13,14 severe head trauma,19 or inhalational anesthetics (halothane).20 At least part of the larger difference between \(\text{SvO}_2\) and \(\text{ScvO}_2\) in shock in one earlier study could be because catheters did not reach the vena cava in half the patients.13

We propose that this technology will enable the clinician to observe changes in \(O_2\) saturation as they occur rather than relying on chance observations obtained by spot sampling. While \(\text{ScvO}_2\) can not replace \(\text{SvO}_2\) for exact calculations of \(O_2\) uptake or pulmonary shunt fraction, continuous monitoring of \(\text{ScvO}_2\) may provide a useful adjunct to noninvasive pulse oximetry in patient monitoring. Whereas pulse oximetry only indicates adequacy of arterial oxygenation, the addition of \(\text{ScvO}_2\) would reflect alterations in the relationship between total oxygen transport and tissue \(O_2\) consumption.5,7 The combination of the two could be used for continuous approximation of intrapulmonary shunt,21,24 and the addition of noninvasive techniques for cardiac output measurements would provide on-line trends in oxygen uptake. Conversely, if \(\text{Vo}_2\) were measured by gas exchange, cardiac output could be readily estimated. With such information the clinician would be better able to evaluate the effect of therapeutic interventions, such as changes in respiratory setting, fluid challenge, or the application of catecholamines or other vasoactive drugs, on the \(O_2\) supply/demand ratio.

The correlation between right atrial and mixed venous \(O_2\) saturations has been reported to be even higher than that between central and mixed venous blood.16,21 Recently, continuous fiberoptic monitoring of right atrial \(O_2\) saturation has been advocated as a less invasive alternative to PA catheterization.25 Right atrial catheterization, however, is not routinely practiced or recommended due to the danger of complications such as arrhythmia or atrial perforation.26,27 Central venous catheters, however, are routinely required in critically ill patients; thus, \(\text{ScvO}_2\) can be measured without additional risk.

The accuracy of modern in vitro monitoring devices for \(O_2\) saturation is generally very good. A high correlation between in vitro measured \(O_2\) saturation using a fiberoptic catheter and in vitro determination with a CO-oximeter has been shown in both human13,20 and animal studies.25,26 The output from fiberoptic catheters in the present study remained in close agreement with the reference CO-oximeter; in vitro recalibration due to saturation differences greater than 5 percent, averaged less than once per animal. Further, the overall correlation obtained with the in vitro technique (0.96) was almost as high as that which we obtained in our first series with in vitro determination (0.97).

To summarize, the differences between \(\text{ScvO}_2\) and \(\text{SvO}_2\), particularly when monitored continuously, are compensated by the lesser risks and costs associated with central venous catheterization. These data suggest that further clinical studies would be desirable to determine the extent to which continuous central venous \(O_2\) saturation monitoring could affect patient outcome.

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