Continuous Monitoring of Mixed Venous Oxygen Saturation during Aortic Surgery*

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The correlation between mixed venous oxygen saturation ($S\text{\textsubscript{V}}O\text{\textsubscript{2}}$) and hemodynamic measurements was studied in 13 patients undergoing descending thoracic aortic aneurysm resection (DTAAR). A significant correlation ($p<0.05$) was found between cardiac index (CI) and $S\text{\textsubscript{V}}O\text{\textsubscript{2}}$ after the induction of anesthesia and at the end of surgery. However, no significant correlation could be found between $S\text{\textsubscript{V}}O\text{\textsubscript{2}}$ and CI during the most critical periods of the surgery that included the collapse of the left lung, the aortic clamping, and the aortic declamping. During DTAAR, continuous $S\text{\textsubscript{V}}O\text{\textsubscript{2}}$ monitoring is useful, but it cannot substitute for intermittent cardiac output and oxygen consumption ($V\text{\textsubscript{O}}2$) determinations.

Maintenance of tissue normoxia is an important goal in patient care under all circumstances, but especially during unstable conditions such as surgery or other critical illness.\textsuperscript{1,2} While it is not possible presently to measure tissue oxygenation directly,\textsuperscript{3} its adequacy may be inferred from indirect measurements. For many years, serial determinations of arterial and mixed venous oxygen content, cardiac output (CO), systemic vascular resistance (SVR), and lactate levels have been the variables on which the physician relies to determine the adequacy of tissue oxygenation. However, by using these indices, it is possible to collect data only intermittently, and often there is a time delay between diagnosis and therapeutic intervention.

The assumption that the saturation of mixed venous blood ($S\text{\textsubscript{V}}O\text{\textsubscript{2}}$) was a good index of tissue oxygenation under most clinical circumstances prompted the introduction of the fiberoptic catheter which could continuously display $S\text{\textsubscript{V}}O\text{\textsubscript{2}}$. The fiberoptic catheter has proven to be reliable and useful in a variety of clinical circumstances; however, its usefulness during vascular surgery, and particularly during descending thoracic aortic aneurysm repair (DTAAR), has not been documented. We designed this prospective study to address this question.

**Patients and Methods**

Thirteen patients undergoing DTAAR using the technique of simple aortic cross-clamping without the use of a shunt or pump were included in the study. There were 12 men and one woman; the mean age was 62 years (range 22 to 73 years). The protocol was approved by the Human Research Review Board at our institution. In addition to thoracic aneurysms, preoperative risk factors included hypertension (55 percent), coronary artery disease (40 percent), previous myocardial infarction (25 percent), chronic obstructive pulmonary disease (50 percent), and diabetes mellitus (20 percent).

On the day of surgery, patients were premedicated with diphenhydramine (50 mg). After induction with fentanyl (50 µg/kg), diazepam (0.1 mg/kg), and pancuronium (0.1 mg/kg), intubation was accomplished with a double lumen endobronchial tube. Temporary single lung ventilation was used in all patients for better exposure of the aneurysm and to avoid trauma to the left lung and compression of the heart by retractors applied to the inflated left lung. In all cases, ventilation was accomplished with a tidal volume of 10 ml/kg, respiratory rate of 10/min and $F\text{\textsubscript{O}}2$ of 1. Adjustments in respiratory rate were made during the operation to maintain a $P\text{\textsubscript{CO}}2$ between 40 and 44 mm Hg. In addition to three peripheral venous catheters for volume replacement and therapeutic infusions, a 7.5 French four lumen balloon-tipped catheter mounted with two fiberoptic bundles was floated into the pulmonary artery through the internal jugular vein. Finally, a urinary catheter (Foley) and a rectal thermometer were inserted. Patients were placed in the right lateral decubitus position. Left thoracotomy was performed, and the left lung was collapsed. Before the aorta was cross-clamped, an infusion of nitroprusside was started at a rate of 3 µg/kg/min, and its rate was adjusted in order to prevent hypertension.\textsuperscript{4} A 1 molar bicarbonate infusion was started at the same time (0.05 mEq/kg/min).\textsuperscript{5} Immediately before the aortic clamp was removed, volume loading was increased, nitroprusside and sodium bicarbonate infusions were discontinued, and furosemide, 10 to 20 mg, was administered together with 50 mg of indigocarmine. During surgery, volume replacement was accomplished with crystalloid, fresh frozen plasma, and packed red blood cells. In addition, the blood was suctioned from the operative field, washed, and reinfused as packed red blood cells with a hematocrit value of 50 to 60 percent using the Baylor rapid antologous transfusion system. The choice of the infused fluid was determined in order to maintain hemoglobin concentration within a range of normal values. The rate and volume of fluid were adjusted to maintain pulmonary capillary wedge pressure (PCWP) within a range of 8 to 13 mm Hg. From the pulmonary catheter, pulmonary arterial pressure (PAP) and $S\text{\textsubscript{V}}O\text{\textsubscript{2}}$ were recorded continuously. The PCWP and CO were determined intermittently in duplicate using an Edwards cardiac output computer. Arterial pressure (AP) was directly measured from a radial catheter via a Statham PD233 transducer. At the time of hemodynamic measurements, arterial and mixed venous blood samples were drawn to determine arterial venous pH, arterial pressure of oxygen ($PaO2$), and carbon dioxide ($PaCO2$). From the hemodynamic varia-

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Table 1—Hemodynamic Values

<table>
<thead>
<tr>
<th></th>
<th>Control Supine</th>
<th>R Lat Decubitus Chest Closed</th>
<th>R Lat Decubitus Chest Open</th>
<th>1 Lung</th>
<th>Aortic Clamp</th>
<th>Aortic Declamp</th>
<th>End Supine Chest Closed</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP* mm Hg</td>
<td>78 ± 7.7</td>
<td>80 ± 3.5</td>
<td>80 ± 2.7</td>
<td>80 ± 2.4</td>
<td>95 ± 2.6†</td>
<td>73 ± 3.4</td>
<td>94 ± 3</td>
</tr>
<tr>
<td>HR beats × min</td>
<td>79 ± 4.2</td>
<td>84 ± 4.7</td>
<td>77 ± 4.3</td>
<td>86 ± 5.2</td>
<td>95 ± 4.5†</td>
<td>100 ± 4.6†</td>
<td>94 ± 3.5†</td>
</tr>
<tr>
<td>CVP mm/Hg</td>
<td>11 ± 0.8</td>
<td>7.5 ± 0.6†</td>
<td>9 ± .9</td>
<td>11 ± 0.8</td>
<td>11 ± 1.1</td>
<td>11 ± .83†</td>
<td>10 ± .73</td>
</tr>
<tr>
<td>CMS H₂O <em>L/min/m²</em></td>
<td>13 ± 0.5</td>
<td>10 ± .5†</td>
<td>12 ± 0.6</td>
<td>13 ± 3.6</td>
<td>14 ± 1.0</td>
<td>13 ± 1.24</td>
<td>12 ± .76</td>
</tr>
<tr>
<td>PCWP mm/Hg</td>
<td>2.4 ± 0.24</td>
<td>2.2 ± 0.2</td>
<td>2.6 ± 0.19</td>
<td>2 ± 0.2</td>
<td>2.1 ± 0.28</td>
<td>3.6 ± 0.34†</td>
<td>2.7 ± 0.22</td>
</tr>
<tr>
<td>CI <em>g/mm/m²</em></td>
<td>27 ± 2.8</td>
<td>25 ± 2.9</td>
<td>32 ± 2.3</td>
<td>24 ± 0.6</td>
<td>25 ± 2.6</td>
<td>28 ± 1.8</td>
<td>29 ± 2.3</td>
</tr>
<tr>
<td>SVR <em>Dynes/cm²</em></td>
<td>1282 ± 121</td>
<td>1591 ± 226</td>
<td>1179 ± 92</td>
<td>1505 ± 142</td>
<td>1991 ± 250†</td>
<td>834 ± 130</td>
<td>1220 ± 157</td>
</tr>
<tr>
<td>Po₂ <em>mm/Hg</em></td>
<td>474</td>
<td>465 ± 15</td>
<td>473 ± 20.9</td>
<td>413 ± 30</td>
<td>477 ± 37</td>
<td>231 ± 32†</td>
<td>382</td>
</tr>
<tr>
<td>SVO₂ %</td>
<td>84 ± 2.7</td>
<td>86 ± 1†</td>
<td>89 ± 1.8</td>
<td>89 ± 1.6†</td>
<td>95 ± 1.5</td>
<td>74 ± 3.1†</td>
<td>91 ± 1.62†</td>
</tr>
<tr>
<td>VO₂ <em>ml/min/m²</em></td>
<td>88 ± 5.9</td>
<td>76 ± 5.25</td>
<td>42 ± 5.6†</td>
<td>154 ± 20.1†</td>
<td>70 ± 4</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*MAP is mean arterial pressure; HR, heart rate; CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; CI, cardiac index; LVSWI, left ventricular stroke work index; SVR, systemic vascular resistance; Po₂, arterial pressure of oxygen; SVO₂, mixed venous oxygen saturation; VO₂, oxygen consumption.
†p<0.05 Wilcoxon signed rank test.

bles recorded (CI and SVR), left ventricular stroke work index (LVSWI), and oxygen consumption (VO₂) were also calculated. Patients were studied immediately after the induction of anesthesia in supine and right lateral decubitus positions while the chest was still closed, again after thoracotomy, after the left lung was deflated, after aortic clamping, after aortic declamping, and after closure of thoracotomy.

Data were analyzed using the Wilcoxon signed ranks test. Alpha was set up at 0.05. In addition, the correlation between SVO₂, CI, VO₂, mean AP, SVR, PaO₂, and LVSWI changes were established. Data are presented as mean ± SEM.

RESULTS

Hemodynamic values collected at different intervals during surgery are presented in Table 1 and Figure 1. Only the significant changes are reported. In the right decubitus position, PCWP and central venous pressure (CVP) decreased by 23 percent and 32 percent, respectively. Immediately after aortic declamping, we recorded an increase in CI of 50 percent. The VO₂ decreased by 52 percent during aortic clamping and increased by 75 percent during aortic declamping.

Compared to values recorded after induction of anesthesia in the supine position, SVO₂ increased by 2 percent in the right decubitus position, 13 percent after aortic clamping, and 8 percent at the end of the surgery when the chest was closed. Additionally, a 12 percent decrease in SVO₂ was recorded immediately after aortic declamping. Decreases in PaO₂ of 13 percent and 52 percent, respectively, were also observed as a result of collapsing one lung and aortic declamping.

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21569/ on 06/24/2017)
A linear relationship between $\text{SVO}_2$ and CI was obtained when the chest was closed both before the beginning and at the end of surgery, while no correlation could be demonstrated between $\text{SVO}_2$ and $\text{Vo}_2$.

**DISCUSSION**

The continuous monitoring of $\text{SVO}_2$ has a prominent place in the daily care of critically ill patients. Modern technology has overcome many of the problems associated with the initial use of reflective spectrophotometry. Current oxymetric catheters are accurate over the range of 30 to 95 percent saturation and only require calibration every 24 hours. Before 1980, the clinical application of $\text{SVO}_2$ was primarily in infants. In dealing with postcardiac surgery in children, De la Rocha et al reported a good correlation between $\text{SVO}_2$ and CO. In a group of infants with respiratory failure, Wilkinson et al concluded that $\text{SVO}_2$ greater than 85 percent indicated a stable state and avoided frequent blood sampling for arterial blood gases. In adults with ARDS, the continuous monitoring of $\text{SVO}_2$ has been useful in the determination of the level of positive end-expiratory pressure associated with the greatest $\text{O}_2$ delivery to tissues (optimal PEEP). Also in this group of patients, continuous monitoring of $\text{SVO}_2$ has obviated the need for frequent determination of arterial blood gases during weaning. In coronary care units, monitoring of $\text{SVO}_2$ has been useful in determining the value of therapeutic intervention and hemodynamic stability. During cardiac surgery, a good correlation between CI and $\text{SVO}_2$ has been found by some investigators, conversely, Kneus and Venden reported lack of correlation between $\text{SVO}_2$ and CI.

In our study, we found a significant correlation between $\text{SVO}_2$ and CI only immediately after the induction of anesthesia, when the patient was turned to the right lateral position, and at the end of surgery. No significant correlation between these values was found at any other time during the surgery. If we consider that the mathematical expression for $\text{SVO}_2$ is

$$\text{SVO}_2 = \text{SaO}_2 - (\text{Vo}_2/\text{hemoglobin} \times 1.36 \times \text{CO}) \times 10,$$

a significant correlation between $\text{SVO}_2$ and CI is logical and easy to understand in a relatively stable group of patients in which the hemoglobin and $\text{Vo}_2$ are relatively constant. However, in patients undergoing DTAAR, changes in CO, $\text{Vo}_2$, and arterial content of oxygen occur rapidly, making it difficult to correlate directly the value of $\text{SVO}_2$ with only one of those three variables. Moreover, simple manipulation of the variables in the equation will demonstrate that rather dramatic changes in the relationship between total body oxygen supply and demand occur with little or no change in $\text{SVO}_2$.

Cross-clamping of the aorta imposes an interesting phenomenon when measuring $\text{SVO}_2$. The reading obtained is the result of a mixed venous blood coming from different vascular beds. Individual organ and tissue beds may be notably underperfused, yet $\text{SVO}_2$ may be normal or even supranormal. In our group of patients, when the aorta was clamped, a significant and uniform rise in $\text{SVO}_2$ was observed secondary to a markedly decreased $\text{Vo}_2$. The physiologic explanation for the rise in $\text{SVO}_2$ and the decrease in $\text{Vo}_2$ with an unchanged $\text{Vo}_2$ is the significant reduction in the arteriovenous oxygen content difference. The extraction of oxygen is limited by the precarious flow to many vascular beds during aortic clamping even in the presence of a "normal" CO.

The question of the validity of calculated $\text{Vo}_2$ versus actual measurement could be raised. However, different authors have shown a good correlation between these two methods of obtaining the $\text{Vo}_2$.

It is our conclusion from the present study that during DTAAR, once the chest is opened, there is no significant correlation either between $\text{SVO}_2$ and CO, or between $\text{SVO}_2$ and $\text{Vo}_2$. During DTAAR, normal values of $\text{SVO}_2$ do not preclude adequate tissue oxygenation, $\text{SVO}_2$ monitoring, therefore, cannot substitute for intermittent CO and $\text{Vo}_2$ determinations. Our results are in agreement with those of other investigators in the sense that under most conditions, $\text{SVO}_2$ reflects overall oxygen extraction.

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Chest Imaging, 1988

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