Continuous Monitoring of Mixed Venous Oxygen Saturation in Hemodynamically Unstable Patients*

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A balloon-tipped catheter has recently become available which, when placed in the pulmonary artery, in addition to enabling the usual measurements of pulmonary arterial pressure, pulmonary capillary wedge pressure, and cardiac output by thermodilution, measures mixed venous oxygen saturation (SVO₂) by spectrophotometry. Unlike the measurement of cardiac output by thermodilution, which is done intermittently, the continuous measurement of SVO₂ is an effective method of monitoring hemodynamically unstable patients, since changes in cardiac output will immediately become apparent via a corresponding change in SVO₂. This is of particular benefit in patients in whom knowledge of the immediate effects of therapy is important. It is also of value in assessing the time of onset of action and duration of action when a cardioactive drug is given to increase cardiac output. We suggest that monitoring SVO₂ will provide an earlier indication of the effect of both diagnostic and therapeutic interventions and, therefore, will improve our management in such patients.

Although the intermittent measurement of cardiac output by thermodilution (COₑ) has, in recent years, been used to monitor the hemodynamically unstable patient and to assess the response to pharmacologic therapy, the technique does have drawbacks which limit its usefulness in both of these conditions. Because cardiac output is measured intermittently, it cannot be relied upon to signal an acute deterioration in clinical status. Indeed, usually a clinical event, such as hypotension, tachycardia, or oliguria, must occur first, which then prompts the measurement of cardiac output. Used in this way, measuring cardiac output is not a sensitive way to detect a worsening clinical condition, since it relies on the clinical appearance of deterioration, which may be a late manifestation. Using measurements of cardiac output to assess the effect of therapy with drugs suffers from the same limitations. First, the onset of action of the drug will not be accurately determined when cardiac output is measured intermittently, and the duration of activity will also be difficult to assess. It is possible that the significant effect of a vasoactive drug will be missed when monitoring is done by the sporadic measurement of COₑ. Furthermore, titration of the administration of a drug, such as sodium nitroferricyanide (sodium nitroprusside), to achieve optimum results in critically ill patients is difficult and time-consuming if intermittent measurements of cardiac output are used in making decisions. Thus, although cardiac output is an important measurement, the ability to measure it only intermittently with thermodilution makes it less valuable.

Recently, a flow-directed balloon-tipped pulmonary arterial catheter has been introduced (Oximetrix Shaw catheter-oximeter system) that, in addition to the standard measurements of pulmonary arterial pressure, pulmonary capillary wedge pressure, and COₑ, measures via spectrophotometry the oxygen saturation of blood when connected to an oximeter at bedside. The measured saturation is displayed at the bedside and optionally recorded on a strip-chart recorder. Accordingly, when properly placed in the pulmonary artery, this catheter measures mixed venous oxygen saturation (SVO₂). In a patient in whom the hemoglobin level and arterial oxygen saturation (SaO₂) are constant and in whom there is no acute change in oxygen consumption, the SVO₂ reflects cardiac output. Thus, measuring SVO₂ continuously in a hemodynamically unstable patient is, indirectly, a method of continuously monitoring the cardiac output and provides an additional sensitive way of detecting a change in a patient’s clinical condition. In addition, measuring SVO₂ on a continuous basis will identify the onset and duration of action of a drug that is given to increase cardiac output. The purpose of this communication is to illustrate the value of such a catheter in managing hemodynamically unstable patients.

CASE REPORTS

CASE 1

A 55-year-old woman was admitted to the coronary care unit with an acute myocardial infarction. Because of oliguria and hypotension, a thermodilution flow-directed catheter capable of measuring oxygen saturation was inserted.

Figure 1 is a tracing of SVO₂. Note the increase in SVO₂ from 32 percent to 51 percent after the infusion of dopamine was started. The COₑ increased from 2.0 L/min to 3.1 L/min (Table 1).

CASE 2

A 52-year-old man with a history of hypertension was admitted with acute renal failure. In the recovery room, after an open renal biopsy to ascertain the cause of the renal failure, he developed pain in the chest and shortness of breath and was noted to have pulmonary edema. The patient was admitted to the coronary care unit, where a thermodilution flow-directed catheter capable of measuring oxygen saturation was inserted.

Note the abrupt increase in SVO₂ (Fig 2) after therapy with sodium nitroferricyanide was begun, signifying an increase in cardiac output. Table 1 details the changes in hemodynamic variables prior to

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and after therapy. Changes in the dosage of the drug could be guided by following such changes in SVO₂, in addition to other variables such as blood pressure and capillary wedge pressure.

These two patients (cases 1 and 2) illustrate the use of continuous monitoring of SVO₂ in critically ill unstable patients in an intensive care unit or coronary care unit. In each instance, the availability of continuous measurements of SVO₂ enabled the effect of the administered cardioactive drug to be seen immediately. The following two cases illustrate the application of continuous monitoring of SVO₂ in documenting the onset and duration of action of medications given to increase cardiac output in patients with chronic congestive heart failure.

**CASE 3**

A 73-year-old man had a prolonged history of hypertension, ischemic heart disease, and chronic renal insufficiency. He had had two myocardial infarctions in the past, with persistent hypertension and congestive heart failure refractory to therapy with digoxin, diuretics, and oral hydralazine. A two-dimensional echocardiogram revealed marked concentric left ventricular hypertrophy with good systolic function. A thermodilution flow-directed catheter capable of measuring oxygen saturation was inserted.

Hemodynamic data at 25 minutes before and one hour after receiving an oral 10-mg dose of nifedipine are shown in Table 1.

**CASE 4**

A 47-year-old woman with severe congestive cardiomyopathy was admitted for trial of therapy with amrinone. A thermodilution flow-directed catheter capable of measuring oxygen saturation was inserted.

Figure 4 is a tracing of SVO₂ just before the amrinone was given and for approximately three hours after the drug was administered. Cardiac output was measured just before and at 60 minutes and 160 minutes after the drug was given (Table 1). The timing of the measurements of cardiac output was guided by the observed changes in SVO₂. If cardiac output was measured earlier than 60 minutes or later than 160 minutes after the administration of the drug, the changes in cardiac output might have been missed. Thus, monitoring the SVO₂ allows us to adjust the dosing interval of the drug in a way which would be difficult if CO₉₀ alone was used.

**DISCUSSION**

These four patients illustrate the value of using continuously monitored SVO₂ as an estimate of cardiac output. This direct application of SVO₂ assumes a relatively constant hemoglobin level and SaO₂, since a decrease in either of these two variables, as well as a decrease in cardiac output, will result in a fall in SVO₂; however, both hemoglobin level and SaO₂ are easily measured, and there should be no difficulty in recognizing these changes. Furthermore, it is an advantage, not a drawback, that SVO₂ will decrease when there is anemia or arterial destruction, since these events should be drawn to the attention of the physician and may be acted upon promptly in an otherwise overlooked situation. Clinically important examples of this include the patient with occult bleeding and the patient with pulmonary disease. The SVO₂ would fall in both of these situations, even with a maintained cardiac output, since both anemia and arterial desaturation, respectively, reduce oxygen transport and delivery to the tissues.

Finally, it is true that even if the hemoglobin level and SaO₂ remain constant, SVO₂ may change without a corre-
**Table 1—Hemodynamic Data in Patients Studied with Continuous Monitoring of SVO₂**

<table>
<thead>
<tr>
<th>Data</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
</tr>
<tr>
<td></td>
<td>(Prior to nitroprusside)</td>
<td>(85 min Later)</td>
<td>(15 min Later)</td>
<td>(Prior to amrinone)</td>
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<tr>
<td>CO₂P, L/min</td>
<td>2.0</td>
<td>3.1</td>
<td>3.1</td>
<td>4.6</td>
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<td>SVO₂, percent</td>
<td>32</td>
<td>51</td>
<td>42</td>
<td>64</td>
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<tr>
<td>Blood pressure, mm Hg</td>
<td>105/60</td>
<td>110/74</td>
<td>160/90</td>
<td>120/60</td>
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<tr>
<td>Pulmonary arterial pressure, mm Hg</td>
<td>50/32</td>
<td>42/26</td>
<td>43/22</td>
<td>34/16</td>
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<td>Pulmonary wedge pressure, mm Hg</td>
<td>32</td>
<td>24</td>
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<td>16</td>
</tr>
<tr>
<td>PaO₂, mm Hg*</td>
<td>74</td>
<td>139</td>
<td>78</td>
<td>107</td>
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<tr>
<td>SVO₂, percent</td>
<td>93</td>
<td>99</td>
<td>93</td>
<td>98</td>
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<td>Hemoglobin level, g/100 ml</td>
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<td>12.4</td>
<td>9.3</td>
<td>10.4</td>
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<tr>
<td>VO₂, ml/m†</td>
<td>212</td>
<td>259</td>
<td>197</td>
<td>218</td>
</tr>
</tbody>
</table>

*Arterial oxygen tension.
†Oxygen consumption (VO₂ = cardiac output × arteriovenous oxygen content difference).

**Figure 3.** Tracing of SVO₂ in patient 3. At point A, cardiac output was 4.6 L/min, and SVO₂ was 39 percent. Ten milligrams of nifedipine was given orally at point B. At point C (55 minutes later), cardiac output was 6.6 L/min, and SVO₂ was 51 percent.

**Figure 4.** Tracing of SVO₂ in patient 4. At point A, cardiac output was 1.8 L/min, and SVO₂ was 17 percent. Amrinone (75 mg) was given orally immediately after A. At point B (60 minutes later), cardiac output was 2.9 L/min, and SVO₂ was 53 percent. At point C (160 minutes after A), cardiac output was 1.9 L/min, and SVO₂ was 24 percent.
spending change in cardiac output if oxygen consumption changes; however, major changes in oxygen consumption are unlikely in patients restricted to limited activity, except in easily detectable clinical events such as generalized seizures, exercise, or fever. Moreover, the same considerations are essential when CO,,, is measured intermittently during monitoring of therapy such as a trial of a drug. Either an increase in oxygen consumption, a fall in hemoglobin level, or arterial desaturation would lead to a compensatory rise in cardiac output, which would make it difficult to attribute any increase in cardiac output to an effect of the drug.

In summary, the continuous measurement of SvO₂ by an indwelling pulmonary arterial catheter is a more appropriate way to monitor hemodynamically unstable patients than is the intermittent measurement of CO,. Such measurements of SvO₂ provide a means of early recognition of hemodynamic deterioration in the critically ill patient. These measurements are also valuable in guiding therapy with drugs by indicating the onset of action and the effects and duration of such therapy. Finally, measurement of SvO₂ can be used as a means of determining when cardiac output should be measured by thermodilution.

REFERENCES