blood sampling and/or blood pressure measurement, and inexpensive.

An electrode designed for this purpose was first described by Parker in 1971 and used by Huch in newborns. Modification of this electrode has since been incorporated into umbilical arterial catheters to allow monitoring of oxygen tension in premature infants to prevent hypoxaemia and to minimize the occurrence of retrolental fibroplasia. This electrode has also been mounted on catheter tips for monitoring of PO2 in critically ill patients. The early sensors, however, were fraught with problems including wide in vivo drift partly due to fibrin deposition, and partly due to flow dependency. As well, some sensors simply failed to activate. Many of these problems have been overcome with the current generation of sensors. In vivo drift is now less than ten per cent in 24 hours, stabilization time is about ten minutes, and 90 per cent response to change in oxygenation is complete within 120 seconds. It is little influenced by change in flow, and change in temperature can be corrected. Good correlations have been demonstrated in critically ill patients and newborn infants. In a comparative study in premature infants, Finer showed that more episodes of hypoxaemia were observed with the continuous PO2 sensor than using the conventional method of intermittent blood gas sampling. Few studies have been conducted during anaesthetic conditions, and our own study indicated that its performance was satisfactory during hypotensive anaesthesia.

Despite these promising reports, this type of monitoring has not gained popularity and there are many reasons. In terms of anaesthetic applications, it cannot be used during halothane anaesthesia because halothane is also reduced at the cathode and causes a falsely high reading. The technique requires an in situ blood sample calibration, and in order to allow simultaneous recording of arterial blood pressure, at least an 18-gauge catheter must be used. Perhaps the most important reasons against its routine use are the invasive nature of the monitor and the cost entailed. The lack of clear indications for its use also hampers its utilization.

A newer generation of catheter tip-mounted sensors with further improvement over predecessors is now being developed. Heparin-bonded catheters will minimize fibrin deposit and therefore decrease the in vivo drift. The utilization of fiberoptic sensors will allow only on-line PO2 monitoring, but also PCO2 as well as pH. Although these new sensors have not yet reached the stage of clinical trial and the cost factor remains a prohibitive one, the need for a method of continuously measuring the oxygen tension of blood in vivo exists today as it did ten years ago and though ear or pulse oximetry would satisfy the demand for continuous monitoring of oxygenation in most situations, in selected cases where invasive monitoring is indicated and where rapidly deteriorating arterial oxygenation can occur, continuous monitoring of PO2 would be highly desirable.

References

Mixed venous oxygen saturation

The development of flow-directed balloon-tipped catheters has provided the opportunity to measure pulmonary artery pressures, pulmonary occlusion pressures ("wedge" pressure) and cardiac outputs on a routine basis. Use of these data can be improved by obtaining and interpreting continuous mixed venous oxygen saturations ($\text{SvO}_2$) from the pulmonary artery catheter. Only thoroughly mixed blood which flows from capillary beds that extract oxygen while not being contaminated by blood which may have
TABLE Interpretation of \( \text{S'O}_2 \) values

<table>
<thead>
<tr>
<th>( \text{S'O}_2 )</th>
<th>Variables</th>
<th>Clinical interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>80-95%</td>
<td>( \text{O}_2 ) Supply (( \text{S'O}_2 ) ↑)</td>
<td>↑ ( \text{FiO}_2 )</td>
</tr>
<tr>
<td></td>
<td>( \text{O}_2 ) Demand (( \text{V'O}_2 ) ↓)</td>
<td>Hypothermia, anaphylaxis, paralysis (relaxants)</td>
</tr>
<tr>
<td></td>
<td>Haemodynamics (( \text{CO} ) ↑)</td>
<td>Septic shock, vasodilation, catheter migration</td>
</tr>
<tr>
<td>60-80%</td>
<td>( \text{O}_2 ) Supply normal</td>
<td>Adequate tissue perfusion</td>
</tr>
<tr>
<td></td>
<td>( \text{O}_2 ) Demand normal</td>
<td>Hyperthermia, shivering, position, pain, shivering</td>
</tr>
<tr>
<td></td>
<td>( \text{CO} ) Adequate</td>
<td>Cardiac tamponade, cardiogenic shock, tension pneumothorax, arrhythmias, shock, vasoconstriction, excessive PEEP</td>
</tr>
<tr>
<td>30-60%</td>
<td>( \text{O}_2 ) Supply (( \text{S'O}_2 ) ↓)</td>
<td>Anemia, airway obstruction, endotracheal suctioning</td>
</tr>
<tr>
<td></td>
<td>( \text{O}_2 ) Demand (( \text{V'O}_2 ) ↑)</td>
<td>Hyperthermia, shivering, position, pain, shivering</td>
</tr>
<tr>
<td></td>
<td>Unstable Haemodynamics (( \text{CO} ) ↓)</td>
<td>Cardiac tamponade, cardiogenic shock, tension pneumothorax, arrhythmias, shock, vasoconstriction, excessive PEEP</td>
</tr>
</tbody>
</table>

bypassed capillaries due to pre- or post-capillary shunts can be considered true mixed venous blood. Pulmonary arterial blood, with rare exceptions, fulfills these requirements.

\( \text{S'O}_2 \) reflects the status of the oxygen supply/transport system. This system consists of oxygen demand and oxygen extraction components.\(^1\)

Oxygen demand is directly related to the metabolic rate which may be altered by anaesthetics, temperature, endocrine function, muscular work and disease states such as sepsis and shock. The oxygen requirements of each organ vary according to the activity of the organ and regional differences commonly occur. Oxygen extraction, or oxygen consumption, is the amount of oxygen actually used by the cells, tissue and organs. Under normal and steady-state conditions, oxygen demand and oxygen extraction are equal. The oxygen transport system may be so compromised in shock that demand exceeds extraction resulting in anaerobic metabolism producing lactic acidosis. Such an event is invariably associated with life-threatening disease and an ominous prognosis.

Oxygen supply/transport to cells and tissues is dependent on cardiac output, haemoglobin concentration and the arterial oxygen saturation. With a cardiac output of 5 L·min\(^{-1}\), a haemoglobin of 15 g·dL\(^{-1}\), an arterial \( \text{O}_2 \) saturation of 97 per cent and normal oxygen extraction, the \( \text{S'O}_2 \) will approximate 75 per cent.\(^2\) Any disease process which causes anaemia, decreased cardiac output or arterial \( \text{O}_2 \) desaturation stimulates compensatory processes to meet the oxygen demand by increasing cardiac output or increasing oxygen extraction. Cardiac output may triple (from 5 to 15 L·min\(^{-1}\)) while \( \text{S'O}_2 \) may decrease to as little as 31 per cent thus tripling the arterial-venous oxygen saturation difference from a normal of 22 per cent (97 to 75 per cent) to 66 per cent (97 to 31 per cent). A nine-fold protective mechanism is thus possible assuming cardiac performance and tissue extraction are not compromised.\(^2\)

Compensatory mechanisms may include changes in viscosity, vasodilation, preload, afterload, sympathetic tone and increased oxygen extraction. The onset of lactic acidosis heralds the inability to meet oxygen consumption requirements. Once this finite level of \( \text{S'O}_2 \) is reached, compensatory mechanisms for matching oxygen demand and oxygen consumption are exhausted. This key value of \( \text{S'O}_2 \) is difficult to estimate because of metabolic and shunt factors - a commonly accepted value is a \( \text{S'O}_2 \) of less than 40 per cent. As a general rule, a significant decrease in \( \text{S'O}_2 \) (10-20 per cent) indicates to the clinician that a careful review of the patient is indicated.\(^3\) An absolute value of \( \text{S'O}_2 \) less than 40 per cent suggests compensatory mechanisms have been exhausted, with lactic acidosis likely to evolve with a corresponding dismal prognosis (Table).

These conclusions are supported by correlations observed between \( \text{S'O}_2 \) and the prognosis in respiratory failure, myocardial infarction, septic shock, cardiac tamponade and tension pneumothorax. Reversal of the falling \( \text{S'O}_2 \) or reversal of the trend indicated by continuous monitoring, has resulted in improved management of patients in a postoperative cardiac unit.\(^3\)

Seemingly paradoxical increases in \( \text{S'O}_2 \) may be seen in patients who clinically have compromised tissue oxygenation. This situation occurs in some cases of sepsis where the \( \text{S'O}_2 \) exceeds 60 per cent in the presence of lactic acidosis. A failure of oxygen extraction or contamination of mixed venous blood due to peripheral arteriole shunts, may occur in haemorrhagic or septic shock. Maldistribution of blood flow (shifts from skin, kidney) may result in lactic acidosis in the end organ with concomitant normal \( \text{S'O}_2 \) values. A normal or high \( \text{S'O}_2 \) in the presence of significant hypotension suggests that an immediate review of the patient is necessary.

This discussion can be summarized by reviewing the Fick principle and its relationship to oxygen, haemoglobin and blood flow as suggested by Schmidt et al.\(^5\)

Cardiac Output (\( \text{CO} \)) =

\[
\text{Arterial O}_2 \text{ Content (CaO}_2\) \ - \ \text{Venous O}_2 \text{ Content (CvO}_2\)}
\]
Since
\[ \frac{\dot{O}_2}{DO_2} = \text{oxygen supply or delivered} = (CAO_2)(CO) \]
\[ \frac{V_O_2}{DO_2} = \text{oxygen extraction rate} \]
then the Fick equation becomes:
\[ \frac{\dot{O}_2}{DO_2} = \frac{V_O_2}{CaO_2} - \frac{(CvO_2)}{CaO_2} \]
Simplified:
\[ \frac{\dot{O}_2}{DO_2} = \frac{CaO_2 - (CvO_2)}{CaO_2} = 1 - \frac{CvO_2}{CaO_2} \]
Since
\[ CvO_2 = So_2 \]
and
\[ CAO_2 = SaO_2 \]
\[ \frac{\dot{O}_2}{DO_2} = \frac{1 - So_2}{SaO_2} \]
but
\[ SaO_2 = 100\% \]
\[ \therefore \text{oxygen extraction ratio} = 1 - So_2 \]

Thus when SaO_2 is 100 per cent continuous Svo_2 measurement reflects continuous oxygen extraction. The normal patient with Svo_2 of 75 per cent has an oxygen extraction ratio of 25 per cent based on the assumptions of close to 100 per cent arterial saturation, negligible oxygen in solution, constant haemoglobin and no lactic acidosis.

This expression allows one to continuously track oxygen transport variables as shown in the Table, even though there may not be a direct correlation with cardiac output alone.

Continuous Svo_2 monitoring may provide early warning of a deleterious event in a patient's recovery process. This useful indicator of circulatory imbalance may help in the intraoperative management of the difficult patient such as those undergoing cardiac surgery, vascular surgery or those who are haemodynamically unstable or in shock.

References

Monitoring with transcutaneous PO_2 and pulse oximetry devices

In spite of its paramount importance, oxygenation has only been monitored in the past by the absence of the clinical signs of inadequate oxygenation. These signs, which include cyanosis, tachycardia or bradycardia, hypertension or hypotension, hyperventilation or hyperventilation, are subjective, nonspecific and unreliable. Over the past 20 years technological advances have allowed the development of devices for the objective assessment of oxygenation, first by analysis of blood samples and more recently, by continuous monitoring techniques.

This article will describe the experimental and clinical aspects of the two currently available noninvasive oxygen monitoring techniques: transcutaneous PO_2 (PtcO_2) and pulse oximetry (NSaO_2).

Transcutaneous PO_2 (PtcO_2)
The transcutaneous oxygen sensor utilizes a polarographic oxygen electrode to measure oxygen that diffuses to the skin surface from the dermal capillary bed beneath it. To obtain a reliable PO_2 reading with a fast response time, the skin must be heated to at least 43°C. The heat affects the three basic layers of the skin, which in turn influence the PO_2 measured at the surface. The increase in