linked to systemic arterial pressure. From a practical standpoint, at operation, it is possible to relieve turgor and pulsatility in subarachnoid conducting vessels and associated aneurysms by lowering arterial blood pressure. We did observe that, with the elevated systemic blood pressures reported in these cases, bounding subarachnoid vessels were encountered in the non-spatitic segments. Using careful sharp microdissection, we felt that it was possible to operate relatively safely on the hyperdynamic cerebral vessels. Despite the anticipated brief interval of relative “hypotension” of 150 mmHg used in the second case, we felt that this preoperatively symptomatic level of blood pressure could potentially place the patient at risk for ischemic complications. Due to that concern, we empirically administered etomidate 0.2 mg/kg for potential cerebral protection. Recent encouraging experience in this institution with use of etomidate-induced burst-suppression for temporary arterial occlusion in the management of giant cerebral aneurysms supports this maneuver.6 The marked cardiovascular stability and shorter elimination half-life of etomidate makes it an attractive agent for this use.9,10

In summary, the use of induced hypertension and hypertervolemia as part of the anesthetic management of intracranial aneurysms is described. This technique may have a role to play in the management of a select group of patients suffering from symptomatic vasospasm after SAH, as part of the definitive surgery for their lesion and ongoing optimal medical therapy.

REFERENCES


Unplanned Intraoperative and Postoperative Hemodilution: Oxygen Transport and Consumption during Severe Anemia

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Treatment of unanticipated severe intraoperative blood loss in a patient who refuses blood transfusion presents a particularly difficult dilemma for the anesthesiologist. To effectively treat such a patient for a short time, oxygen delivery can be increased while minimizing oxygen consumption. Although others have described survival of anemic patients despite a low hematocrit,1 our patient had hemodynamic variables and gas exchange measured with the help of a pulmonary artery catheter during severe anemia.

REPORT OF A CASE

A 37-yr-old, 47-kg, Jehovah’s Witness woman with a year-long history of Crohn’s Disease was admitted for a small bowel resection and
Table 1. Arterial and Mixed Venous Blood Gas Data

<table>
<thead>
<tr>
<th>Type of Sample</th>
<th>Sample Number</th>
<th>Temp °C</th>
<th>Hct %</th>
<th>Fio₂</th>
<th>pH</th>
<th>pCO₂ mmHg</th>
<th>pO₂ mmHg</th>
<th>SaO₂ STO₂</th>
<th>Base Deficit mEq/L</th>
<th>CaO₂ g/dl</th>
<th>CO₂ ml/O₂/100 ml</th>
<th>CO₂ ml/O₂/100 ml</th>
<th>DO₂ ml/O₂/min</th>
<th>VO₂ ml/O₂/min</th>
<th>ER %</th>
<th>CO l/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial</td>
<td>1</td>
<td>32</td>
<td>5</td>
<td>1.0</td>
<td>7.48</td>
<td>28</td>
<td>580</td>
<td>99.9</td>
<td>3</td>
<td>2.2</td>
<td>2.0</td>
<td>0.1</td>
<td>272</td>
<td>138</td>
<td>50</td>
<td>6.5</td>
</tr>
<tr>
<td>Mixed venous</td>
<td>2</td>
<td>30</td>
<td>4</td>
<td>1.0</td>
<td>7.53</td>
<td>27</td>
<td>555</td>
<td>99.9</td>
<td>1</td>
<td>1.8</td>
<td>1.9</td>
<td>0.1</td>
<td>197</td>
<td>105</td>
<td>53</td>
<td>5.3</td>
</tr>
<tr>
<td>Arterial</td>
<td>3</td>
<td>29</td>
<td>22</td>
<td>1.0</td>
<td>7.30</td>
<td>47</td>
<td>477</td>
<td>99.9</td>
<td>3</td>
<td>11.5</td>
<td>9.8</td>
<td>0.2</td>
<td>587</td>
<td>92</td>
<td>15</td>
<td>5.1</td>
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<tr>
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<td>4</td>
<td>33</td>
<td>24</td>
<td>.4</td>
<td>7.43</td>
<td>34</td>
<td>182</td>
<td>99.7</td>
<td>1</td>
<td>11.3</td>
<td>10.7</td>
<td>0.6</td>
<td>542</td>
<td>262</td>
<td>51</td>
<td>4.8</td>
</tr>
<tr>
<td>Arterial</td>
<td>5</td>
<td>37</td>
<td>28</td>
<td>.21</td>
<td>7.44</td>
<td>39</td>
<td>98</td>
<td>97.3</td>
<td>-2</td>
<td>12.5</td>
<td>12.2</td>
<td>0.3</td>
<td>623</td>
<td>33</td>
<td>33</td>
<td>5.0</td>
</tr>
<tr>
<td>Mixed venous</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>7.38</td>
<td>47</td>
<td>33</td>
<td>65.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Oxygen saturations calculated with Severinghaus's equations, correcting for changes in temperature and pH. 3 Dissolved oxygen contents calculated with Hedley-Whyte and Laver's equations. 4 Temp = temperature; Hct = hematocrit; Fio₂ = fractional inspired oxygen concentration; pH =-log of hydrogen ion concentration; pCO₂ = arterial or mixed venous partial pressure of carbon dioxide; pO₂ = arterial or mixed venous partial pressure of oxygen; SaO₂ = percent oxygen saturation of arterial blood; STO₂ = percent oxygen saturation of mixed venous blood; CaO₂ = oxygen content of arterial blood; CO₂ = oxygen content of mixed venous blood; CAVO₂ = oxygen content difference between arterial and mixed venous blood; CAVO₂ = arterial or mixed venous oxygen content bound to hemoglobin; VO₂ = arterial or mixed venous oxygen content dissolved in plasma; DO₂ = oxygen delivery; VO₂ = oxygen consumption; ER = oxygen extraction ratio (VO₂/DO₂); and CO = cardiac output.
DISCUSSION

Since we were restricted from transfusing red blood cells in this patient, we employed several therapies designed to optimize the relationship between oxygen delivery and oxygen consumption.

_Hypothermia._ Hypothermia causes a significant reduction in oxygen consumption. Michenfelder et al. found oxygen consumption to be 48% below control values at 30°C, as well as an approximately 6% reduction of oxygen consumption for every 1°C decrease in temperature. The oxy-hemoglobin dissociation curve shifts leftward during hypothermia, increasing hemoglobin-oxygen affinity. No evidence has been found that this increased hemoglobin affinity impairs oxygen extraction at the tissues. During hypothermia, the affinity of tissues for oxygen may increase to the same degree as hemoglobin’s affinity for oxygen, and, thus, no diffusion imbalance occurs. When our patient’s temperature was at its lowest value (29°C), the calculated base deficit was only 3 mEq/l, indicating adequate oxygen extraction by tissues.

Oxygen solubility in plasma increases as blood temperature decreases. Hedley-Whyte and Laver reported a 10% increase in dissolved oxygen as blood is cooled to 30°C, a 19% increase at 25°C, and a 30% increase at 20°C. At normal levels of hematocrit, the increased plasma O2 solubility is of minor importance, but, at the extremely low levels of hematocrit measured in our patient, dissolved oxygen played an important role in oxygen delivery.

An analysis of the patient’s oxygen delivery and consumption at a hematocrit of 4% and a temperature of 30°C shows that 51% of her oxygen delivery was carried by oxygen dissolved in plasma and 49% by oxygen bound to hemoglobin, the normal predominant supplier. This dissolved oxygen provided for 90% of her oxygen consumption. (Oxygen bound to hemoglobin = 1.84 × [Hb] × SO2, where [Hb] is the hemoglobin concentration in grams per deciliter, and SO2 is the arterial or mixed venous oxygen saturation in percent. Dissolved oxygen = PO2 × ao2, where PO2 is the arterial or mixed venous oxygen tension in mmHg, and ao2 is the Bunsen solubility coefficient of O2 in milliliters of oxygen per deciliter of blood at temperature t.) This differs markedly from the normal circumstance of breathing air at Hct 45% where only 2% of the oxygen delivered by arterial blood consists of dissolved oxygen, which contributes a meager 5% of normal oxygen consumption. The 10% increase of the dissolved plasma oxygen concentration resulting from cooling to 30°C thus provided important additional delivery to our patient, who was living almost exclusively (90%) on dissolved oxygen.

Several characteristic changes can be observed on the electrocardiogram during clinical hypothermia. Below 29°C, sinus bradycardia develops with the “Osborn wave,” followed by T wave inversion, prolongation of the PR and QTc intervals, atrial fibrillation, and ventricular fibrillation as temperature progressively decreases.

Blood viscosity increases with hypothermia. At 30°C, blood viscosity is 23% higher than at 37°C. Peripheral resistance increases during hypothermia decreasing cardiac output. Therefore, hypothermia is best accompanied by hemodilution to avoid increasing viscosity and depressing cardiac output.

In our patient, once profound blood loss was noted, the decision was made to cool the patient. A target core temperature of 30°C was chosen, since, at this temperature, arrhythmias do not usually occur, yet temperature is low enough to significantly increase dissolved oxygen delivery and decrease oxygen consumption.

_Hemodilution._ Hemodilution reduces blood viscosity due to decreased hematocrit, decreased viscosity of the diluent, and a decreased tendency towards rouleaux formation. This decreased viscosity increases microvascular flow.

Cardiac output increases linearly with the reduction of hematocrit during hemodilution. This is predominantly caused by an increased stroke volume, although heart rate can increase. Blood flow to most organs rises in proportion to the increased cardiac output, with flow to the stomach, skin, skeletal muscle, and left ventricular epicardium remaining unchanged. Coronary blood flow increases out of proportion to the increased cardiac output. At hemoglobin levels below 5 g/dl blood flow is reduced to subendocardial heart muscle, and subendocardial ischemia and cardiac failure may ensue. However, in our patient, electrocardiographic signs of ischemia did not occur; stroke volume was maintained at normal levels; and overt pulmonary edema did not develop, as evidenced by an unchanging Pao2 and chest radiograph.

Increased oxygen extraction by tissues remains in reserve during isovolemic hemodilution, usually occurring when the hematocrit is reduced to less than 20% or if oxygen consumption is increased. Accordingly, in our patient, the hematocrit of 4% was associated with a markedly increased oxygen extraction ratio of 53%. Under conditions of maximal exercise, the oxygen extraction ratio can rise above 70%. Thus, despite a hematocrit of 4% in our patient, there was additional reserve for tissue oxygen extraction and a high mixed venous oxygen tension of 40 mmHg (table 1, sample 2). When the hematocrit was raised to 22% by transfusion, the extraction ratio decreased to 15%.

_Sedation._ Induction of general anesthesia reduces ox-
ygen consumption by 15–20% below baseline levels. Narcotic administration decreases total body oxygen consumption 4–9%. Sedation was maintained in our patient with large doses of fentanyl.

Skeletal Muscle Paralysis. Voluntary muscle movement, spontaneous respiration, and shivering all increase oxygen consumption. Shivering increases oxygen consumption by 35–40% during postoperative rewarming. Thus, we induced muscle relaxation until core body temperature was normal in order to prevent an increase in oxygen consumption from shivering.

The baseline oxygen consumption after recovery was 212 ml O₂/min, a value calculated when their body temperature was 37°C while spontaneously breathing air, without sedation or muscle relaxation. Her lowest measured oxygen consumption was 92 ml O₂/min at a temperature of 29°C, a reduction of 57% below her baseline level.

In summary, the lack of any identifiable organ damage, despite the dramatically low 4% hematocrit, demonstrates that the combination of induced hypothermia, isovolemic hemodilution, skeletal muscle paralysis, and sedation can provide a successful strategy for safely waiting over 4 h to treat a patient with unanticipated intraoperative hemorrhage until red blood cells could be transfused.

REFERENCES