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-spastic 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 130 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 hypervolemia 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.

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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

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drainage of an abdominal abscess. During the preoperative visit, the patient stated that, due to her religious beliefs, she would not consent to transfusion of any blood products "under any circumstances." Although her preoperative hematocrit was 30%, the prothrombin time, partial thromboplastin time, and bleeding time were normal. She was premedicated with im morphine and oral diazepam.

Thiopental and succinylcholine were given iv to induce anesthesia and facilitate endotracheal intubation. Routine monitoring was employed. Anesthesia was maintained with isoflurane (0.75–1.0% inspired) and nitrous oxide 66% in oxygen, as well as supplemental iv morphine. Skeletal muscle paralysis was obtained with pancuronium and d-tubocurarine iv.

The surgery proceeded uneventfully until the intraabdominal abscess cavity was opened, whereupon profuse bleeding ensued. Over the next hour, approximately 3000 ml of blood was lost. The hematocrit midway through this period was 17%. The patient was cooled by infusing room temperature intravenous fluids and by uncovering the patient to increase heat loss by convection.

Fentanyl 2.6 mg iv was given and isoflurane and nitrous oxide were discontinued, raising the fractional inspired oxygen concentration to 1.0. Oxygen saturation by pulse oximetry was 99–100%. The systolic blood pressure remained between 90 and 110 mmHg throughout the operation. Intraoperative fluid infusion totalled 10 l lactated Ringer's solution and 1.5 l of 6% hydroxyethyl starch. The estimated blood loss was 3.2 l and the urine output was 1.3 l in 3.5 h.

The patient was transferred to the recovery room where skeletal muscle paralysis was maintained with iv pancuronium, and sedation with iv morphine. As the patient was believed to be intravascularly volume depleted, further infusions of lactated Ringer's solution and 6% hydroxyethyl starch were begun.

Pulmonary and radial arterial catheters were placed, revealing: pulmonary arterial pressure 27/20 mmHg, pulmonary capillary wedge pressure 20 mmHg, central venous pressure 18 mmHg, arterial blood pressure 130/70 mmHg, mean arterial blood pressure 90 mmHg, cardiac output 6.5 $l \cdot min^{-1}$, heart rate 88 beats $\cdot min^{-1}$, stroke volume 74 ml, and systemic vascular resistance 890 dyne $\cdot sec^{-1} \cdot cm^{-5}$. The arterial and mixed venous blood gas tensions are shown in table 1, sample 1.

With the permission of the patient's family, an order from a Massachusetts Superior Court Judge was obtained and transfusion of blood was initiated. Immediately before transfusion, laboratory studies were obtained and the values are listed in table 1, sample 2. Blood samples were obtained for analysis after 6 units of packed red blood cells, 4 units of fresh frozen plasma, and 10 units of platelet concentrate were transfused (table 1, sample 3). An additional three units of packed red blood cells were subsequently transfused (table 1, sample 4).

The patient was externally warmed with a warming blanket to a temperature of 37° C, paralysis dissipated, her trachea was extubated, and repeat blood gases were drawn (table 1, sample 5). Serial creatine phosphokinase isoenzymes were within normal limits without elevation of the MB band, and postoperative electrocardiograms were unchanged from preoperatively. Neurological examination, blood urea nitrogen, creatinine, serum glutamic-oxaloacetic transaminase, and alkaline phosphatase remained unchanged from baseline, and the patient felt well.

Total perioperative fluid intake over the first 24 h was 27 l, consisting of 19.3 l lactated Ringer's solution, 2.5 l 6% hydroxyethyl starch, 2.25 l of packed red blood cells, 0.75 l of fresh frozen plasma, and 10 units of platelets. Urine output was 8.2 l and the estimated blood loss was 3.2 l, for a total output of 11.4 l and a net positive fluid balance of 16.6 l. As the urine output remained brisk throughout, diuretics were not given.

The patient was discharged from the intensive care unit after 24 h, and her postoperative course was uneventful.

TABLE 1. Arterial and Mixed Venous Blood Gas Data

Type of Sample	Sample Number	Temp °C	Hct %	F_{IO_2}	pH	P_{CO_2} mmHg	P_{O_2} mmHg	S_{aO_2} $S\bar{v}O_2$ %	Base Deficit mEq/l	C_{aO_2} $C\bar{v}O_2$ ml O_2/100 ml	$C_{cap}O_2$ ml O_2/100 ml	$C_{dis}O_2$ ml O_2/100 ml	$C_{(a-\bar{v})}O_2$ ml O_2/100 ml	$\dot{D}O_2$ ml O_2/min	$\dot{V}O_2$ ml O_2/min	ER %	CO l/min
Arterial	1	32	5	1.0	7.48	28	580	99.9	3	4.2	2.2	2.0		272			
Mixed venous					7.40	37	40	90.1		2.1	1.9	0.1	2.1		138	50	6.5
Arterial	2	30	4	1.0	7.53	27	555	99.9	1	3.7	1.8	1.9		197			
Mixed venous					7.40	37	40	89.8		1.7	1.6	0.1	2.0		105	53	5.3
Arterial	3	29	22	1.0	7.30	47	477	99.9	3	11.5	9.8	1.7		587			
Mixed venous					7.27	56	51	96.4		9.7	9.5	0.2	1.8		92	15	5.1
Arterial	4	33	24	.4	7.43	34	182	99.7	1	11.3	10.7	0.6		542			
Mixed venous					7.38	41	21	49.9		5.4	5.3	0.1	5.9		282	51	4.8
Arterial	5	37	28	.21	7.44	39	98	97.3	−2	12.5	12.2	0.3		623			
Mixed venous					7.38	47	33	65.0		8.2	8.1	0.1	4.3		212	33	5.0

Oxygen saturations calculated with Severinghaus's equations, correcting for changes in temperature and pH.[2] Dissolved oxygen contents calculated with Hedley-Whyte and Laver's equations.[7] Temp = temperature; Hct = hematocrit; FIO_2 = fractional inspired oxygen concentration; pH = −log of hydrogen ion concentration; P_{CO_2} = arterial or mixed venous partial pressure of carbon dioxide; P_{O_2} = arterial or mixed venous partial pressure of oxygen; SaO_2 = percent oxygen saturation of arterial blood; $S\bar{v}O_2$ = percent oxygen saturation of mixed venous blood; CaO_2 = oxygen content of arterial blood; $C\bar{v}O_2$ = oxygen content of mixed venous blood; $C_{(a-\bar{v})}O_2$ = oxygen content difference between arterial and mixed venous blood; $C_{bnd}O_2$ = arterial or mixed venous oxygen content bound to hemoglobin; $C_{dis}O_2$ = arterial or mixed venous oxygen content dissolved in plasma; $\dot{D}O_2$ = oxygen delivery; $\dot{V}O_2$ = oxygen consumption; ER = oxygen extraction ratio ($\dot{V}O_2/\dot{D}O_2$); and CO = cardiac output.

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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.[3,4] 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.[5]

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.[6] 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[7] 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 O_2 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 $= 1.34 \times [Hb] \times SO_2$, where [Hb] is the hemoglobin concentration in grams per deciliter, and SO_2 is the arterial or mixed venous oxygen saturation in percent. Dissolved oxygen $= P_{O_2} \times \alpha_b{}^t$, where P_{O_2} is the arterial or mixed venous oxygen tension in mmHg, and $\alpha_b{}^t$ is the Bunsen solubility coefficient of O_2 in milliliters of oxygen per deciliter of blood at temperature t.[7]) 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 QT_c intervals, atrial fibrillation, and ventricular fibrillation as temperature progressively decreases.[8,9]

Blood viscosity increases with hypothermia. At 30° C, blood viscosity is 23% higher than at 37° C.[10] Peripheral resistance increases during hypothermia decreasing cardiac output.[11] 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, arrythmias 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.[12] This decreased viscosity increases microcirculatory flow.[13]

Cardiac output increases linearly with the reduction of hematocrit during hemodilution.[12,14,15] This is predominantly caused by an increased stroke volume, although heart rate can increase.[13,16] 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.[9,14] Coronary blood flow increases out of proportion to the increased cardiac output.[14] At hemoglobin levels below 5 g/dl blood flow is reduced to subendocardial heart muscle, and subendocardial ischemia and cardiac failure may ensue.[17] 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 Pa_{O_2} 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%,[13,18] 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%.[19] 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-

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ygen consumption by 15–20% below baseline levels.[20] Narcotic administration decreases total body oxygen consumption 4–9%.[21] 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%[22] 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 O_2/min, a value calculated when her body temperature was 37° C while spontaneously breathing air, without sedation or muscle relaxation. Her lowest measured oxygen consumption was 92 ml O_2/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.

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