J Anesth (1996) 10:289–291

Journal of
Anesthesia

Clinical reports

Brain oxygenation monitored by near-infrared spectroscopy during recovery from hemorrhagic shock

MASAYASU ARAI, TOSHIYUKI OKUTOMI, TOMOAKI TAKENAKA, SEIJI KATO, and FUMIO GOTO

Department of Anesthesiology, Kitasato University School of Medicine, 1-15-1 Kitasato, Sagamihara, Kanagawa 228, Japan

Key words: Near-infrared spectroscopy, Brain oxygenation, Hemorrhagic shock, Monitoring

Introduction

Cerebral oxygen saturation monitoring using near-infrared (NIR) spectroscopy has previously been described as being able to estimate regional brain tissue oxygenation and the balance of oxygen supply and demand in the brain [1,2]. Near-infrared light is generated in a forehead sensor and measured at two distances from the light source. The resultant signals are processed to emphasize oxygen saturation in the brain and subtract the effects of extracranial changes. Because it is a noninvasive technique, placement of the sensor on the forehead is simple and straightforward, making the device useful in emergency and critical situations. Since the device does not discriminate between arterial and venous blood, pulsatile blood flow is not a necessary factor in the measurement of hemoglobin (Hb) saturation, and the resultant number reflects predominantly venous saturation.

This case report describes a patient in hemorrhagic shock who was successfully resuscitated and monitored for cerebral oxygen saturation by NIR spectroscopy.

Case report

A 31-year-old woman (weight, 48kg; height, 157cm), ASA physical status IVE, suffering from massive intraabdominal hemorrhage due to ectopic pregnancy, was treated. Before the induction of anesthesia, her systolic arterial pressure was 80mmHg, pulse rate was 155 beats·min^{-1}, and radial pulse was weak. Her hemoglobin (Hb) concentration was 8.4g·dl^{-1} and hematocrit (Hct) was 25.1%. Her respiratory rate was 40·min^{-1} and the Glasgow Coma Scale (GCS) score was 10 points. Her peripheral skin was cold to the touch and her face was pale. Rapid infusion of Ringer's lactated solution was started, and we placed the NIR sensor (INVOS 3100 cerebral oximeter, Somanetics, Troy, MI, USA) on the left side of her forehead to measure regional oxygen saturation (rSo_2). The initial value of rSo_2 was 55% with the patient breathing room air, and her peripheral oxygen saturation (Spo_2) was 98%. The changes in rSo_2, Hb concentration, and Hct over time are shown in Fig. 1.

Anesthesia was induced with thiopental (3mg·kg^{-1}) and succinylcholine (1mg·kg^{-1}). After induction of anesthesia and initiation of fluid volume replacement, rSo_2 gradually increased and, following endotracheal intubation, had risen to 62%. Following rapid infusion of the crystalloid, her systolic arterial pressure gradually improved and we were able to maintain anesthesia with 4l·min^{-1} N_2O, 2l·min^{-1} O_2, and 1% sevoflurane. Arterial blood gas analysis showed metabolic acidosis after induction of anesthesia (pH, 7.22; partial pressure of arterial carbon dioxide ($Paco_2$), 33.8mmHg; partial pressure of arterial oxygen (Pao_2), 197.0mmHg; HCO_3^-, 13.3mEq·l^{-1}; base excess (BE), −13.1mEq·l^{-1}; arterial oxygen saturation (Sao_2), 99.2%). After the infusion of 1000ml of Ringer's lactated solution we administered 500ml of 6% hydroxyethyl starch (HES) solution. There was little intraoperative hemorrhage and massive intraabdominal coagulation was observed. During the infusion of Ringer's lactated solution and HES solution, the patient's systolic blood pressure gradually rose to 120mmHg and her heart rate decreased to 118 beats·min^{-1}. However, rSo_2 leveled off at 68%–69% (a 20% improvement over the initial value)

Address correspondence to: M. Arai
Received for publication on December 5, 1995; accepted on May 31, 1996

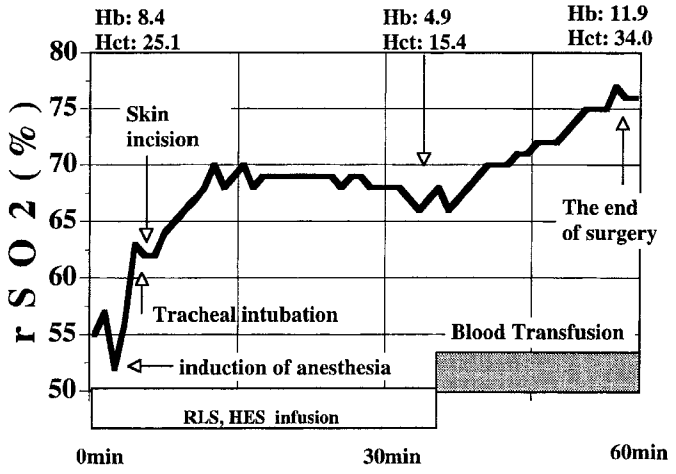

Fig. 1. Changes in regional oxygen saturation (rSo_2; %) over time before and during the blood transfusion. The hematocrit at this point was 15.4%. *Hb*, hemoglobin concentration ($g \cdot dl^{-1}$); *Hct*, hematocrit (%); *RLS*, Ringer's lactated solution; *HES*, hydroxyethyl starch solution

and before the start of the blood transfusion, Hb concentration and Hct were $4.9\,g \cdot dl^{-1}$ and 15.4%, respectively. As the blood transfusion began, rSo_2 began to increase again. Following transfusion of 7 units (approximately 840 ml) of packed red blood cells, the Hb concentration and Hct were $11.4\,g \cdot dl^{-1}$ and 34.0%, respectively, and rSo_2 had risen to 76%, a rise of 38% from the preinduction baseline. The systolic blood pressure increased to 140–150 mmHg and the heart rate decreased to 85 beats·min^{-1}. The patient was fully conscious 10 min after the inhalation anesthesia was discontinued. Blood gas analysis from samples taken in the recovery room showed metabolic acidosis (pH, 7.27; $Paco_2$, 29.5 mmHg; Pao_2, 251.9 mmHg; HCO_3^-, $13.2\,mEq \cdot l^{-1}$; BE, $-12.4\,mEq \cdot l^{-1}$; Sao_2, 99.6%) but there were no other complications. Spo_2 was over 98% throughout the operation.

Discussion

In the present patient, hypoperfusion of the brain due to hemorrhagic shock was suspected based on the GCS score upon arrival at the operating room, and the initial value of rSo_2 at induction of anesthesia was 55%. McCormick et al. [2] noted that the range of normal rSo_2 results in their six healthy patients was 59%–69% (mean ± SD; 63.86 ± 3.43%) with the patients breathing room air. Because there is no "gold standard" for the measurement of regional cerebral oxygen saturation, we made decisions based on the changes in saturation rather than the absolute values. However, from the patients' clinical symptoms and our experience of measuring rSo_2 using the INVOS device in surgical patients, we believe that the initial value of rSo_2 55% in this patient was abnormal.

Impairment of cerebral oxygenation in this case may have been induced by: (a) a decrease of arterial oxygen content (Cao_2) due to anemia, and (b) a decrease of cerebral blood flow (CBF) due to hypovolemia. These factors led to a mismatch between oxygen supply and demand in the brain, resulting in a lower rSo_2 value.

Rapid infusion of Ringer's lactated solution and induction of anesthesia improved rSo_2 by 20%. The infusion exacerbated the patient's anemia and decreased her Cao_2; on the other hand, it increased the circulating blood volume and CBF, which may have resulted in improved oxygen delivery to the brain. Moreover, general anesthesia suppressed the cerebral metabolic ratio of oxygen ($CMRo_2$), which also improved the balance of oxygen demand and supply in the brain.

Next, we observed a plateau in the progress of rSo_2. At this plateau, hemodilution and restoration of blood volume were progressing at the same time. Further restoration of blood volume by infusion of Ringer's lactated and HES solution did not improve rSo_2. Hb concentration and Hct measured immediately before the start of blood transfusion were $4.9\,g \cdot dl^{-1}$ and 15.4%. Following the start of blood transfusion, an additional improvement in rSo_2 was observed. In this case, CBF, $CMRo_2$, and jugular venous saturation were not measured. Sevoflurane, depth of anesthesia, and surgical stress may affect these factors. Moreover, it is uncertain if the patient was euvolemic or hypovolemic because of the urgency of the situation and the limited testing performed. Clinically, the increase in blood pressure and decreased heart rate suggested that the circulating blood volume had recovered to near normovolemia. Other factors which could affect brain circulation such as artificial ventilation, $Paco_2$, sevoflurane concentration, and surgical stress were estimated to be nearly constant. The patient's bladder temperature fell only 0.5°C from the initial value so that the decrease in $CMRo_2$ due to temperature change is considered to have had little effect on the rSo_2 value. Therefore, the increase in rSo_2 was considered to be the result of blood transfusion which induced an increase in arterial oxygen content, restoration of blood volume, and CBF.

In comparison with reports concerning the limit of hemodilution in the systemic circulation [3–5], there have been few reports on the brain, especially regarding clinical practice. In the systemic circulation, the limit of hemodilution was reported as Hct 15% [3], Hb 3.9 ± $0.7\,g \cdot dl^{-1}$ [4], and Hb $4.0\,g \cdot dl^{-1}$ [5] in animals, being $4.0\,g \cdot dl^{-1}$ [6] at the critical point of oxygen delivery

($\dot{D}o_2$) in a human case report. Fan et al. [7] observed that oxygen delivery to the brain was decreased as Hct went below 30% but was maintained 50% above the baseline even if Hct reached 12%. Matsuda [8] reported that Hct should be maintained above 15% during hemodilution to avoid brain tissue ischemia during normothermia. It is suspected from these previous reports that the lowest value of hematocrit in our case was near the limit of hemodilution; however, we were unable to estimate brain function at that particular point in time, making it difficult to determine the limit of hemodilution by the rSo_2 value. The infusion of Ringer's lactated solution and HES caused an improvement in rSo_2; however, we consider that an effective increase in rSo_2 can be achieved not only by Ringer's lactated solution and HES, but also by blood transfusion when blood is hemodiluted to the value obtained in this case.

The estimation of brain oxygenation by INVOS entails certain problems. It is not clearly understood whether the value measured by INVOS completely excludes the changes in extracranial tissue and whether rSo_2 includes only the changes in the regional brain [9]. Germon et al. [10] reported that rSo_2 measured by INVOS was affected significantly by changes in extracranial blood flow and oxygenation. Because the cerebral hypoperfusion that occurred in this case was caused by systemic hypoperfusion, extracranial tissue hypoperfusion must have occurred in the same situation. If INVOS cannot exclude the effect of extracranial tissue changes, we cannot discriminate between alterations in the brain and outside the cranium. The degree to which NIR light penetrates the extracranial tissue is also an important issue in this monitoring system. Further study is necessary to interpret the absolute value of rSo_2.

References

1. McCormick PW, Stewart M, Goetting MG, Dujovny M, Lewis G, Ausman JI (1991) Noninvasive cerebral optical spectroscopy for monitoring cerebral oxygen delivery and hemodynamics. Crit Care Med 19:89–97
2. McCormick PW, Stewart M, Goetting MG, Balakrishnan G (1991) Regional cerebrovascular oxygen saturation measured by optical spectroscopy in humans. Stroke 22:596–602
3. Takaori M, Safer P (1976) Critical point in progressive hemodilution with hydoxy-ethyl starch. Kawasaki Med J 2(4): 211–222
4. Räsänen J (1991) Supply dependent oxygen consumption and mixed venous oxyhemoglobin saturation during isovolemic hemodilution in pigs. Anesthesiology 75:A260
5. Trouwborst A, Tenbrick R, Van Woerkens ECSM (1990) Blood gas analysis of mixed venous blood during normoxic acute isovolemic hemodilution in pigs. Anesth Analg 70:523–529
6. Van Woerkens ECSM, Trouwborst A, Van Lanschot JJB (1992) Profound hemodilution: What is the critical level of hemodilution at which oxygen delivery-dependent oxygen consumption starts in an anesthetized human?. Anesth Analg 75:818–821
7. Fan FC, Chen RYZ, Schussler GB, Chien S (1980) Effects of hematocrit variation on regional hemodynamics and oxygen transport in the dog. Am J Physiol 238 (Heart Circ Physiol 7):H545–H552
8. Matsuda K (1993) Noninvasive brain tissue oxygenation in euvolemic hemodilution with near-infrared spectroscopy (in Japanese with English abstract). Circ Contr 14(4):521–529
9. Harris DNF (1995) Near infra-red spectroscopy. Anaesthesia 50:1015–1016
10. Germon TJ, Kane NM, Manara AR, Nelson RJ (1994) Near-infrared spectroscopy in adults: effects of extracranial ischemia and intracranial hypoxia on estimation of cerebral oxygenation. Br J Anaesth 73:503–506