Neuroanesthesia and Intensive Care

Intraoperative jugular bulb desaturation during acute aneurysmal rupture

[Désaturation peropératoire du golfe de la jugulaire pendant la rupture aiguë d'un anévrysme]

Irene Rozet MD,* David W. Newell MD,† Arthur M. Lam MD FRCPC‡

Purpose: To describe an episode of acute jugular venous desaturation during intraoperative rupture of a cerebral aneurysm.

Clinical features: A 57-yr-old patient was scheduled for clipping of a large unruptured basilar tip aneurysm. Abrupt bulging of the brain was observed after bone flap removal, but before dura was opened. This was associated with concurrent development of systemic hypertension to 200/120 mmHg and jugular venous bulb ($S_{jv}O_2$) desaturation to 13%. Rupture of aneurysm was confirmed by frank blood in cerebrospinal fluid drainage from the lumbar subarachnoid catheter.

Conclusions: Abrupt $S_{jv}O_2$ desaturation prior to dural opening may suggest an acute increase in intracranial pressure, which in our case followed aneurysmal rupture; the systemic response to increased intracranial pressure (Cushing's response) may be ineffective in maintaining cerebral perfusion.

***Objectif** : Décrire un épisode de désaturation aiguë de la veine jugulaire pendant la rupture peropératoire d'un anévrysme cérébral.*

***Éléments cliniques** : Un patient de 57 ans devait subir la ligature d'un important anévrysme non rupturé de la pointe basilaire. Un bombement soudain du cerveau a été observé après le retrait du volet osseux mais avant l'ouverture de la dure-mère. Une hypertension générale à 200/120 mmHg s'est développée concurremment ainsi qu'une désaturation à 13 % du golfe de la jugulaire ($S_{jv}O_2$). La rupture de l'anévrysme a été confirmée par la présence franche de sang dans le liquide céphalo-rachidien décelée par le cathéter lombaire sous-arachnoïdien.*

***Conclusion** : La désaturation soudaine de la $S_{jv}O_2$ avant l'ouverture de la dure-mère peut évoquer une hausse soudaine de la pression intracrânienne, faisant suite à une rupture anévrysmale ; la réponse généralisée à l'hypertension intracrânienne (réaction de Cushing) peut être inefficace à maintenir la perfusion cérébrale.*

MONITORING of jugular venous bulb saturation ($S_{jv}O_2$) has been shown to be helpful in neurosurgical anesthesia.[1] In this report, we present a case of acute jugular bulb desaturation associated with intraoperative aneurysmal rupture. Rupture of aneurysm occurred before dura was opened, and this was associated with the development of systemic hypertension and temporary profound $S_{jv}O_2$ desaturation. This was accompanied with the classic Cushing response. The physiological aspects of Cushing response and the clinical applications of intraoperative continuous $S_{jv}O_2$ monitoring are discussed.

Case report

According to institutional guidelines, case reports do not require approval from Institutional Review Board.

A 56-yr-old male with a past medical history of stable coronary artery disease, hypertension, and heavy smoking, was transferred to our hospital for investigation of subarachnoid hemorrhage. Computed tomography of head was negative for subarachnoid hemorrhage, but angiography revealed a large aneu-

From the Departments of Anesthesiology,*‡ Neurosurgical Intensive Care Unit,*‡ and Neurological Surgery,†‡ University of Washington, Seattle, Washington, USA.

Address correspondence to: Dr. Irene Rozet, Assistant Professor, Harborview Medical Center, Box 359724, 325 Ninth Avenue, Seattle, Washington 98104-2499, USA. E-mail: irozet@u.washington.edu

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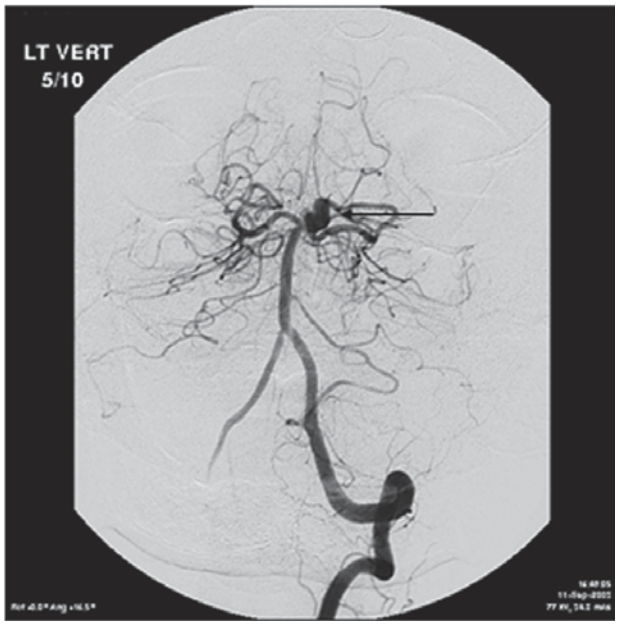

FIGURE 1 Left vertebral artery angiography. Arrow shows a large multilobular basilar tip aneurysm, 1 × 2 cm.

FIGURE 2 Left lateral neck and head *x-ray*. The arrow points to the tip of the retrograde jugular catheter, positioned in the jugular bulb at the level of the lower border of the mastoid process and C1 vertebra.

rysm of basilar artery (Figure 1). After unsuccessful attempts to coil the aneurysm, the patient was scheduled for surgical clipping on day 16 post-admission. Preoperatively, the patient was alert and awake with a Glasgow coma scale score of 15. After uneventful induction of anesthesia with fentanyl, propofol and rocuronium, the trachea was intubated and the patient was ventilated with a mixture of oxygen/air, and anesthesia was maintained with sevoflurane (1.5–2% end-tidal), remifentanil (infusion at 0.125 $\mu g \cdot kg^{-1} \cdot hr^{-1}$), and vecuronium as needed. In addition to routine monitoring, a 4-Fr oximetric catheter (model: 040HF4, Edwards Lifesciences, LLC, Irvine, CA, USA) was inserted into the right jugular bulb,[1] and calibrated *in vivo* for continuous $S_{jv}O_2$ measurement (Vigilance Monitor, Edwards SAT-2™ computer, Irvine, CA; USA oximetry range of 0–99%). Positioning of the catheter tip was verified radiologically (Figure 2). For induction of moderate hypothermia an intravascular cooling device (Innercool Therapies, San Diego, CA, USA) was inserted into the inferior vena cava via the left femoral vein.

During cranium opening, vital signs were stable: $PaCO_2$ 33 to 36 mmHg, arterial pressure (AP) 110/60 to 130/70 mmHg, heart rate (HR) 70 beats·min^{-1}, $S_{jv}O_2$ 50 to 55%. The patient was cooled to 33.0°C. Mannitol (1g·kg^{-1}) was given. After the bone flap was raised, the brain initially appeared to be slack. Immediately before opening of dura, however, the dura suddenly became tense, and the brain started bulging out, making dural incision impossible. Arterial pressure abruptly increased to 200–210/120 mmHg, HR decreased to 46 beats·min^{-1}, and $S_{jv}O_2$ precipitously decreased to 13% (Figure 3). The lumbar subarachnoid catheter was opened, which returned bright red blood, confirming acute rupture of the aneurysm, and then was clamped. The operation was halted, and the patient was treated with hyperventilation, *iv* boluses of sodium thiopental (2 g *iv* total), labetalol (100 mg *iv*), and mannitol (2 g·kg^{-1} *iv*). After approximately five minutes, $S_{jv}O_2$ started to increase, AP decreased to 150/70 mmHg, and HR increased to 70 beats·min^{-1}. There was visible improvement in dural tension. At constant end-tidal CO_2, $S_{jv}O_2$ increased progressively to 84% and remained elevated for ten minutes (Figure 3), followed by a gradual decline to 55%. The jugular venous bulb lactate was 1.8 mmol·L^{-1}, compared to 1.0 mmol·L^{-1} in arterial blood. After 30 min, the brain appeared to be slack again, and with the patient hemodynamically stable, the surgical procedure was allowed to resume. The lumbar subarachnoid catheter was opened and drained

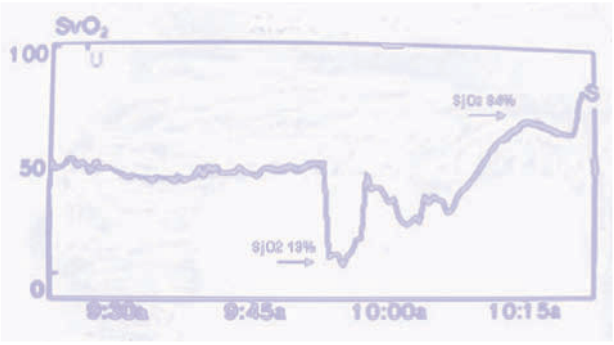

FIGURE 3 Continuous $S_{jv}O_2$ monitoring, recorded during the event. With the rupture of the basilar tip aneurysm, $S_{jv}O_2$ abruptly decreased from 53% to 13% for about five minutes, indicating nearly "no flow state" in the brain. Following the treatment with sodium thiopental and mannitol, $S_{jv}O_2$ started to increase, and stayed at the high level of 84%, indicating luxury perfusion state.

100 mL of bloody cerebrospinal fluid. Under microscopic dissection, the aneurysm was identified and found to be leaking. Temporary cardiac arrest lasting up to 25 sec was induced with escalating boluses of adenosine up to 24 mg *iv*, and the aneurysm was clipped successfully. Transient decrease of $S_{jv}O_2$ lasting five to ten seconds occurred with the two high doses of adenosine.

On postoperative day one (POD 1), the patient awoke, opened his eyes, but was aphasic, and had right hemiplegia. On POD 5, the patient became unresponsive to painful stimuli (Glascow coma scale score of 3). Angiography confirmed severe vasospasm of left middle cerebral artery, which was treated with angioplasty, and triple-H therapy (hemodilution to a hematocrit of 30, hypervolemia to central venous pressure of 16 mmHg, and induced hypertension of systolic AP of 180 mmHg). This was complicated by pulmonary edema and rapid atrial fibrillation, but without cardiac enzymes changes. Following brief improvement, the patient's neurologic status again deteriorated. Magnetic resonance imaging of the brain revealed left temporal lobe edema and infarction of left midbrain. Subsequent intensive care unit stay was complicated by the development of ventilator-associated pneumonia and adult respiratory distress syndrome, meningitis, sepsis, and multi-organ failure. On POD 62, the patient expired.

Discussion

Intraoperative aneurysm rupture is not an infrequent occurrence, but this usually occurs during dissection and/or clipping.[2] Diagnosis of rupture before dural opening may be obvious or difficult, and the present case illustrates the physiological changes associated with this complication. Although the cranium is open, because the dura remains closed, rupture of the aneurysm with subsequent bleeding still leads to an abrupt increase in intracranial pressure (ICP) with systemic changes indicative of the classic Cushing response. The associated $S_{jv}O_2$ change provided insight into this response, and facilitated the perioperative management.

The use of $S_{jv}O_2$ monitoring has been described in neuroanesthesia[3] and can be used to adjust mechanical ventilation and blood pressure to maintain adequate oxygen delivery to the brain. In our institution, continuous $S_{jv}O_2$ monitoring is a part of routine practice during clipping of basilar tip aneurysms, when transient cardiac arrest with adenosine is planned. In our patient, $S_{jv}O_2$ decreased abruptly from 52% to 13%, accompanied by an increase in lactate, strongly suggesting a nearly "no flow" state, confirming the diagnosis of aneurysmal rupture. Although the accuracy of oximetry at such low values has not been carefully studied (accuracy range at $SatO_2$ = 30 to 99% is ± 2%, according to manufacturer manual), this is the lowest $S_{jv}O_2$ value ever reported. This was preceded by systemic hypertension, but the time lag was short, and within a minute. This is consistent with low cerebral oxygen reserve with a highly metabolically active organ requiring a high blood flow. Systemic hypertension, however, is a nonspecific sign during surgery and not diagnostic for aneurysmal rupture.

After treatment with sodium thiopental and mannitol, dural tension decreased, and $S_{jv}O_2$ gradually increased; both are indicative of decrease in brain compression and restoration of cerebral blood flow. Subsequent increase in $S_{jv}O_2$ to 84% suggests reactive hyperemia post-brain injury, or "luxury" perfusion. Had the hyperemia persisted, it would suggest that the brain might have suffered irreparable damage, and was no longer able to extract oxygen. A joint neurosurgery-anesthesia decision had been made to abandon the procedure, if the $S_{jv}O_2$ did not normalize within the 30 min period.

With regard to systemic blood pressure changes, $S_{jv}O_2$ monitoring provides some interesting insights worthy of discussion. The classical Cushing response consists of systemic hypertension and bradycardia secondary to increase in ICP.[4] According to the classic hypothesis, increase of systemic BP during intracranial hypertension represents a compensatory mechanism intended to maintain cerebral perfusion pressure (CPP).[5] Different mechanisms of BP elevation secondary to increased ICP had been suggested in animal studies. Systemic hyper-

tension has been described to be a result of the reflex increase of catecholamine secretion,[6–8] and a result of the sympathetic overactivity due to brainstem compression,[9] or due to craniocaudal ischemia.[10–13] Some animal studies suggest Cushing's response is a late feature, when ICP approaches mean arterial pressure, signalling breakdown of cerebral autoregulation.[14-17] In this situation, hypertension leads to further increase of ICP, adversely influencing cerebral perfusion.[14-17] It is likely that both the level of maximal ICP, and the rate of ICP change influence the hemodynamic response. Animal studies showed "classic" Cushing's response to be apparent and beneficial to CPP only with gradual increases in ICP, with maximal ICP staying within the limits of cerebral autoregulation.[13,18] In our case, the sudden occurrence of tense dura and appearance of the bright blood from the lumbar subarachnoid catheter confirmed the diagnosis of aneurysmal rupture. In centres where subarachnoid catheters are not placed routinely, the sudden and severe $S_{jv}O_2$ desaturation may facilitate the diagnosis of aneurysmal rupture. Although the cranium was open, the acute profound fall of $S_{jv}O_2$ indicated a rapid extreme cerebral hypoperfusion secondary to increased "intraparenchymal pressure". This is the first time jugular desaturation has been recorded during acute aneurysmal rupture accompanied by the Cushing response. Although the increase in systemic blood pressure represents a compensatory mechanism to maintain CPP, the severe jugular venous desaturation in our case suggests that 1) there was cortical cerebral ischemia, 2) the Cushing response may be an ineffective compensatory mechanism.

Although not essential in routine clinical practice, the presence of $S_{jv}O_2$ monitoring in our case facilitated the perioperative management of unexpected aneurysmal rupture, and provided some insight regarding the mechanism and efficacy of the Cushing response.

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