Abstract: Objective: To describe a case of reversible visual loss after a neurosurgical intervention and to discuss the role of the prone position as a potential risk factor.

Observation: A 63-year-old woman without significant medical previous history underwent elective resection of a left parieto-occipital meningioma. Preoperatively, the patient presented a right homonymous lower quadrantanopsia. The surgical procedure was not complicated. The patient was positioned in prone with a mild inclination of the table in reverse Trendelenburg position. The head was maintained in a Mayfield skull clamp, and ocular compression was excluded. There was no significant hypotension, hemodilution or vasopressors infusion during the procedure. Immediately after recovery from anesthesia, the patient experienced total blindness and flash visual evoked potentials confirmed the absence of retinal, primary or late occipital activities. A progressive, but finally complete recovery started after 24 hours.

Conclusion: This case illustrates the individual risk for visual injury after the prone position during some neurosurgical interventions.

Key words: Perioperative visual loss; neurosurgery; prone position; evoked potentials; magnetic resonance imaging.

INTRODUCTION

Visual loss is a rare but severe complication that can occur following anesthesia and non-ophthalmological surgery (3, 4). It has been suggested that spine and heart surgery may be associated with a higher incidence of perioperative visual loss (POVL), but other surgical procedures are also concerned (3, 4). Prone position may be necessary during some neurosurgical interventions, with the head fixed in a skull clamp. We here present the case of a patient with fully reversible POVL following elective uncomplicated resection of a left parieto-occipital meningioma.

CASE REPORT

An elective resection of a cerebral meningioma was planned in a 63-year-old woman (152 cm, 45 kg). She had no significant past medical history. In particular, she had no history of chronically
During surgery, the systolic blood pressure was always kept between 90 and 130 mmHg, and the diastolic blood pressure between 60 and 80 mmHg. Fluid replacement and loading was used to maintain normal blood pressures. It induced mild hemodilution. During the procedure, the plethysmography variability index (PVI), and pulse pressure variation (delta PP) remained between 16 and 18%. Total fluid infusion amounted 4000 ml of crystalloids, 1000 ml of colloids, and 90 ml of 20% mannitol. A continuous infusion of norepinephrine at the low rate of 0.1 mg/h was also used to maintain blood pressure. No significant blood losses were noted during surgery (<500 ml). The total intraoperative diuresis was 1140 ml. At the end of the procedure, hemoglobin concentration was 9.0 g/dL, and hematocrit was 28%. Arterial blood gas analysis was performed every 2 hours and arterial PaCO2 was maintained between 32 and 33 mmHg. The surgical procedure itself was not complicated and a complete resection of the meningioma was possible. The patient was elevated arterial blood pressure, or of cerebrovascular disease. A few years before, she had undergone surgery under general anesthesia for tubal ligation, without any complication. The meningioma (46 × 40 × 37 mm) was located in the left internal parieto-occipital territory (Fig. 1). Preoperative ophthalmological examination revealed a right homonymous lower quadrantanopia. Fundoscopy was normal. General anesthesia was induced using a propofol target-controlled infusion (TCI) and sufentanil. The propofol TCI was also used for maintenance of anesthesia. The patient was positioned prone for surgery, with a mild inclination of the table towards a reverse Trendelenburg position (Fig. 2). The head was maintained in a Mayfield skull clamp, with a mild flexion and right rotation. There was no possibility for external compression on the eyes during the whole procedure.

The preoperative arterial blood pressure was 130/80 mmHg, and hemoglobin concentration was 13.6 g/dL. The total procedure duration was 8 hours and 30 minutes. The prone position lasted for 7 hours and a half. During surgery, the systolic blood pressure was always kept between 90 and 130 mmHg, and the diastolic blood pressure between 60 and 80 mmHg. Fluid replacement and loading was used to maintain normal blood pressures. It induced mild hemodilution. During the procedure, the plethysmography variability index (PVI), and pulse pressure variation (delta PP) remained between 16 and 18%. Total fluid infusion amounted 4000 ml of crystalloids, 1000 ml of colloids, and 90 ml of 20% mannitol. A continuous infusion of norepinephrine at the low rate of 0.1 mg/h was also used to maintain blood pressure. No significant blood losses were noted during surgery (<500 ml). The total intraoperative diuresis was 1140 ml. At the end of the procedure, hemoglobin concentration was 9.0 g/dL, and hematocrit was 28%. Arterial blood gas analysis was performed every 2 hours and arterial PaCO2 was maintained between 32 and 33 mmHg. The surgical procedure itself was not complicated and a complete resection of the meningioma was possible. The patient was
then transferred to the Intensive Care Unit (ICU). After recovery from anesthesia and extubation of the trachea, the patient immediately complained of total blindness. Neurological examination was otherwise normal, except for a mild spatiotemporal disorientation. The examination of the eyes was also normal, without evidence of chemosis. The pupils were not reactive to light stimulation. The intracocular pressure was in a normal range. Fundoscopic examination was also normal, without any evidence of papilledema or retinal changes. Laboratory investigations showed a hemoglobin level at 8.2 g/dL, and hematocrit at 26%. Brain computed tomography (CT) excluded any surgical complication. Likewise, brain magnetic resonance imaging (MRI) failed to reveal any recent ischemic lesion or compression on the visual pathways. Flash visual evoked potentials (fVEPs) were performed and showed no retinal, primary or late occipital activities (Fig. 3a). Of note, full field VEPs recording could not be performed at the acute stage, in so far as the patient was confined in the ICU. A progressive recovery of visual acuity was noted after 24 hours. On the third postoperative day, the patient had fully recovered her pre-existing visual status, including the pre-existing quadrantanopsia. At 6 months follow-up, electrophysiological examination was also normalized, both on flash (Fig. 3b) and full field VEPs (data not shown).

Fig. 3. — Flash visual evoked potentials (VEPs) showed postoperatively (a) no reproducible waveform, indicating the absence of retinal, primary and late occipital activities. At follow-up (b), VEPs demonstrated full recovery of retinal (I), primary (III) and late occipital activities.
Differential diagnosis of perioperative visual loss (POVL). Legend: CRAO, central retinal artery compression; AION, anterior ischemic optic neuropathy; PION, posterior ischemic optic neuropathy; CB, cortical blindness; PRES, posterior reversible encephalopathy syndrome; IVEPs, flash visual evoked potentials; MRI, magnetic resonance imaging; CT, computed tomodensitometry; FLAIR, fluid attenuated inversion recovery; DWI, diffusion-weighted imaging

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**DISCUSSION**

Perioperative visual loss (POVL) is a rare but devastating complication after non ophthalmological surgery (3, 4). The true incidence is not precisely known. According to a recent analysis, the highest frequencies were observed after spine (0.03%) and heart (0.086%) surgery (5).

The three different causes of POVL must be considered after spine and heart surgery. They include central retinal artery occlusion (CRAO), ischemic optic neuropathy (ION), and cortical blindness (CB) (5). Theoretically, the differential diagnosis should also include the posterior reversible encephalopathy syndrome (PRES), that has been exceptionally reported after some neurosurgical procedures (2) (Table 1). PRES is characterized by headache, confusion, visual loss, and posterior edema of the brain that can be evidenced by an MRI. It may occur in the absence of elevated systemic blood pressure.

Retinal vascular occlusion is mainly the result of external compression, with ischemia-reperfusion injury of retinal cells. The prognosis is usually poor, and the treatment generally non efficient. Regarding ischemic optic neuropathy (ION), the specific mechanism and location of the vascular insult remain largely unknown. Visual loss typically occurs within the first 24-48 h after surgery. Optic disc examination appears normal with posterior ION, while optic disc edema is usually noted in anterior ION (5). Several risk factors are cited in the literature. Among them, prone position and long duration of surgery may predispose patients to ION and visual loss.
Studies in normal volunteers and patients have shown a time-dependent increase in intraocular pressure in the prone position (1). This increase in intraocular pressure is minimally influenced by mild reverse Trendelenburg position, or by intravenous fluid administration. Surgery was longer in patients with postoperative blindness after spine surgery, as compared to unaffected patients. Hypotension may also play a role in case of anatomic variation of the vasculature, or in case of abnormal perfusion pressure-dependent auto-regulation (5). However, hypotension itself is not enough to cause ION. The literature does not provide any clue on the blood pressure threshold below which hypotension may potentially be dangerous. Blood losses and hemodilution are probably two additional risk factors (5). Again, no threshold values can clearly be delineated. ION has also been reported in patients with massive fluid replacement during surgery in the prone position, raising the possibility that positioning contributes to altered venous hemodynamics within the optic nerve. The leading theory regarding the etiology of ION is the development of a compartment syndrome within the optic nerve, due to long duration in the prone position, large fluid shifts, and edema formation. Finally, the role of vasopressors is also questionable.

According to fundoscopic examination and other investigations, the most probable diagnosis in our patient appears to be reversible posterior ION. Indeed, prone position was maintained for a prolonged period of time (7 hours and a half), with moderate neck flexion and rotation. Additional risk factors were abundant fluid replacement, and use of low doses of vasopressors. There was no external compression on the eyes.

This case outlines the importance of some preventive measures for patients undergoing surgery in the prone position:

1. Hemodilution and anemia should be avoided. The hematocrit should always be maintained above 28-30%.
2. High doses of vasopressors should be avoided.
3. The duration of prone position should be reduced to its minimum, while maintaining the head in a neutral forward position.
4. Cephalic venous pressure should be limited using a reverse Trendelenburg position, and avoiding cervical compression.
5. Each patient should be informed of the potential for blindness when surgery is performed in the prone position.

References