Mild visual symptoms, such as photophobia or diplopia, are common after anesthesia. Recently, Dhamee et al. found an incidence of visual disturbances after minor gynecologic procedures of 7–14%. Because these symptoms seem to be both transient and benign in nature, they seldom are investigated or regarded as cause for concern.

In contrast, the complaint of visual loss in the recovery room is unusual and alarming. Recognized causes of severe visual symptoms in the postoperative period include central retinal artery occlusion, acute corneal epithelial edema, and ketamine. Decreased or absent vision also occurs in association with transurethral prostatectomy, where it has been ascribed to the use of atropine, the development of cerebral edema, or the intravascular absorption of glycine.

We report a case of postoperative visual impairment diagnosed as Valsalva hemorrhagic retinopathy.

REPORT OF A CASE

A 31-year-old man was admitted for laparoscopy and tubal insufflation. She had undergone two uncomplicated anesthetics in the remote past. Otherwise, the review of systems and medical history were unremarkable. Laboratory studies and physical examination were normal, with the exception of mild scoliosis.

No premedication was administered. Glycopyrrolate 0.2 mg was given iv, after which anesthesia was induced with methohexital 200 mg iv, in divided doses of 50 mg. Tracheal intubation was facilitated by succinylcholine 100 mg iv and was accomplished without difficulty on the second attempt. Anesthesia was maintained with nitrous oxide 50% and halothane at an inspired concentration of 1–1.5%, with controlled ventilation at a tidal volume of 800 ml and a respiratory rate of 8 breaths/minute. Surgery was performed in the Trendelenburg position without complication. At the end of surgery, frequent premature ventricular contractions occurred, which disappeared promptly with hyperventilation. Tracheal extubation and initial emergence from anesthesia were otherwise uneventful.

Upon awakening in the recovery room, the patient complained of "seeing red" through her left eye. She denied photophobia or ocular pain, nor did she notice any change in vision in her right eye. Fundoscopic examination by a consulting ophthalmologist revealed a single retinal venous hemorrhage that involved the left macula. Visual acuity was 20/20 OD and 20/80 OS. These findings were diagnosed as Valsalva hemorrhagic retinopathy.

No treatment was recommended. Three days later, examination by an ophthalmologist revealed no change. Thirty days later, the patient's vision had improved to 20/20 OD and 20/30 OS. Fluorescein angiography revealed no leakage, and fundoscopic examination was normal, with complete resolution of the previously apparent retinal hemorrhage.

When seen by her ophthalmologist 6 months later, the patient had no complaints. Fundoscopic examination was again normal, and visual acuity was 20/20 OD and 20/20-2 OS.

DISCUSSION

Ophthalmologists have reported the rare and somewhat surprising occurrence of retinal venous hemorrhages in otherwise healthy individuals. In these cases, the hemorrhages were attributed to the hemodynamic changes accompanying protracted vomiting, blowing up air mattresses, or "stormy" emergence from anesthesia. Since these activities are physiologically similar to a Valsalva maneuver, ophthalmologists describe such hemorrhages as Valsalva hemorrhagic retinopathy.

Retinal venous hemorrhages also are observed in a wide variety of disease states, such as cystic fibrosis, chronic obstructive lung disease, migraine headache, and the battered child syndrome. They are found in up to 50% of high-altitude climbers, and in some of these cases, the consequent visual impairment has been permanent. Such hemorrhages were observed in 20% of patients undergoing pneumoencephalography in head-dependent positions, but do not occur in patients undergoing this procedure while entirely supine. Parallel hemodynamics may explain why 20–40% of neonates delivered vaginally have retinal venous hemorrhages, versus a 0–3% incidence in neonates delivered by Cesarean section.

Theories proposed to explain hemorrhagic retinopathy have focused on control of the retinal microcirculation. Retinal vasodilation will take place in response to hypoxia, hypercarbia, or head-dependent positions. If there is obstruction to venous return, as from increased intracranial pressure, retinal venous pressure will rise. Increased intracranial pressure, in turn, also will result...
from hypoxia and hypercarbia, as well as from tracheal stimulation alone.\textsuperscript{24}

Thus, retinal vessels may rupture when the microcirculation is under intolerable pressure due to combinations of the above factors. Alternatively, a vessel so maximally dilated may be vulnerable to the sudden insult of an abrupt pressure wave associated with a Valsalva maneuver.

Multiple factors were probably responsible for the retinal hemorrhage in our patient. The Trendelenburg position could increase intraocular venous pressure. A further increase in retinal venous pressure could have resulted from carbon dioxide insufflation, since this procedure has been shown to raise intraabdominal pressure,\textsuperscript{25} which is potentially transmissible to the retinal circulation. The tracheal stimulation of intubation and extubation could have raised the intracranial pressure, and thereby also elevated retinal venous pressure. Carbon dioxide insufflation also carries a slight risk of hypercarbia with concomitant vasodilation. Finally, after the effects of succinylcholine had subsided, deep halothane anesthesia should lower intraocular pressure,\textsuperscript{26} an effect known to increase the likelihood of intraocular vascular rupture.

Our patient did not have any obvious episodes of coughing, bucking, or laryngospasm, but she certainly could have performed a Valsalva maneuver at some point during her anesthetic. This stress, superimposed on a circulation made susceptible by the preceding conditions, could have precipitated the retinal bleed.

In most cases of symptomatic retinal venous hemorrhage, there will be complete recovery of vision over a matter of days to months.\textsuperscript{27} However, if there is massive hemorrhage or if optic atrophy develops due to bleeding into the optic nerve, a permanent decrease in vision may ensue.\textsuperscript{28} There is no treatment for retinal venous hemorrhages unless they are so massive as to involve the vitreous, in which case vitrectomy eventually might be indicated.

Most cases of retinal hemorrhage are asymptomatic, since patients will notice no visual changes unless the macula is involved. In studies of neonates with retinal hemorrhages, the macula was involved in 1.8–11.6%.\textsuperscript{29} It has been suggested that neonatal macular hemorrhage may cause organic amblyopia and strabismus.\textsuperscript{30} Subsequent studies have tended to refute this hypothesis,\textsuperscript{29,31} but the number of patients followed was probably too small to definitively answer this question.

Retinal bleeding in the absence of prior intraocular disease also may arise primarily from the arterial side of the circulation. When this bleeding occurs subsequent to extraocular trauma, the fundoscopic examination typically also reveals cotton–wool exudates and is described as Purtischer's Retinopathy. This retinal angio-

pathy is thought to develop when a sudden extreme elevation in ocular arterial pressure secondary to abrupt decelerative forces produces reflex arterial spasm and consequent ischemia. Thus, cotton–wool exudates, a sign of ischemia,\textsuperscript{32} appear in cases of arterial rather than venous angiopathy. Either ischemia or extreme pressure changes could injure the microvasculature. Altered permeability with hemorrhage then may ensue.

The prognosis of Purtischer's Retinopathy is poor. In the over 200 reported cases, more than half had permanently impaired vision.\textsuperscript{33} There are European reports describing successful treatment with steroids, nicotinic acid, papaverine,\textsuperscript{34} and cysteine diphosphocholine.\textsuperscript{35} As these reports are anecdotal, these interventions must be considered of unproven efficacy.

Since anesthesia frequently is administered to trauma victims, Purtischer's Retinopathy occasionally should be diagnosed in the immediate postoperative period. Thus, both Purtischer's Retinopathy and retinal venous hemorrhage should be considered in the differential diagnosis of postanesthetic visual loss.

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Respiratory Depression Following Orally Administered Flunitrazepam for Preanesthetic Medication in Children

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Because children fear needles, we advocate preanesthetic sedation by oral or rectal route.1,2 Benzodiazepines can cause respiratory depression3,4 although contradictory data have been published.5,6 These conflicting results probably are due to the varying conditions of investigation, such as dosage and route of administration of different drugs, age of subjects, and measuring methods utilized. In adults the apparatus used for respiratory measurements may itself alter breathing pattern. Face mask, mouth piece, and nose clips produce an increase in tidal volume (Vt) and a decrease in respiratory rate.7,8 Thus, respiratory measurement with standard methods in awake children are likely to be altered by measuring devices even more than in adults.

Oral administration of a long-acting benzodiazepine, flunitrazepam (FNZP), 9,10 30–45 min before induction of anesthesia provides satisfactory premedication in children. The purpose of this study was to evaluate, with a noninvasive method, the respiratory effects of FNZP, administered orally as premedication in children undergoing elective minor ENT surgery.

Patients and Methods

Ten children, aged 7.1 ± 1.5 years (x ± SD), weighing 24.7 ± 7.7 kg, participated in the study, which was

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