per cent in 13,062 cases. In a series of 4,000 blocks compiled by Dawkins over a period of 25 years,[9] the dural puncture occurred in 7.5 per cent in 210 cases in which loss of resistance method for the identification of epidural space was used.

We conclude that this new device for the identification of epidural space is simple, dependable, inexpensive, and readily available in the operating room in a sterile, disposable form. We have been able to achieve accurate location of the epidural space in 500 cases; on the first attempt in 487 cases and on the second attempt in the adjacent lumbar interspinous space in 13 cases. There has been no incident of inadvertent dural puncture. The "end-point" of entry of the needle in the epidural space is unmistakably evident both in the form of movement of the dispersed bubbles and droplets and the oscillations of the column.

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Visual Disturbances: An Unusual Symptom of Transurethral Prostatic Resection Reaction

ANDRANIK OVASSAPIAN, M.D.,* C. W. JOSHI, M.B.B.S.,† EDWARD A. BRUNNER, M.D., PH.D.‡

Visual disturbances such as blurred vision,[1–3] transient blindness,[2–5] and pupillary dilatation[3,6] have been reported with the transurethral resection reaction syndrome. These visual disturbances have been attributed to cerebral edema[2,4] and atropine injection during the operation.[3]

We describe our experience in five patients who developed visual complication during transurethral resection of prostate (TURP) and propose that glycine from the surgical irrigating fluid was the cause.

Report of Five Cases

Clinical data on each case are summarized in table 1. All resections of the prostate were conducted under spinal anesthesia using 1.5 per cent glycine as irrigating fluid.

Patient 1. A 56-year-old man in good health and with normal laboratory findings had a decrease in heart rate of 100/min to 65/min and developed blurred vision 90 min after the beginning of the TURP. Shortly thereafter he complained of visual halos and blue visual hue. Prostatic resection was complicated by both arterial and venus bleeding, with estimated blood loss of 700 ml. In the recovery room he was awake and oriented but complained of inability to see anything, and had a headache, nausea, tingling, and numbness of both upper extremities. His vision was limited to light perception only, and both pupils were dilated with no reaction to light stimuli. Intravenous crystalloid administration was restricted and 20 mg furosemide was administered iv. A neurologist's evaluation at this time revealed no abnormalities except for the visual changes described above. There was no papilledema. Two and a half hours after admission to the recovery room, he still had tingling and numbness of both hands but vision had improved. Eye examination by an ophthalmologist at this time revealed 20/100 vision, intraocular tension of 15 mm, and dilated pupils that were nonreactive to light. Extraocular muscle movement and fundus examination of the eyes were normal. No definite diagnosis could be established by either the neurologist or ophthalmologist. Thirteen hours after completion of surgery, numbness and tingling of upper extremities had stopped. His vision returned to 20/20, 48 hours later.

Patient 2. An 86-year-old man with treated glaucoma and visual acuity of OD 20/60, OS finger count at two feet, had uneventful TURP until he complained that all he could see was a little light when the room lights were turned on at the end of surgery. He was alert and oriented but could not see objects and stared ahead. Examination by

* Associate Professor of Clinical Anesthesia, Northwestern University School of Medicine, and Chief, Anesthesia Service, V.A. Lakeside Medical Center.

† Assistant Professor of Clinical Anesthesia, Northwestern University School of Medicine, and Medical Director, Respiratory Therapy Section, Anesthesia Service, V.A. Lakeside Medical Center.

‡ Professor and Chairman, Department of Anesthesia Northwestern University School of Medicine.

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Address reprint requests to Dr. Ovassapian: V.A. Lakeside Medical Center, Anesthesia Service, 333 E. Huron Street, Chicago, Illinois 60611.

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TABLE 1. Summary of Clinical Data

Case	Spinal Anesthesia Level	Resection Time	Amount of Prostrate Resected	Fluids IV during TURP	Intake and Output Hours Since Completion of TURP	Serum Sodium	
						Preoperative	Lowest Recorded
1	T7	140 min	41 g	5%D/RL 800 ml	18° Intake 3,250 ml 18° Output 7,100 ml	137 mEq/l	114 mEq/l
2	T6	90 min	35 g	5%D/RL 200 ml	24° Intake 4,000 ml 24° Output 8,300 ml	139 mEq/l	115 mEq/l
3	T6	40 min	5 g	5%D/RL 400 ml	3.5° Intake 350 ml 3.5° Output 4,100 ml	141 mEq/l	121 mEq/l
4	T10	90 min	30 g	5%D/RL 800 ml	24° Intake 2,100 ml 24° Output 5,880 ml	152 mEq/l	102 mEq/l
5	T8	90 min	32 g	5%D/RL 500 ml	24° Intake 1,600 ml 24° Output 3,000 ml	141 mEq/l	111 mEq/l

an ophthalmologist at this time with the patient wearing his corrective glasses revealed visual acuity of both eyes to be limited to light perception with no projection. Extraocular muscle movements and macula were normal, vessels in both eyes were arteriosclerotic, but no cherry red spots were seen. Intravenous crystalloid administration was restricted and 20 mg furosemide was given iv. Vision improved spontaneously but slowly. He was able to see fingers in about four h. Visual examination the following day showed a return of his preoperative acuity.

Patient 3. An 84-year-old man who had visual acuity of finger count at two feet in his right eye as a result of retinal detachment, and 20/50 vision in his left eye because of early cataract, underwent his second TURP. Venous sinuses were opened during the TURP and because of bleeding, the operation was quickly terminated. Estimated blood loss was 300 ml. On admission to the recovery room, he complained that his vision was foggy and that there were halos of light around objects. His visual acuity in the recovery room with corrective glasses was limited to light perception only without projection. He was alert and cooperative. Twenty minutes later, he was nauseated and vomited a small amount of clear fluid. Intravenous crystalloid administration was restricted and oxygen was given by nasal cannula. After four h, his vision had improved and he was discharged to his room. Eight hours after completion of the operation, his vision had returned to its preoperative level.

Patient 4. An 82-year-old man with a history of hypertension and gouty arthritis, and who was taking allopurinal, acetaminophen with codeine, and flurazepam hydrochloride was scheduled for TURP. All laboratory data were within normal limits, except for the electrocardiogram which revealed left ventricular hypertrophy and left anterior hemiblock. About 60 min after the start of TURP, brisk bleeding occurred which interfered with the resection. Shortly thereafter, the patient became nauseated, and noticed that the light shining through the window was not as bright as before. Within the next 10 min, his vision deteriorated to the level of light perception only. The EKG and vital signs were unchanged. He was treated with inhalation of oxygen, restriction of iv crystalloid administration, slow administration of 3% saline, and 10 mg dexamethasone, iv. Five hours after completion of surgery, serum sodium was 128 mEq/l, but visual status was unchanged. He noticed a return of the brightness of lights about 8 h after the onset of symptoms. By the first postoperative day, the patient's vision had returned to normal.

Patient 5. A 72-year-old man with a history of dizziness and black outs for 12 years was scheduled for TURP. These symptoms were thought to be due to basilar artery insufficiency but vertebral arteries angiogram did not show any blockage or narrowing of the arteries. His medications included persantin, meclizine hydrochloride, and aspirin. Thirty minutes after the beginning of the resection, arterial blood pressure suddenly increased from 140/80 to 190/100 mmHg with a heart rate of 70 beats/min. The patient complained of dizziness and mild dyspnea but no chest pain. He was alert, oriented, and cooperative. Oxygen was given via a mask and 20 mg furosemide administered iv. Shortly thereafter, he complained of nausea, abdominal pain, and blurred vision. Preoperative vision with corrective glasses was 20/30 in both eyes. In the recovery room the visual acuity with glasses had deteriorated to 20/100. Serum glycine level was 1029 mg/l shortly after his admission to the recovery room, 228 mg/l four hours later, and 143 mg/l eight hours after completion of surgery. His vision began to improve six hours after completion of surgery and returned to the preoperative level the next morning.

DISCUSSION

Occipital cortical edema was suggested as the possible cause of temporary blindness by Defalque and Miller in their patient.[2] In cortical blindness, visual sensation including perception of light, the blink reflex, and reflex lid closure in response to threatening gesture is lost completely. However, the pupillary response in both light and accommodation are retained, and the fundus has a normal appearance.[7] The visual disturbances observed in our patients and those in other reports do not fit the description of cortical blindness. Presence of light perception, dilated nonreactive pupils[2,3,6] in some patients, and inability of neurologists and ophthalmologists to make definite diagnosis of cerebral cortex blindness[3] cast doubt on the validity of cerebral edema as the cause of this blindness. Patient 1 in our series had dilated nonreactive pupils, numbness, and tingling of upper extremities, and vision limited to light perception; otherwise, he was rational and cooperative. Atropine, 0.8 mg, iv, usually will not cause profound dilatation of pupils, but even if it does, it should only interfere with the accommodation reflex and not the vision as has been men-

tioned.[3] Evidence of bleeding from open venous sinuses during resection, nausea and vomiting, presence of hyponatremia, and postoperative diuresis in all these patients indicate that the irrigating solution had been absorbed through the prostatic bed. Water intoxication and hyponatremia may account for many of the symptoms in TURP reaction syndrome; however, the role played by glycine in these symptoms, especially visual disturbances, is not clear and has not been explored.

Glycine is a nonessential amino acid which occurs normally in the body. The normal plasma level is 13–17 mg/l and it readily passes the blood-brain barrier. Glycine functions as an inhibitory transmitter not only in the spinal cord, but probably also at specific synapses in the medulla oblongata, pons, tectum, and retina.[8] It is released from the cat and rabbit retina after stimulation by light,[9] and it depresses the spontaneous and evoked activity of retinal neurons and hyperpolarizes cells.[10,11] Glycine probably is released from interneurons and acts as an inhibitory transmitter in the retina. When injected intervitreally in rabbits, glycine has an inhibitory action upon electroretinogram, in action which is reversed spontaneously within 24 hours. Glycine has a distribution similar to that of GABA in rat retina, the highest levels occurring in the amacrine cell, inner plexiform, and the ganglion cell layers.[12] Toxicity of intravenously administered glycine has been demonstrated in dogs[13–15] and in humans,[16,17] and the signs and symptoms include nausea, vomiting, fixed and dilated pupils, weakness, and muscular incoordination. Infusion at rates higher than 5.35 mg glycine $\cdot kg^{-1} \cdot min^{-1}$ (1 mg N $\cdot kg^{-1} \cdot min^{-1}$) in dogs is lethal.[13] In humans the infusion of 2.5% glycine at a rate of 3.58 mg glycine $\cdot kg^{-1} \cdot min^{-1}$ (0.67 mg N $\cdot kg^{-1} \cdot min^{-1}$) and of a total dose of 372.9 mg $\cdot kg^{-1}$ (69.7 mg N $\cdot kg^{-1}$) is accompanied by feelings of malaise and nausea. One subject who received 5% glycine experienced a more severe reaction characterized by malaise, marked weakness, intense nausea, and vomiting.[17] The amount of glycine solution (calculated from changes of serum sodium levels) absorbed into vascular system in our patients varied from 1,500 to 3,200 ml which represents 302 mg/kg to 608 mg/kg glycine. This amount is in a range to be toxic in human subjects.

In Case 5 of our series, we had measured serum glycine levels. This patient's serum glycine level on arrival in recovery room was 1,029 mg/l which dropped to 229 mg/l 4 hours later, and to 143 mg/l by the night of the surgery at a time when vision was returning. Because of the very high serum level of glycine which was demonstrated to coexist with visual disturbances in this patient, and considering the possible role of glycine in the neurochemistry of retinal function, we suggest that glycine itself may have caused the visual disturbances as a result of its action on the retina, the central nervous system, or both. This area obviously should be explored further.

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