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, dis-
posable 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

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Visual disturbances such as blurred vision,1–3 transient
blindness,2,5 and pupillary dilatation5,6 have been re-
ported with the transurethral resection reaction syn-
drome. These visual disturbances have been attributed
to cerebral edema2,4 and atropine injection during the
operation.3

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

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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 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, metldizne 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. 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. 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 in some patients, and inability of neurologists and ophthalmologists to make definite diagnosis of cerebral cortex blindness 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. Evidence of bleeding from open venous sinuses during resection, nausea and vomiting, presence of hypotenemia, and postoperative diuresis in all these patients indicate that the irrigating solution had been absorbed through the prostatic bed. Water intoxication and hypotenemia 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. It is released from the cat and rabbit retina after stimulation by light, and it depresses the spontaneous and evoked activity of retinal neurons and hyperpolarizes cells. Glycine probably is released from interneurons and acts as an inhibitory transmitter in the retina. When injected intravitreally in rabbits, glycine has an inhibitory action upon electoretinogram, 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. Toxicity of intravenously administered glycine has been demonstrated in dogs and in humans, 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·kg⁻¹·min⁻¹ (1 mg N·kg⁻¹·min⁻¹) in dogs is lethal. In humans the infusion of 2.5% glycine at a rate of 3.58 mg glycine·kg⁻¹·min⁻¹ (0.67 mg N·kg⁻¹·min⁻¹) and of a total dose of 372.9 mg·kg⁻¹ (69.7 mg N·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. 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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