Transient Global Amnesia Following Spinal Anesthesia

MICHAEL H. M. DYKES, M.B., B.CHIR., BERNARD R. SEABS, M.D., LOUIS R. CAPLAN, M.D.

Transient global amnesia—a syndrome characterized by the occurrence of an isolated episode of transient amnesia in an otherwise healthy patient—was first described by Fisher and Adams in 1958. The number of additional cases reported since that time has been substantial, but in no instance does the syndrome appear to have been observed during the immediate postoperative period. As we have recently observed such an episode, initially misdiagnosed as an acute toxic psychosis, we believe it is important to bring the existence of this rare syndrome to the attention of anesthetists.

REPORT OF A CASE

A previously healthy 69-year-old woman was admitted with a history of two days of painless gross hematuria. Prior to admission an intravenous pyelogram had revealed a large filling defect in the left side of the urinary bladder, and a sessile bladder tumor had been observed at cystoscopy. Past medical history revealed only that she had undergone a partial hysterectomy under spinal anesthesia 23 years prior to admission, and had had iron-deficiency anemia for the past three years. No cause for the latter condition had been found, and it had responded well to iron therapy without recurrence. She admitted to no current symptomatology apart from the hematuria.

On physical examination, the arterial pressure was 140/80 mm Hg and the pulse rate 72 beats/min. The patient was oriented and in no acute distress, and without significant physical findings. The hemoglobin was 40 per cent; fasting blood sugar, urea nitrogen, creatinine, and serum electrolytes were all within normal limits. Radiologic examination of the thorax revealed a normal heart and no acute pulmonary infiltrates, and an electrocardiograph was interpreted as being within normal limits.

Premedication consisted of atropine sulfate, 0.5 mg im, at 6:15 AM, and a spinal anesthetic (to which the patient had consented the previous evening) was administered uneventfully at 7:36 AM. Tetracon, 8 mg, with 10 per cent dextrine, 0.8 ml, resulted in analgesia (no response to pin-prick) to the sixth thoracic dermatome. Cystoscopy and transurethral resection of a carcinoma of the bladder were performed without complication, arterial blood pressure and pulse rate remaining stable throughout. The patient entered the recovery room apparently in satisfactory condition at 8:50 AM and was given mannitol, 12.5 gm iv, at 8:51 AM, and trimethobenzamide hydrochloride (Tigan),
200 mg im, for nausea at 8:55 AM. At 9:30 AM she was noted to be extremely confused and disoriented as to time and place—she knew she was in a hospital, but was not certain which one. She thought that she had had a hysterectomy for vaginal hemorrhage, while having no memory of having had hematuria or an operation on her bladder. The vital signs were stable, and no abnormalities were observed on physical examination. Serum electrolyte, blood urea nitrogen, and arterial blood gas determinations were within normal limits, as was a hematocrit. An acute toxic psychosis of unknown etiology was diagnosed.

Neurologic evaluation at 4:00 pm revealed a woman who maintained a pleasant sociable demeanor, and was neither somnolent nor agitated. She frequently repeated queries concerning the time of day and recent events. She was told the examiner’s name on ten occasions within an hour, and, despite the fact that she commented on the name as being the same as her maiden name, she could not recall the name when asked about it 30 seconds later, and always stated that it had not been told to her before. Similarly, she was unable to retain any other new information for longer than 30 seconds. She had a retrograde amnesia dating back to before her admission to the hospital, approximately three days. Her speech, reading, writing, vocabulary, and distant memory were normal. There were no abnormalities in motor, sensory, or reflex functions; an electroencephalographic examination disclosed no abnormalities. At 8:00 pm the patient seemed improved and was able to retain some new information. By the following morning her retrograde amnesia had shrunk to two days, and she could recall objects shown to her 2 minutes and 15 minutes previously. The improvement continued, and she was discharged on the eighth postoperative day. Five months after discharge, she was found to have amnesia for only the eight-hour period encompassing the operation and the morning and afternoon following it. She has not had any recurrence.

During the postoperative period, the patient’s husband volunteered that 17 years prior to admission, when they had just moved into a new house, the patient had put a turkey in the oven to cook. Later, when he had smelled a burning odor, the husband had suggested to the patient that perhaps the turkey was burning. The patient had stated that she had no recollection of putting a turkey in the oven.

DISCUSSION

Transient global amnesia is a syndrome characterized by an isolated episode of transient amnesia lasting less than 24 hours. The onset is abrupt and is without premonitory symptoms. There is usually a brief period of mild confusion, and when examined during an episode, the patient’s neurologic system is intact except for an inability to lay down lasting memory traces of any kind and a retrograde amnesia extending for days or weeks. The retrograde amnesia subsequently shrinks, and the patient is generally left with permanent amnesia only for the period during which new memories were not recorded, plus a brief retrograde period.

We observed one short episode of amnesia in our patient, which closely fits the above characterization, and obtained in retrospect from her husband the history of an earlier similar episode. We believe, therefore, that there is little doubt that our patient suffered an attack of transient global amnesia. We recommend that this rare but striking diagnosis be considered as a possibility in the case of any patient who abruptly develops amnesia during the postoperative period. This condition should be distinguished from toxic-metabolic cerebral confusional states, aphasia and other focal cerebral disturbances, and hysterical fugue states, all of which have quite different etiologies and treatments. Transient global amnesia is usually a self-limited condition, which generally does not recur and is usually not associated with serious central nervous system sequelae.

The etiology of the condition remains obscure, and several possible mechanisms have been hypothesized. Since concussion can present an identical clinical picture, and pathologic lesions in the hippocampus have been associated with permanent memory deficits, occult trauma and ischemia to the hippocampus and its connections have both been suggested as possible etiologies. In addition, epileptic seizures arising from the temporal lobe is another explanation which is frequently proffered. Fisher and Adams review in detail the arguments against each of these hypothetical causes.

Several studies of the effects of anesthesia on memory function are of interest to the anesthetist in this context. Mazzia and Randt administered inhalation anesthetic agents to human subjects until a phase of poor conjugate eye centering had been reached. These authors demonstrated that in this light level of anesthesia, the human subjects could give accurate data regarding orientation and recent events, and could calculate accurately,
but could not make a new lasting memory trace of a picture shown to them. Since the reticular formation in the brainstem is the presumed locus of action of the anesthetic agents used at that level of anesthesia, and since eye centering is also thought to be a function of the reticular formation, the authors hypothesized that the reticular formation also had an important role in making new memory traces. Further, Abt, Essman, and Jarvick studied the effects of ether inhalation on mice 0–24 minutes after they had been given a single-electric shock conditioning trial. The ether, if administered within the first 8 minutes after the shock, appeared to make the mice behave as if they had retrograde amnesia for the shock. Each of these studies demonstrates that the inhalation anesthetic agents have some action on new memory formation.

Cherkin and Harroun, in a recent review of anesthesia and the memory processes, analyze the factors contributing to amnesia for the events occurring during surgical operations. Sensory stimuli perceived during the operation in the form of pain, noise, or emotionally charged conversation, as well as the type and level of anesthesia, and the premedication, all influence the patient's memory of the procedure. Perhaps careful studies of amnestic function in the postanesthetic period will reveal a greater incidence of memory defects, and will shed some light on this important subject and on the etiology of transient global amnesia.

REFERENCES

Progressive Changes in the Concentration of Ethyl Alcohol in the Human and Canine Subarachnoid Spaces

MICHIKO MATSUMI, M.D.,* Y. KATO, M.D.,† K. ICHIYANAGI, M.D.‡

One difficulty inherent in the use of subarachnoid alcohol block to treat intractable pain of terminal cancers is that the debilitated patient must be held in an unnatural, uncomfortable position for a protracted time. The conventionally recommended time for absolute immobility of the patient, 45 to 60 minutes, seems to have been based on experience only, not on quantitative data. In the present study the changes in the concentration of alcohol in the subarachnoid space with time have been investigated in an attempt to determine how soon after the instillation of alcohol the position of the patient can be changed without danger of complications from further spread of the neurolytic effect of the alcohol.