mained so only while the patient strained. If the influence of the muscles of the abdomen was removed, the stream would cease. The patient voided better in the sitting position. During the first week after the operation on two occasions there was a residual amount of urine in the bladder of 120 and 200 cc., respectively. This became zero by the eleventh postoperative day. Concomitantly with the reduction of the residual urine the urinary stream became larger and more forceful and the muscular straining diminished notably. The urine was alkaline and contained numerous white blood cells. . . . Five weeks after the operation the urine was free of infection. Little straining was required to initiate urination. The stream was continuous, forceful and full. There was no nocturia, and the average interval between voidings by day was four hours. For three months the patient had been forced to give up his position as an engineer and had felt himself virtually a social outcast. Five weeks after operation he returned to work and again took up his customary social activities. Aside from the slight degree of straining required to initiate the urinary stream he has recovered completely and has remained entirely well." 5 references.

J. C. M. C.

MONROE, S. E., AND BENJAMIN, E. L.: *Convulsions Associated with General Anesthesia: "Ether Convulsions": Report of a Case with Findings at Autopsy.* Am. J. Surg. **53**: 172–176 (July) 1941.

"R. W., a 23 year old white man, was admitted to the Evanston Hospital on July 23, 1940, at 6 p.m. . . . Acute appendicitis was diagnosed. A hypodermic injection of morphine sulfate ¼ grain and atropine sulfate $\frac{1}{150}$ grain was given thirty minutes before going to the operating room. Ethylene-oxygen-ether mixture by closed method was administered. His pulse rate was 90; the systolic blood pressure was 120 mm. Hg. The operation began eleven minutes after the anesthetic was started. The patient seemed difficult to anesthetize. . . . When the peritoneum was about two-thirds closed, the patient was seized with a series of clonic contractions. In the beginning they were confined to the muscles of the face and neck. They quickly increased in severity to involve the trunk and lower extremities. Within two or three minutes the convulsions made difficult the completion of the operation. . . . At the time of the first convulsion the operation had been in progress sixty minutes and the anesthetic seventy-three minutes, including the time of the induction. The closure of the operative incision was continued and consumed nine minutes, during which time the anesthetic was stopped and intravenous medication started. Calcium gluconate, 10 gr., was given by vein. A few minutes later five more grains was administered. This had no effect on the convulsions. Sodium luminal, 2 gr., was given subcutaneously without apparent effect. About fifteen minutes after the onset of the convulsions the rectal temperature was 105 F. In spite of tepid sponging it rose to 107 F. The patient was placed in an ice pack; an ice water enema was given. About one hour after the onset, the severity of the convulsions had slightly decreased. They ceased after the intravenous administration of 3.75 gr. of sodium amytal. The rectal temperature was 102 F. The patient was given intravenous saline solution (3.25 Gm. per liter). One and seven-eighths grains of sodium amytal was given to control recurring convulsions. Three hours after the onset of convulsions the systolic blood pressure fell to 60 mm. Hg. The lower extremities were wrapped and elevated. Two cc. of

coramine and intravenous fluids elevated the systolic blood pressure within the next fifteen minutes to 118 mm. Hg. His pulse rate varied from 140 to 160; the respiration rate varied from 30 to 50. Perspiration was practically absent at the height of, and subsequent to, the attack. . . . Supportive treatment consisted of intravenous fluids and adrenal cortex extract. Sodium bicarbonate and lactate Ringer's solution were given to combat acidosis. Recurring convulsions were treated with sodium amytal. He had involuntary bowel movements. Catheterization was necessary. . . .

"A neurological examination was made by Dr. Harry Paskind thirty-six hours after the onset of convulsions. The patient was in profound stupor. There was no response to pin pricks. The ankle jerks were present and about equal. The knee jerks were present and fairly brisk. The left arm jerks were brisk. . . . No pathologic reflexes were present. The cremasteric and abdominal reflexes were absent. No facial asymmetry was present. The patient did not respond to external stimuli. The patient died on the seventh postoperative day. Necropsy was performed four hours after death. . . . The essential anatomic diagnosis was: Bilateral confluent bronchopneumonia; unresolved pneumonia with beginning carnification; necrotic hemorrhagic infarct of the left upper pulmonary lobe; pulmonary thrombosis; dilatation of the right heart; mild diffuse hemorrhages of the pia-arachnoid; edema and perivascular hemorrhages of the brain; hypoplasia of the aorta; involution of the thymus; atrophy of the thyroid; hyperplasia of the mediastinal and abdominal lymph-nodes; status post appendectomy; healing McBurney's incision. Dr. Arthur Weil, Associate Professor of Neuropathology at Northwestern University Medical School, re-examined the microscopic sections of the brain. The severe generalized ganglion-cell degeneration found as a result of heat stroke or hyperthermia was not observed. . . . The official temperature of the outside air as given by the Chicago weather bureau was 92 F. at 9 p.m., which was approximately the time that the operation started. . . .

"The etiology of convulsions under general anesthesia is not established and the treatment is not standardized. The recommendations made by Lundy in 1937 are of interest. They are: '1. Convulsions can probably be controlled by the use of barbiturates intravenously. 2. The most dangerous cases are those in which there is profound toxemia and therefore in selecting the anesthetic for such cases it might be better to use spinal, infiltration, or block anesthesia or avertin to produce basal anesthesia, than to use an inhalation anesthetic only.'" 7 references.

J. C. M. C.

BUCHSTEIN, H. F.: *Acute Spinal Epidural Abscess: Report of Case.* Minnesota Med. **24**: 593–595 (July) 1941.

"Pyogenic infections of the spinal epidural space may result from the direct extension of an adjacent lesion, such as a carbuncle or perinephritic abscess. More commonly, however, they arise by hematogenous metastasis from a distant focus of infection. Cutaneous lesions, such as furuncles, paronychiae and infected blisters are the most frequent sources of the infection. Less commonly, infections of the paranasal sinuses, the respiratory passages or the teeth may be responsible. Many such metastatic epidural infections follow minor trauma to the region of the spine, which is then presumed to have created there a focus of lowered resistance in which a septic embolus lodges. The infecting organism is most often the staphylococcus. Metastatic