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.

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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

spinal epidural infections appear to occur more frequently than the brief comment they receive in most surgical texts would suggest. During the course of a year I have had occasion to operate upon two patients with such infections, one of whom is here reported.

"A young married woman of 19 years was in the fifth month of her first pregnancy when she was found to have a cystic ovarian tumor. Removal was advised, for which purpose she was admitted to the Gynecological Service of the Minneapolis General Hospital. . . . Laparotomy was performed on June 5, 1940, under spinal anesthesia. Difficulty was encountered in entering the vertebral canal, repeated withdrawals and reinsertions of the needle being made, all in the same interspace. Eventually the dura was pierced and the drug (metycaine, 125 mg.) was injected. Satisfactory anesthesia was produced and the operation was completed without further untoward incident, a large ovarian cyst being removed. The patient's wound healed promptly and she was afebrile and comfortable upon being discharged from the hospital on the tenth postoperative day. Three days later she developed low back pain which became progressively more intense and which presently radiated in girdle fashion about her lower abdomen, just above the inguinal ligaments. Straining and bearing down intensified the pain. Subjective numbness was noted in the left foot. . . . The subjective numbness spread upward over both lower extremities, and both lower extremities became weak. On the second hospital day the patient was unable to void and catheterization was thereafter necessary. Presently the skin over the lower lumbar spine became reddened and elevated, and the patient cried out with pain upon any movement of her lumbar spine. On June 25, because of the development of headache and a stiff neck, spinal puncture was attempted. However, when a needle was inserted in the site of the previous puncture pus was obtained before the needle had penetrated the dura. A diagnosis of acute spinal epidural abscess having been made, the patient was transferred to the surgical service and preparation was made for immediate laminectomy. . . .

"Postoperatively the patient was placed on sodium methylthiazol orally, in sufficient dosage to maintain a blood level of 11 to 14 mg. per cent, and this was continued until her temperature reached a normal level, which it did at the end of seven days. During this week she was given daily blood transfusions, a total of 4,825 cc. of blood being given. On the advice of the Obstetrical Service, 100 per cent oxygen was administered via a B.L.B. mask at frequent intervals during the day for several weeks. Under this regimen the patient made steady improvement. . . . On the eleventh postoperative day the patient complained of pain in the right side of her face, had a chill, and her temperature rose to 101.5°. The following day there was a spontaneous discharge of pus into the right maxillary sinus and the mouth. This was found to be coming from a large dental root abscess beneath a necrotic third upper molar. The tooth was extracted and the patient's temperature promptly returned to normal. This dental abscess is regarded as the probable source of the metastatic epidural infection, since it is the only other infectious focus found in the patient's body. . . . The patient was allowed to sit in a chair at the end of twenty-five days, and was walking about in comfort upon her discharge from the hospital on the thirty-fifth postoperative day. Her wound was healed. The patient was readmitted to the Minneapolis General Hospital on the Obstetrical Service on October 25, 1940, just four months

from the day of operation, and was delivered of a normal full term female infant without incident. At this time her wound was firmly healed and she had no symptoms or complaints referable to her back or lower extremities.

"This appears to be the first recorded instance of spinal epidural abscess arising as a complication of spinal puncture. It seems reasonable to assume that as a result of the repeated insertions of the spinal puncture needle a hematoma formed in the epidural space at the site of puncture, or sufficient injury was done to the interspinal ligaments to provide a necrotic area in which, subsequently, a septic embolus from the dental route abscess lodged. The long interval between the spinal puncture and the onset of symptoms, and the absence of leptomeningitis make it unlikely that the infecting organisms were introduced by the spinal puncture needle. . . . Without surgical drainage, acute spinal epidural abscesses are almost invariably fatal, and even with such drainage the mortality has in the past been high. . . . The survival with good functional recovery which followed in the two cases which I have operated upon may be attributed to three factors: (1) prompt and adequate laminectomy when the diagnosis was made; (2) the free use of postoperative transfusions as a supportive measure; and (3) intensive chemotherapy before and after operation." 5 references.

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MOUSEL, L. H., AND LUNDY, J. S.: *Some Pulmonary Problems Related to Anesthesia.* Southwestern Med. 25: 166–169 (June) 1941.

"Preliminary medication may be responsible for the development of pulmonary complications during the induction stage of anesthesia, during surgical anesthesia or during the postoperative period. If excessive premedication has been given, a great deal of difficulty may be encountered in anesthetizing the patient. Respirations may be so shallow that it is impossible to administer enough of the anesthetic agent to the patient to produce relaxation unless the anesthetic is administered by artificial respiration. On the other hand, if the patient has had an insufficient amount of preliminary sedation, he may inhale the anesthetic well but will require considerably more ether than the one who has been prepared adequately. Morphine may depress the respiration sufficiently during and immediately after operation so that an anesthetic such as ether is expelled very slowly from the lungs. This might be partly responsible for an increase in the incidence of postoperative pneumonia. It is of particular advantage under such circumstances to allow the patient to breathe 5 per cent carbon dioxide and 95 per cent oxygen in order to eliminate the ether as rapidly as possible, for once the ether has been eliminated, the patient is usually no longer depressed. . . .

"During spinal anesthesia, whether it is used alone or in combination with intravenous anesthesia, respiration must be watched closely, for anoxemia may develop and may be unrecognized until it advances to a point where it may terminate fatally regardless of one's efforts to combat it. This situation is essentially a pulmonary problem occurring as a result of the spinal anesthetic paralyzing the intercostal muscles and the intravenous anesthetic paralyzing the respiratory center. One must recognize the decrease in volume of respiration rather than wait for respiration to stop entirely, for if such a point is reached, the outcome may well be fatal. The treatment is the early administration of oxygen, perhaps by artificial respiration, that is, by intermittent compression of the bag containing the oxygen. . . . The routine postoperative administration of mor-