level of anesthesia on the dependent side with continuous cramps on the upper side.

"The continuous caudal equipment is quite difficult to manage when the patient is being moved. A long tube protrudes from the back of the patient. One must be careful not to pull on that tube the slightest as the needle will be displaced or the tubing pulled from the needle hub. The accompanying tray of bottles, syringes, etc., which one must keep sterile, is difficult to manage for eight hours.

"No special equipment is needed for single caudal blocks."

A. W. F.

ROMAN-VEGA, D. A., AND ADRIANI, JOHN: *The Efficiency of "Oenethyl" (2-methyl-amino-heptane) as a Vasopressor Substance for Spinal Anesthesia.* South. M. J. **38**: 635–641 (Oct.) 1945.

"It is a well recognized and established fact among anesthetists that vasopressor substances are the most effective therapeutic agents for combating the hypotension which frequently accompanies spinal anesthesia. The vasopressor substances usually employed are ephedrine, 'neosynephrin,' and to a certain extent, propadrine and epinephrine. These substances belong to a group of chemically related aromatic amines. Although these aromatic amines are used extensively and serve the purpose satisfactorily so far as control of hypotension is concerned, they possess certain side effects and a variability of action which often limit their usefulness. Tachycardia, palpitation, dizziness, tremor, nausea, sweating, pallor and coldness of the skin are some of the objectionable features which curtail their clinical value and often cause another vasopressor to be desired. Recently, a number of aliphatic or straight chained amines have been prepared which possess not only a vasopressor action, but also cause responses which suggest sympathetic stimulation. One of these, 2-amino-heptane, also known as 'tuamine' has been used as a vasoconstrictor in the nasal passages. Another of these, 2-methyl-amino-heptane or 'oenethyl,' likewise is pharmacologically related to 'tuamine.' . . . Inasmuch as aliphatic amines have never been employed for overcoming the hypotension of spinal anesthesia, the writers have been interested in determining the clinical value of this type of compound for this purpose. A drug of this sort suggests other clinical uses, particularly those for which epinephrine is employed. However, this report, which includes experiences with the drug in seven hundred surgical patients, deals exclusively with its use in spinal anesthesia. . . . 'Oenethyl,' or, 2-methyl-amino-heptane, was used effectively to combat hypotension during spinal anesthesia in seven hundred operative cases. With few exceptions, side actions such as nervousness, palpitation, headache, dizziness, sweating and so forth were not encountered. The substance appears to be a satisfactory vasopressor for spinal anesthesia and is worthy of further clinical trial." 2 references.

J. C. M. C.

BELINKOFF, STANTON: *Coma During and Following Spinal Anesthesia.* Ann. Surg. **122**: 278–286 (Aug.) 1945.

"It has long been known that severe anoxemia produces damage to the brain tissues, the cortical areas being the most vulnerable. . . . It has been demonstrated experimentally and seen clinically that the extent of brain damage is directly proportional to the duration and severity of the anoxemia. In this report, anoxia of sufficient extent to produce coma during spinal anesthesia will be considered. . . .

The etiology of anoxia in spinal anesthesia is varied. Three chief factors are involved. If the anesthesia reaches too high a level, the resultant paralysis of the intercostal muscles causes a decrease in the vital capacity. The impaired respiratory excursions lead to decreased oxygenation, which, if not counteracted with inhalations of high concentrations of oxygen, leads to fatal anoxia. If the level of anesthesia reaches inordinate heights, it may involve the diaphragm, in which case respirations must be maintained by artificial means such as intermittent manual compression of the rebreathing bag of an anesthesia machine or artificial respiration by any of the accepted methods of manual compression of the chest, such as the Schaeffer. The former is the better since efficient ventilation with high oxygen concentrations can be attained.

"Secondly, the drop in blood pressure associated with any of the factors in an operative procedure, such as hemorrhage, reflexes from traction on the mesentery, or the vasodilatation of the spinal anesthesia itself, if allowed to persist, leads to surgical shock. The hypotension results in severe anoxia of the cerebral centers. When the respiratory center is subjected to acute anoxemia it may be markedly depressed, resulting in apnea of central origin. Third, mechanical factors may cause anoxia from impaired respiratory excursions. The use of abdominal packs against the diaphragm, retractors against the costal margins, assistants leaning on the chest, steep Trendelenburg position, and kidney or gallbladder bars, all tend to inhibit respiratory excursions and may lead to anoxic anoxia intense enough to precipitate a collapse.

"Anoxia, when severe, produces unconsciousness for varying periods of time, and the effects may be divided into four groups, depending upon the severity of the anoxia and the changes it produces. When remedied it may resemble syncope, with rapid recovery of consciousness. In cases where the anoxia has persisted for a slightly longer time, the patient may remain unconscious for periods up to 24 or 36 hours, but recover without sequelae. After sublethal periods of anoxia, the patient eventually recovers consciousness and lives, but shows signs of permanent brain damage. In the final group, the patient either dies on the operating table or remains comatose in the postoperative period, with signs of cortical degeneration until death occurs in several days. . . . The occurrence of coma during spinal anesthesia is a sign of severe anoxia of the cerebral centers. If this anoxia is allowed to persist for more than several minutes, death ensues, but when it is recognized as such as soon as it occurs and the proper physiologic therapy instituted immediately, the condition can be remedied and the patient saved. In some instances the recovery is preceded by a period of coma lasting up to 24 hours. The therapy consists of prompt administration of oxygen, vasopressor drugs when necessary, Trendelenburg position, patency of the airway and intravenous fluids. In order to be able to carry out this regimen the following precautions should be observed: (1) A competent anesthetist should always be present and in charge of the patient's vital functions. (2) An anesthesia machine should be in the room. (3) A tray of stimulant drugs and syringe at hand. (4) Pharyngeal airways and endotracheal equipment at hand. (5) Use of Trendelenburg position. (6) An infusion in major cases to provide a patent vein in case of peripheral collapse. If these precautions are observed and immediate corrective measures taken when trouble does occur, the diagnosis of death from col-

lapse of the patient under spinal anesthesia will rarely be made." 8 references.

J. C. M. C.

YASKIN, H. E., AND ALPERS, B. J.: *Neuropsychiatric Complications Following Spinal Anesthesia.* Ann. Int. Med. **23**: 184–200 (Aug.) 1945.

"In the past few years we have been concerned with a number of neurologic and psychiatric problems in which the question of a relationship to a previously administered spinal anesthetic was pertinent. We are reporting six personally observed instances of neuropsychiatric complications following spinal anesthesia. We are also describing a case of metastatic spinal cord neoplasm which came to light following spinal anesthesia in which the anesthetic agent was for a while suspected as being the cause of the myelitic syndrome. These cases are presented not as a disparagement of a very valuable, if not indispensable, form of anesthesia, but with the intent to emphasize the necessity of looking for and recognizing complications, and, if possible, preventing them. . . . In all of the first four cases whose postanesthetic toxic neural complications were definitely established, the cocaine derivative used was known; one received pontocaine, two procaine and one metycaine. The sites of injection were the lumbar subarachnoid spaces between the third and fourth lumbar vertebrae or lower. In this series there was an instance of transverse myelitis, two cases of cauda equina neuritis and conus medullaris involvement and a case of pure pyramidal tract involvement. The time elapsing between the spinal anesthesia and the appearance of the neural complications was almost immediate in all cases. In one of the four cases the neurologic disturbances appeared within two to five days after the anesthetic. Clinically all of these cases of post-spinal anesthetic neural complication showed little recovery after periods ranging from one to four years. The spinal fluid showed no characteristic picture. There was no pleocytosis or increase in spinal fluid total protein except in . . . [one]case. . . . Occasionally spinal anesthesia may be falsely accused of causing neurological disturbances. . . . Two of the reported cases were in the nature of conversion hysteria 'paralysis' of the lower extremities. The conversion mechanism was conditioned by the patient's subjective experience with spinal anesthesia. A case of metastatic spinal cord neoplasm, which came to light immediately following spinal anesthesia, . . . illustrates the importance of keeping in mind the possibility of preexisting neurologic disease when evaluating the role of spinal anesthesia in the causation of postoperative neurologic sequelae." 59 references.

J. C. M. C.

KREMER, MICHAEL: *Meningitis after Spinal Analgesia.* Brit. M. J. **2**: 309–313 (Sept.) 1945.

"Seven cases of meningitis following spinal analgesia and one following diagnostic lumbar puncture [occurred in the Middle East]. The condition is a low-grade meningitis caused by a variety of organisms introduced at the time of lumbar puncture. The main clinical features are the chronic nature of the illness and the tendency to relapse. It is suggested that this is due to the formation of adhesions containing infected C.S.F., which is liberated from time to time. These adhesions may cause spinal block or hydrocephalus. . . . The irritation produced by the analgesic may enable an organism to gain a foothold. Treatment is mainly prophylactic." 21 references.

J. C. M. C.