late in the pharynx. . . . The control of barbiturate addiction largely rests with the medical profession. First, the physician should determine the cause of the insomnia and not merely treat it as a symptom, which is so often done. Second, under no circumstance should the patient be told the name of the drug being prescribed. Third, a prescription for the barbiturates should be refilled only under the same conditions which govern the refilling of prescriptions for morphine. Finally, when prescribing drugs which may be habit forming, every practitioner should bear in mind that prevention is better than cure, and, if the patient has any psychopathic stigmata, they should be prescribed only when all other methods of treatment are exhausted." 5 references.

J. C. M. C.

RAY, B. S., AND MARSHALL, V. F.: *Convulsions During General Anesthesia: Report of Twelve Cases.* Ann. Surg. **118**: 130–148 (July) 1943.

"It is not often that there is justification for emphasizing a single symptom when considering the nature of a pathologic process but in the case of convulsions that occur during anesthesia there may be some excuse. A convulsion from whatever cause is always a dramatic event and when it occurs during an operation it is frequently the first and, indeed, may be the only recognized sign of what is believed to represent a serious and potentially fatal state. . . . The ages of the patients varied from two to 44 years. Three were children under seven years, and the rest were 27 years, or over. One-third were males. . . . One case occurred in March, two in April, two in June, three in August, two in September, and two in October. . . . All of our patients were operated upon in air-conditioned rooms where constant temperature and humidity were maintained. . . . Two of the adults had had convulsions in early childhood, and one was given to syncopal attacks under conditions of fatigue. . . . The diagnosis in three patients was appendicitis with peritonitis; appendicectomy and drainage were performed in each. In two patients the diagnosis was chronic cholecystitis, and in one, acute cholecystitis. . . . In two patients there was disease of the common bile duct requiring tedious exploratory operation, with choledochostomy in one and repair of a stricture in the other. Two patients required pelvic operations, one for tubal pregnancy, the other for an ovarian cyst, and each had had preceding uterine curettage. One patient was explored abdominally for an hypernephroma with extensive metastasis to the liver. One patient, an infant of two years, underwent a long operation and difficult anesthesia for repair of a cleft palate. . . . In our series, four of the 12 patients were acutely ill with high fever when they came to operation and, in addition, one child had not made a complete recovery from a respiratory infection. . . .

"Dehydration and acid-base imbalance, other states which so frequently accompany febrile diseases, may contribute to the initiation of convulsions by inhibiting the cells of the brain in their ability to utilize oxygen. Significance may be attached to the fact that many of the factors which have been thought to account for convulsions during anesthesia cause an increase in the H-ion concentration of blood and tissues. Deficiency in the nutritional state was present in at least three of our patients. . . . It can be postulated that heavy retraction of abdominal wound edges, trauma, excessive exposure of viscera to cooling, loss of blood, and other circumstances that are ordinarily associated with shock and 'stagnant anoxia,' may be said to be conducive, as well, to the development of con-

vulsions. But we refer more particularly to the position of the patient on the operating table. . . . The medication used in all patients of this series was limited to a standard dose of atropine with the addition, in adults, of 10 mg. of morphine sulfate administered hypodermically 30 to 40 minutes before induction of the anesthesia. Barbiturates were not given to any of the patients preoperatively. . . . The types of anesthesia employed in the cases reported were: 'Open mask' ether in three (namely in the three children), 'closed mask' ether vapor with nitrous oxide and oxygen in six cases, ethylene (with a small amount of ether vapor) in two, and cyclopropane (with a small amount of ether vapor) in one. . . . It is to be emphasized that each patient was under deep anesthesia; perhaps several were too deep, but all were thought to be in no danger up to or near the time convulsions appeared. . . . The possibility that some patients have an idiosyncrasy to the anesthetic agent is difficult to evaluate but some have thought that such a state might exist, particularly with ether. . . . In our series one patient had an uneventful 45-minute anesthetic two days prior to the operation during which she developed convulsions, and ether vapor, by the closed method, was used both times. Another patient had a longer and more difficult operation without convulsions six months after the one in which convulsions occurred, and again ether vapor, by the closed method, was used both times. These experiences, and others, tend to refute arguments in favor of individual idiosyncrasy or allergy to ether. . . . The analysis in each [of three samples of ether] showed: acetic acid eight parts per million; aldehyde and peroxide less than one-half per million. Tests made of control specimens of ether taken from freshly opened small cans and drums of ether showed the same values and these are far below the U.S.P. levels for impurities. . . .

"It appears significant that the time found to elapse between the induction of anesthesia and the appearance of convulsions was between 40 and 90 minutes, with an average of 70 minutes. This suggests that whatever elements combine to cause the convulsions, time is required for their effects to appear. . . . In none of the patients was there any cyanosis observed prior to the onset of convulsions and in four there were no untoward signs of any kind. In two there were respiratory changes only, such as 'labored,' 'jerky,' 'increased' breathing. In four there was, in addition to respiratory changes, a significant increase in cardiac rate. In only two were there impressive changes in respiration, increase in cardiac rate and rise in blood pressure. The changes noted usually occurred gradually over a period of 20 to 30 minutes but in other instances they developed within 5 to 10 minutes prior to the convulsions. . . . Only one of the patients gave evidence prior to the onset of convulsions of developing an increase in body temperature and in this patient nearly an hour passed between the appearance of hyperthermia and convulsions. . . . There can be little doubt that under the conditions of increased body temperature, whether it be due to fever, overdose of atropine or external heat, patients, and especially children, are more prone to develop convulsions. It is also true that convulsions of themselves cause an increase in body temperature. . . . The convulsions in all cases followed the same pattern, beginning in the face and spreading to the entire body. . . . Death occurred in three patients, making a mortality rate for the series of 25 per cent. . . . All the patients who did not die showed either delayed recovery from anesthesia or late sequelae or both. . . . One patient has what ap-

pears to be permanent changes in temperament and personality while another has a residual hemiparesis. . . . It has been especially illuminating to find that in four of the seven patients subjected to electro-encephalography at varying periods after their operations, there were pathologic changes recorded. It would be useful to know whether changes which may be found early after the convulsive episode would persist but our data does not supply this information. Also, it would be necessary always to take into consideration the possibility that patients may have had abnormal brain waves prior to the time of the operation. . . . The clinical evidence for damage to other organs of the body as a result of the anoxia, presumed to have caused the convulsions, is to be found in the high incidence of postoperative pulmonary complications (in seven of the ten patients who lived longer than a day after operation) and of albuminuria (in five of the ten). . . . Various blood studies made soon and late after the occurrence of convulsions in a number of the patients failed to disclose any significant changes. . . .

"When convulsions do occur it is advisable to (1) discontinue the anesthetic, (2) terminate the operation as quickly as possible; (3) administer oxygen; (4) correct any unfavorable position on the operating table; (5) keep the airway open (bronchoscopic aspiration may be required in case of atelectasis); (6) give some form of soluble barbiturate intravenously to control the convulsions, such as sodium amytal, sodium phenobarbital, or pentothal sodium; (7) replace blood or fluid loss; and (8) allay hyperthermia by sponging the body or irrigating the rectum with cold water. An oxygen tent provides the dual service of cooling and supplying adequate oxygen. There may be advantage in administering hypertonic glucose solution intravenously, particularly to combat unrecognized hypoglycemia and intravenous calcium gluconate or intramuscular parathormone to correct calcium imbalance." 61 references.

J. C. M. C.

MAHONEY, E. B., AND HOWLAND, J. W.: *Treatment of the Severely Burned Patient with Special Reference to Controlled Protein Therapy.* New York State J. Med. **43**: 1307–1310 (July 15) 1943.

"The entire premise of fluid therapy depends on the daily evaluation of the patient and the replacement of plasma, whole blood, water, or electrolytes as may be required. . . . The immediate and adequate instigation of replacement therapy (fluid and protein) is the most important feature in the primary treatment of severe burns. Replacement therapy should be carefully controlled by continuous observation of plasma specific gravity (protein) and hematocrit or hemoglobin. The toxemia of burns will be minimized if not entirely prevented by adequate fluid and protein replacement." 28 references.

J. C. M. C.

MURRAY, LT. C. K.; HALE, LT. COMDR. D. E., AND SHAAR, CAPT. C. M.: *The Preparation and the Use of Red Blood Cell Suspensions in Treatment of Anemia.* J. A. M. A. **122**: 1065–1067 (Aug. 14) 1943.

"The red blood cells which remain after the plasma has been aspirated by means of a closed aseptic technic are used for the preparation of the red blood cell suspension. After the plasma is removed, the aspirating needle is plunged to the bottom of the red blood cell layer and 200 cc. of cells is drawn over by means of a vacuum into a sterile 300 cc. dispensing bottle which contains 100 cc. of 5 per