thene deteriorates rapidly at any temperature, with the formation of aldehydes in considerable quantity. These aldehydes, on the addition of an acid or any hydrogen ion, will polarize and form resins which are definitely toxic to laboratory animals, causing convulsions. On the other hand, the mental symptoms would appear to indicate some form of cerebral damage, fortunately not of a permanent nature. So far as the author is aware no death under vinesthene anesthesia has yet been recorded, although in experimental animals an overdose has resulted in death from a condition resembling acute yellow atrophy. It is therefore recommended that anesthesia with vinesthene should not exceed an hour in duration. The author has, however, given it for three and a half hours without the occurrence of any untoward symptoms. Until a fatality occurs, which he hopes may never take place, it is unlikely that the pathology of vinesthene convulsions will be determined.

A. S.

COOK, W. B.: *Convulsions associated with nitrous oxide-ether anesthesia.* Northwest Med. **39**: 182–183 (May) 1940.

"This patient, a thin female of 29 years of age, was operated on at the Swedish Hospital on August 7, 1939. She had never had chorea or epilepsy.

"Nitrous oxide and ether anesthesia was administered by one of the regular anesthetists of the hospital. She had one-sixth of a grain of morphine and $\frac{1}{150}$ grain of atropine three quarters of an hour before operation. The cervix was coned out with a cervical electrode and a supravaginal hysterectomy for fibroid of the uterus was done. She was a very excitable and apprehensive person and had refused spinal anesthesia. She took the anesthetic poorly and there was more or less labored breathing at times.

"As the fascia was being closed, which was one hour and fifteen minutes from the beginning of the operation, she began to have twitching of the eyelids which rapidly spread to the muscles of the face and neck, and then to the arms and finally the entire body was in generalized convulsions. There was embarrassment of respiration and her pulse was weak. The convulsions actually lasted twelve minutes, but it seemed a lot longer than that to me.

"She was given oxygen without any improvement and then 15 cc. of calcium gluconate were given in the vein, thinking the condition might be related to tetany. This did not improve the situation. Five cc. of a five per cent. sodium solution were injected in the vein, and the convulsions rapidly subsided. The operation was completed and she was returned to her room. The convulsions did not recur.

. . .

"Perhaps the causes of convulsions during general anesthesia are many and varied, and cannot be satisfactorily explained. It may suffice to know that such a condition, paradoxical as it may seem, may occur when you think the operation is almost completed. These convulsions occur late. They begin in the muscles around the eyes and face. The pupil is dilated, and instead of crowding the anesthetic, it should be removed entirely. Dr. Lundy recommends intravenous barbiturates, and in this case pentothal sodium was very helpful." Bibliography—4 references.

J. C. M. C.

MURPHY, FRANK J.: *Anesthesia and anoxemia in relation to the use of nitrous oxide.* Surg., Gynec. & Obst. **70**: 741–743 (Apr.) 1940.

Recent medical literature has brought forth a wealth of articles deal-

ing with the untoward effects following nitrous oxide anesthesia. Some hold that anoxemia or tissue anoxia is responsible; others maintain there is a toxic action of nitrous oxide itself.

There have been certain errors present in the clinical use of this drug, and these errors have come to be looked upon as truths, through long usage, particularly by those with little or no clinical experience. Unfortunately, some recognized authorities have set forth the dictum that anoxemia during the administration of nitrous oxide is a normal and harmless condition. Undoubtedly a large amount of damage has been done by administering nitrous oxide without sufficient oxygen. This is because wrong methods have been used, and it should be recognized that the damage is due to anoxemia.

It has been accepted by clinical anesthetists that nitrous oxide is neither toxic nor is it irritating to the tissue. There seems to be no evidence that patients have suffered ill effects when nitrous oxide has been given in the presence of sufficient oxygen. Nitrous oxide is a very weak anesthetic, and although it will produce sleep in most patients, it does not possess the property of producing muscular relaxation. Adjuncts which will produce the necessary relaxation fall under four headings: (1) premedication, (2) block, (3) addition of another general agent, and (4) asphyxia. Asphyxia is not a part of normal anesthesia.

It can not be too strongly stated that asphyxia should not be a normal accompaniment of nitrous oxide or any other anesthetic agent.

If by the term "surgical anesthesia" we mean a state in which muscular relaxation is present with adequate oxygen concentration, there is no such thing as surgical anesthesia produced by nitrous oxide and oxygen.

It is true that nitrous oxide properly given has very definite limitations as an anesthetic agent. If muscular relaxation is present with nitrous oxide, there must be more or less anoxemia. Therefore, any report of a laparotomy done under nitrous oxide anesthesia is also a report of a case of anoxemia.

If the limitations of nitrous oxide are understood and too much is not expected, it has a proper place in the armamentarium of the anesthetist.

If nitrous oxide is given with sufficient oxygen, it can be given for any length of time and to patients of any age.

The abandonment of secondary saturation technique, the promiscuous use of nitrous oxide by unskilled attendants and others in dental offices, and the "pushing" of nitrous oxide in surgical cases will soon prove that asphyxia, not anesthesia with nitrous oxide, is responsible for the untoward effects which recently have been receiving attention.

B. B. S.

BUREAU OF LEGAL MEDICINE AND LEGISLATION, AMERICAN MEDICAL ASSOCIATION: *Regulation of the sale of barbiturates by statute.* J. A. M. A. **114**: 2029–2036 (May 18) 1940.

"Twenty-seven states have enacted laws, as of May 1, 1940, regulating the sale of barbiturates. In all but one of these states, retail sales of such drugs to consumers may be made only on prescription. . . . The laws that have been enacted follow no well defined pattern with respect either to the framework of the law or to the drugs included. . . . In practically all of the states compounds, derivatives and preparations of the included drugs are covered. . . .

"In some of the laws reference will be found to a requirement imposing on pharmacists a duty to retain prescriptions for barbiturates or other included drugs in their files for a definite period of time. The absence of such a refer-