fairly normal respiration, and the cyanosis was relieved. The blood pressure gradually rose to 140/60. However, hemorrhage continued from the tracheobronchial tree, nasopharynx, and into the paraorbital tissues with marked swelling of the right cheek. An incision was made inside the mouth to decompress the swelling of the right cheek, and a pressure bandage was placed over the eyes. The nasopharynx was packed through the mouth to control the hemorrhage and also to prevent blood from running into the trachea; the anterior nares were then packed with gel foam. The bronchoscope was left in place for fifty minutes, but bleeding from the tracheobronchial tree continued, so an endotracheal tube was inserted to facilitate aspiration and to maintain an adequate airway. The patient was returned to his room in fair condition. After returning to his room, the patient was placed on continuous oxygen, the tracheobronchial tree was aspirated frequently, and a blood transfusion was started. There were definite Cheyne-Stokes respirations for the first hour after being returned to his room, and he did not regain consciousness for about three hours. . . . On the afternoon following the explosion, subcutaneous emphysema developed over the right anterior chest. Bleeding from the tracheobronchial tree continued, and about 600 c.c. of bloody fluid had been aspirated through the endotracheal tube. However, hemorrhage from the nasopharynx and into the paraorbital tissues was apparently controlled. A portable chest roentgenogram examination was made which revealed marked subcutaneous emphysema of the thoracic wall and cervical area; marked bilateral infiltration of the lung fields, although more extensive on the right side; and a small pneumothorax space on the right side with mediastinal emphysema.

"The following morning (Jan. 22, 1950) the patient appeared in fairly good condition. The blood pressure was normal (preoperative level), the subcutaneous emphysema had not increased, there was no dyspnea, and bleeding from the tracheobronchial tree had apparently stopped. The packing from the nasopharynx and nose was removed; also the endotracheal tube. Following this, the patient was able to breathe freely, and was quite comfortable. In the afternoon of the second postoperative day (Jan. 23, 1950) the patient developed progressive respiratory distress, the subcutaneous emphysema increased and extended to the head and neck, and the degree of pneumothorax was greater by physical examination. An intercostal catheter was introduced on the right side and attached to underwater drainage. There was an immediate escape of air with relief of the dyspnea, and the patient was again comfortable. . . . On the fourteenth day following the explosion, Feb. 4, 1950, the patient was discharged to the care of the ophthalmologist. . . . The exact mechanism of the anesthetic explosion in the case presented has not been ascertained."

A. A.


"Although one of the main undesirable effects of the antihistamines in therapeutic usage appears to be due to depression of higher centers, the symptoms observed after overdosage often appear to result from central nervous system stimulation. . . . Inasmuch as the anticonvulsant properties of the barbiturates are well established, it was felt prudent to ascertain whether or not they would serve as a useful antidote in instances of poisoning by antihistamines. . . . Sodium pentobar-
ABSTRACTS

hital increases the intraperitoneal LD₅₀ of tripelennamine (Pyribenazine), diphenydramine (Benadryl), chloroprophénypyridamine (Chlortrimeton), and phenindamine (Theophorin) in mice. Sodium pentobarbital acts similarly for tripelennamine and diphenhydramine in rats but not for chlorophrophénypyridamine. In rats overdosed orally with tripelennamine or diphenhydramine, the death rate is not significantly affected by sodium pentobarbital, although convulsions are aborted in part and survival time may be increased. It is suggested that the degradation products of the antihistamine, which are present in greater amounts after oral administration, may enhance the actions of sodium pentobarbital and contribute to the over-all toxicity. It is recommended that should the attempt be made in the clinic to antagonize antihistamine overdosage with a barbiturate, immediate treatment and removal of any unabsorbed material are prime requisites in addition to the usual supportive therapy.”

A. A.


“For the reduction of Colles’s fracture and similar injuries, the anaesthetic which is probably most often employed is nitrous oxide. This, however, has several drawbacks..." Brachial-plexus block has been used for the reduction of Colles’s fracture; but it is time-consuming, not without danger, and, in inexpert hands at any rate, often fails. The anaesthetic of choice in the treatment of this condition appears to be local infiltration anaesthesia. . . . A modification of this technique has recently been tried at the Upton Hospital with promising results. Before injection the local anaesthetic agent (for Colles’s fracture 20 ml. of 1% procaine) is mixed with 1000 ‘Benger units’ of hyaluronidase (“Hyalase”), which promotes diffusion of injected substances. Two injections are made: the bulk of the solution is put directly into the fracture hematoma from the extensor aspect of the forearm, and 2–3 ml. is infiltrated around the ulnar styloid process. The anaesthetic solution diffuses rapidly all around the injured area and the fracture can be manipulated as soon as the needle is withdrawn. . . . To date, this technique has been used in 22 cases of Colles’s fracture and 4 cases of Pott’s fracture. . . . Though the series is very small, we have been struck by the rapidity with which analgesia is achieved and by the fact that it has always been complete.”

A. A.