Local analgesic drugs, either from overdose or hypersensitivity may produce clonic muscle spasms or generalized convulsions. An intravenous barbiturate, sufficient to relax the muscles is considered the best antidote. Fall in blood pressure during spinal anesthesia may result in secondary hemorrhages. Constant blood pressure control is essential during spinal anesthesia.

Intravenous anesthesia may be made safer by the general rule "the weaker, the safer." Over-premedication, expecting full relaxation, perivascular or intra-arterial injection, and administration of intravenous anesthesia without all safety devices at hand, are some of the hazards of the method.

Every patient under general anesthesia deserves an artificial airway. Every anesthetist should master the technic of endotracheal intubation. Intravenous fluids, blood, plasma and saline-dextrose solutions, should be available for immediate use. Vasopressor drugs should be ready for use when necessary. Curare may be classified as one of the safety agents in the modern anesthetist's armamentarium.

Suction as a safety measure cannot be overestimated. Mechanical augmentation of shallow or slow respirations has largely replaced the older methods of chest compression and withdrawal of the anesthetic.

Cardiac arrest differs from failing circulation and is treated by such emergency measures as support of the respiration, cardiac puncture, cardiac massage and injection of adrenaline or other similar drugs into the cavity of the the left ventricle.

Postanesthesia care begins in the operating room when the anesthetist takes steps to revive the patient as much as he can. Suction of the mouth and trachea if indicated, suitable artificial airway and ensuring that no depression or collapse exists, are some of the immediate steps in postanesthesia care. Nurses on the ward should be instructed in proper care of the unconscious or semi-conscious patients. A recovery section is the very best answer to the problem of immediate postanesthetic care. The anesthetist, in addition to his other duties, should be safety adviser to the staff and hospital. 7 references.

F. A. M.

LEEVY, C. M.; STRAZZA, J. A., AND JAFFIN, A. E.: *Fluids in Heart Failure.* J. A. M. A. **131**: 1120–1125 (Aug. 3) 1946.

This article presents a different concept of fluid intake for patients in heart failure. The patients, all in congestive failure, were assigned by rotation to one of three treatment regimes. Drugs, diet, and bed rest were kept as constant as possible. The only variable was the fluid intake.

The diet was acid ash, salt poor. This diet prevents neutralization of injested acidifying diuretics and metabolic acids which mobilize already stored sodium. Since the sodium intake was limited there was no need to restrict fluid intake, and as a result the patients felt more comfortable. Salt depletion was not observed. This was thought to be due to the regulatory mechanism of the kidney and sweat glands which preserves electrolytic balance.

Therapy in congestive failure, besides the above mentioned diet included bed rest, dieuretics, sedatives and a digitalis preparation.

The rationale for forcing fluids is that cardiac failure reflects itself upon the kidneys by impairing their function. Therefore more fluid is needed to eliminate normal waste products. The amount of water should be sufficient to allow excretion of waste products without having the kidneys work at maximum capacity.

There were 36 patients treated on a restricted fluid regime (1200 cc.). Discomfort and thirst was seen even though these patients were clinically edematous. Treatment of their dehydration by increasing the intake gave relief to the patients. This increase in the fluid intake did not interfere with cardiac compensation.

There were 48 patients of the regime "fluids ad lib." These were the most comfortable patients of the entire group. No ill effects were noted from this regime.

There were 38 patients on a forced fluid regime (3,000 cc. and up). Some (18 per cent) were nauseated by this volume of water. There was no evidence of either pulmonary or cerebral edema under this regime. Over 70 per cent of this group felt greatly improved. The highest recorded intake was 7,600 cc. a day.

Decompensated cardiacs are not retarded by forcing fluids, nor does restricting fluids facilitate compensation. It is possible for these decompensated cardiacs to have thirst, become dehydrated, and still have edema, on restricted fluids. Cases are reported in which daily intake of 2500 to 3000 cc. is associated with outputs of 4500 to 6000 cc. The highest output under forced fluids being 10,000 cc. 19 references.

P. C. K.

BERRY, ROBERT L.; CAMPBELL, KENNETH N., ET AL.: *The Use of Tetraethylammonium in Peripheral Vascular Disease and Causalgic States.* Surgery **20**: 525–535. (Oct.) 1946.

"The role of vasoconstriction has been evaluated in the past by means of local nerve, paravertebral, or spinal block. Recently, a new method of producing a blockade of autonomic ganglia by means of parenteral injection of the tetraethylammonium ion has been introduced. . . . Acheson & Moe studied the action of (this drug) . . . and concluded that the response of the nictitating membrane, blood pressure, and heart rate could all be explained by a blockade of autonomic ganglia, both sympathetic and parasympathetic. . . . This interpretation was further confirmed by Acheson & Pereira. Lyon and co-workers demonstrated that the drug could be administered safely to man in doses sufficient to produce an autonomic blockade. . . . After suitable control studies had been made, tetraethylammonium was injected intravenously or intramuscularly in a 10 per cent solution. . . . The intravenous dosage ranged from 100 mg. (1 cc.) to 500 mg. (5 cc.) . . . injected slowly during fifteen to sixty seconds, using significant changes in the volume of the pulse and the general reaction of the patient as a guide to cease or delay further administration of the drug. . . . Intramuscular injections were utilized to prolong the effect of autonomic blockade only in patients under observation in the hospital . . . and were given in doses less than 20 mg. per kg., 1.0 to 1.5 grams, half of the dose administered in each buttock. . . . The intravenous administration produces a metalic taste in fifteen to twenty seconds followed by a sense of numbness and tingling in the extremities which is associated usually with a fall in blood pressure in hypertensive patients and with a rise in heart rate. Sweating, if present, stops, the mouth becomes dry, and there is incomplete dilatation of the pupil with loss of accommodation. . . . There is an increase in skin temperature of the toes and fingers usually within five minutes, which persists after the blood pressure returns to its initial level. . . . Postural hypotension may exist in diminishing intensity for fifteen to sixty minutes. The vasoconstrictor gradient present in the extremities is largely abolished, so that toe and thigh temperatures are equal-