tion of this artificial airway and its use appeared in ANESTHESIOLOGY **18**: 904, 1957.) (Supported by the Research and Development Division of the Surgeon General, Department of the Army, Contract DA-49-007-MD-858.)

Effect of Succinylcholine on Intraocular Pressure in Human Beings. HERMAN SCHWARTZ, M.D., AND ANDREW DEROETTH, JR., M.D., Departments of Anesthesiology and Ophthalmology, Columbia University, College of Physicians and Surgeons, New York, New York.

SINCE 1955, three papers [Am. J. Ophth. **40**: 501, 1955; Am. J. Ophth. **43**: 440, 1957; ANESTHESIOLOGY **18**: 44, 1957] have been published demonstrating a significant rise in intraocular pressure after the intravenous administration of succinylcholine. Since this drug is used to facilitate orotracheal intubation of patients to have ocular surgery and also to keep patients motionless during cataract extractions and corneal transplants we have undertaken a study of the effects of succinylcholine on ocular tensions during clinical situations.

Twenty-four normotensive eyes and 12 eyes in patients with chronic wide-angle glaucoma were studied in a total of 18 subjects. Twelve patients were given barbiturate premedication together with a narcotic and a belladonna drug, 4 were given a narcotic and a belladonna drug, and 2 received only a belladonna derivative one hour before the study was begun. Intraocular pressures were measured with a Schiotz tonometer after ½ per cent tetracaine was instilled for corneal anesthesia (normal tension is 12 to 20 mm. Hg.). The larynx and trachea were topically anesthetized with cocaine, an intratracheal tube inserted and anesthesia induced with intravenous thiopental and nitrous oxide-oxygen. After equilibrium was established, the ocular tensions were again measured and although these were lower than when the subjects were awake, were used as the baseline pressures. Intravenous succinylcholine was then administered either as a single dose (60 to 100 mg.) or as a dilute drip (0.2% to 0.4%) and the tension changes followed.

Of the 24 normotensive eyes studied, 22 had a rise in intraocular tension which returned to baseline levels in 2 to 5 minutes. The average rise was 6.7 mm. Hg and the highest rise was 17.4 mm. Hg. In 2 subjects the intravenous succinylcholine was repeated when the clinical effects of the first dose were gone (7 to 8 minutes later) and only a small rise in tension (2 to 3 mm. Hg) noted. In the 5 patients who were given the succinylcholine as a drip, the rise in ocular pressure remained at the increased levels for longer periods (4 to 5 minutes) than those who were given single injections. However, although the drug was continued, the tensions dropped back to baseline values. Two of these subjects had a cataract extraction performed and the intraocular pressure measured in the opposite eye during the surgery. No significant elevation of tension was recorded even though the succinylcholine drip was administered continuously. Nine other cataracts and 4 corneal transplants (not included in this study) have also been done uneventfully using this technique.

The probable cause of the rise in intraocular pressure after intravenous succinylcholine is contraction or contracture of the extraocular muscles around the globe. Lincoff and his associates have demonstrated contracture in cats [Am. J. Ophth. **40**: 501, 1955; Am. J. Ophth. **43**: 440, 1957]. However, the physiological effect of succinylcholine in humans as measured by complete respiratory paralysis lasted longer than the elevated ocular tensions and the tensions receded even if succinylcholine was given continuously. One can speculate that either: A peak of depolarization effect is reached after which the extraocular muscles become paralyzed permitting the intraocular tension to fall; or contracture occurs and the tension falls as a result of an increased rate of drainage of aqueous humor through the canal of Schlemm to compensate for the squeezing of the extraocular muscles. In the 12 chronic wide angle glaucomatous eyes, no significant rises in tension were recorded so that these did not serve to answer the problem. No narrow angle glaucomatous eyes (which have low tensions during remissions) were studied for fear of initiating an acute exacerbation.

This problem is being pursued further by studying in humans, the changes in tone which occur in normal extraocular muscles following the intravenous administration of succinylcholine. These are detached from the insertion but the origins remain intact leaving normal blood and nerve supplies. In two subjects thus far studied, neither contracture nor contraction of the extraocular muscles has been obtained, probably as a result of too great a depth of thiopental-nitrous oxide anesthesia.

We conclude that succinylcholine can be used in ocular surgery in single doses for tracheal intubation preoperatively (except in patients with acute narrow angle glaucoma in remission) since any rise in ocular tension is dissipated by the time surgery is begun. It may be used with caution throughout an operation as an intravenous drip, if it is begun well before surgery starts and the patient's succinylcholine level is maintained throughout surgery (that is, kept completely paralyzed). It should never be started after actual surgery on the eyes is begun.

Depression of the Myocardium and of Total Body Oxygen Consumption by Fluothane Anesthesia in Man. J. W. SEVERINGHAUS, M.D., AND S. C. CULLEN, M.D., Division of Anesthesiology, State University of Iowa, Iowa City, Iowa.

ARTERIAL hypotension predictably occurs with the administration of (1,1,1 trifluoro-2,2-bromochlorethylene) Fluothane to man and animals. In man, the pulse slows, pupils are small, skin remains warm and dry, with good color, and capillary refill time is normal with pressure as low as 60/45 mm. of mercury. Some have suggested this hypotension is due to ganglionic blockade. The following experiments suggest, however, that the primary circulatory effect of Fluothane is myocardial depression, with no demonstrable component owing to ganglionic blockade in man.

Patients were paralysed with succinylcholine and artificially ventilated with 75 per cent nitrous oxide in an open circuit. Fluothane (1.5 per cent) was added for fifteen minutes, and the change in cardiac output, arterial pressure and central venous pressure determined. The Fluothane was stopped, the autonomic ganglia were blocked with an Arfonad infusion (in 2 patients high spinal sympathetic block was substituted), and the arterial pressure was returned to control levels with an infusion of phenylephrine; after stabilization, the phenylephrine infusion was held at constant rate to provide a constant vascular tone. Following at least forty-five minutes of washout of the previous dose, Fluothane was again given for fifteen minutes in 1.5 per cent concentration, and determinations made of cardiac output and arterial and venous pressure. If the Fluothane depressor effect had been ganglionic blockade in origin, the patients with blocked and stabilized sympathetic responses should have been less depressed by Fluothane. The results showed the contrary, the reductions in cardiac output and arterial pressure being 31 per cent and 21 per cent before blockade, and 42 per cent and 44 per cent in the presence of blockade (7 subjects). Central venous pressure and peripheral resistance rose with Fluothane administration, more in the presence of intact sympathetics. This suggests that myocardial weakening accounts for the major portion of the circulatory depression, and that the sympathetic nervous system tends to compensate partially for circulatory depression due to Fluothane. In a separate group of 6 subjects, oxygen consumption was determined before and during the spontaneous respiration of Fluothane in air, in an open circuit. The average reduction of oxygen consumption corresponding with 1.5 per cent Fluothane appeared to be 15 per cent to 20 per cent.

Cardiac Arrhythmias Produced by Mechanical Stimulation During Hypothermia. KARL L. SIEBECKER, M.D., AND JOHN E. STEINHAUS, M.D., Department of Anesthesiology, University of Wisconsin Medical School, Madison, Wisconsin.

MECHANICAL stimulation of the heart is one of the factors precipitating ventricular fibrillation during hypothermia for cardiac surgery. This factor has been studied under