sociation. It occurred in 12.5 per cent of the cases and comprised 50 per cent of the arrhythmias diagnosed. It was associated with hypoxia, surgical manipulation or deepening of anesthesia. Premature ventricular contractions occurred in 4 per cent of the cases and made up 40 per cent of the recognized arrhythmias. Other arrhythmias noted in the series were auricular premature contractions, auricular fibrillation, A-V block, and bundle branch block. (5) The incidence of arrhythmia was not affected by the belladonna alkaloid used in premedication, sex of the patient, or type of inhalation agent used. (6) Constant electrocardiographic monitoring of patients under anesthesia has proven to be a valuable adjunct to the administration and supervision of clinical anesthesia.

Studies of Narcotic Antagonists as Analgesics. JANE TELFORD, M.D., YOSHIO KUROSU, M.D., AND ARTHUR S. KEATS, M.D. *Division of Anesthesiology, Baylor University College of Medicine, Houston, Texas.* The previous demonstration that nalorphine was a potent analgesic in man led us to the investigation of other narcotic antagonists as analgesics in the search for a potent analgesic without undesirable side effects. Recently we have studied in man two morphinan derivatives reported to antagonize morphine in animals. These are (−) 3-hydroxy-N-(3,3, dimethylallyl)-morphinan hydrobromide (NIH 7446 and (−) 3 hydroxy-N-propargyl morphinan tartrate (NIH 6045). Initially analgesic potency of these drugs was determined. On a milligram basis NIH 7446 was equally, and NIH 6045 was twice as potent as morphine as an analgesic in relieving postoperative pain. However, when given in equivalent analgesic doses to normal subjects, NIH 7446 was only half as potent and NIH 6045 equally as potent as morphine in depressing respiration. To estimate the potency of these drugs as morphine antagonists, anesthetized patients were used. Patients under light thiopental-nitrous oxide-oxygen anesthesia were given morphine or a morphine antagonist intravenously to a total dose averaging 1 mg./kg. Controlled respiration was maintained throughout the operation with succinylcholine by infusion when necessary. After operation, alveolar ventilation and alveolar P_{CO_2} were measured simultaneously. After a suitable control period, one of the following was given intravenously: placebo, nalorphine 2.5 mg., NIH 7446 2.5 mg., or NIH 6045 2.5 mg. Measurements were repeated at 5 and 10 minutes after drug administration. Nalorphine in doses of 1 mg., 2.5 mg., and 5 mg. dramatically antagonized the respiratory depression of morphine (1 mg./kg.). In 3 patients who had received morphine 1 mg./kg., NIH 7446 produced little antagonism. In an additional 3 patients, the respiratory depression of 1 mg./kg. of NIH 7446 was similar in degree to that of morphine and was dramatically antagonized by nalorphine. NIH 6045 in doses of 2.5 mg. antagonized morphine induced respiratory depression in a manner similar to that of nalorphine. However, when NIH 6045 was given to 5 patients in doses of 1 mg./kg. intravenously, it produced only half the respiratory depression of morphine and only one quarter of that anticipated from studies of respiratory depression in normal subjects. When 2.5 mg. of nalorphine was given after NIH 6045, there was no significant change in respiration. Similarly nalorphine in doses of 10 mg./70 kg. depressed the respiration of normal subjects but in doses of 1 mg./kg. intravenously produced little respiratory depression in anesthetized patients. NIH 6045 appears to act like nalorphine, whereas NIH 7446 appears to be a morphine-like drug. The quantitative discrepancy between the respiratory depression in normal subjects following a small dose of NIH 6045 and a large dose in anesthetized patients suggests that the drug may have a dual action which depends on dosage.

Studies on the Effect of Urea on Blood Pressure and Volume. JOHN TENCH, M.D., MANUCHER JAVID, M.D., AND DAVID GILBOE, PH.D. *Departments of Anesthesiology and Neurosurgery, University of Wisconsin Medical School, Madison, Wisconsin.* Analysis of over 200 anesthetic charts of patients who received urea during craniotomy revealed a consistent pattern of blood pressure increase averaging 26 mm. Hg above stabilized blood pressure. This was later followed by a decrease to an average of 20 mm. Hg below stabilized blood pressure when adequate blood replacement was not initiated. Preliminary studies under-