

tonsillectomy. Postoperatively, 10 per cent of patients receiving trimethobenzamide had symptoms of nausea or vomiting as compared with 47 per cent of those receiving placebo, a statistically significant difference. The duration of effect of trimethobenzamide is estimated to be between 3 and 4 hours. Excessive drowsiness was the only side effect noted; however, this was probably not drug related since it was observed more frequently in the placebo patients. The routine use of trimethobenzamide as prophylaxis against postoperative vomiting is warranted in adenotonsillectomies, since the incidence of emesis was significantly reduced and serious side effects were not encountered. (Marcus, P. S., and Ettenberg, M.: *Antiemetic Prophylaxis in Adenotonsillectomies*, J.A.M.A. 189: 695 (Aug. 31) 1964.)

**BLOOD TRANSFUSION** Homologous serum jaundice occurs in 0.3 per cent to 1 per cent of all people receiving blood, involving one of every 200 transfusions. The acceptable uses of blood should be scrutinized, and hospital transfusion practices reviewed by an appropriate committee. In the usual anemic patient, a hemoglobin level of 10 g. per 100 ml. or less justifies transfusion. In a patient receiving a general anesthetic, a safe hemoglobin level is believed to be 10 g. per 100 ml.; below this level a transfusion is not questioned. Hemoglobin of 8 g. per 100 ml. or less justifies transfusion in the usual obstetrical patient. However, because isosensitization producing hemolytic disease of the newborn is always possible, perhaps transfusions should never be given to obstetrical patients who have iron deficiency anemia and hemodilution of pregnancy as their only problem. Rather, reliance should be placed on the recuperative powers, with iron therapy, of the average young woman. (Walz, D. V.: *An Effective Hospital Transfusion Committee*, J.A.M.A. 189: 660 (Aug. 31) 1964.)

**HYPERBARIC OXYGENATION** Observations in nine patients with clinical tetanus revealed active regression of symptoms following hyperbaric oxygen therapy. Progression of the disease was arrested and reversed, and seizures were reduced. The improved mental clarity and cooperation of these patients re-

sulted in better control of respiratory problems and nutritional requirements. The need for tetanus antitoxin and tracheotomy was avoided. The mechanisms by which hyperbaric oxygen exerts its effect are probably suppression of tetanus bacteria by the penetration of high oxygen tensions into areas of anoxia, thereby preventing toxin formation; direct oxidation of the toxin; and a beneficial effect on the diseased nerve cells by high oxygen partial pressure. No apparent ill effects were noted during therapy, although treatments were interrupted at 15 to 30 minute intervals as prophylaxis against oxygen toxicity. (Pascale, L. R., and others: *Treatment of Tetanus by Hyperbaric Oxygenation*, J.A.M.A. 189: 408 (Aug. 10) 1964.)

**OXYTOCIC DRUGS** A group of 1,459 parturient women were the subjects of a controlled double-blind study assessing the effects of oxytocin, methylergonovine maleate and a placebo upon the fourth stage of labor. Although patients in the placebo group had a higher incidence of hemorrhage and more often required additional treatment in the form of an oxytocic agent, 88 per cent of this group had no difficulty. Methylergonovine maleate was the agent most effective in preventing postpartum hemorrhage. Postpartum blood pressure rises occurred in all three groups, most frequently following methylergonovine maleate and least often following placebo. In patients with toxemia of pregnancy, methylergonovine maleate was associated with a high incidence of severe pressor responses. In patients with toxemia, if an oxytocic agent is required, oxytocin is the drug of choice. (Howard, W. F., and others: *Oxytocic Drugs in Fourth Stage of Labor*, J.A.M.A. 189: 411 (Aug. 10) 1964.)

**CARDIOVERSION** Patients were prepared with quinidine sulfate for a minimum of 18 hours, and digitalis was temporarily withheld until reversion was completed. Patients were anesthetized with intravenous thiopental sodium. They received the precordial electroshock after anesthesia was established, and awakened when normal rhythm had occurred, anesthesia time being three to seven minutes. This short-duration anesthesia proved to be no

problem, even in patients who had recent cardiac surgery, and reversion was not refused to any suitable candidate on the basis of anesthetic risk. The safety of the procedure under the proper circumstances justifies an attempt at the correction of ectopic cardiac arrhythmias in all patients who will benefit from normal sinus rhythm. (Miller, H. S.: *Synchronized Precordial Electroshock for Control of Cardiac Arrhythmias*, J.A.M.A. 189: 549 (Aug. 17) 1964.)

**CARDIOVERSION** Studies have convincingly demonstrated the safety of direct current electroshock; nevertheless, sequential changes in the electrocardiogram invite a modicum of caution in the application of this promising means of terminating cardiac arrhythmias. One patient with chronic atrial fibrillation has been studied in whom, although direct current electroshock successfully restored regular sinus-dominated rhythm, the conversion coincided with the appearance of a striking elevation of the ST segment of the electrocardiogram. Similar ST segment changes endure for many hours or days following acute myocardial infarction, and a variant form of angina pectoris may be associated with strikingly similar ST elevation which disappears with relief of pain. Whether, in the case of cardioversion, such deviations reflect alterations in the distribution of myocardial cellular electrolyte, protein, enzymes, or actual necrosis is unknown. The question as to whether repeated shocks of this nature could be additive and result in permanent injury must await further experience. (Sussman, R. M., and others: *Myocardial Changes After Direct Current Electroshock*, J.A.M.A. 189: 739 (Sept. 7) 1964.)

**CEREBRAL BLOOD FLOW** Cerebral blood flow increased linearly with arterial carbon dioxide tension over a range of tensions from 20 to 60 mm. of mercury during halothane anesthesia in human subjects. Mean cerebral blood flow and cerebral vascular resistance at arterial carbon dioxide tension of

40 mm. of mercury were 49.7 ml./100 g./minute and 1.0 mm. of mercury/ml./100 g./minute, respectively, in anesthetized subjects. These values are different from corresponding values in conscious subjects which are 44.4 ml./100 mg./minute and 1.9 mm. of mercury/ml./100 mg./minute. The shape of the cerebral blood flow-arterial carbon dioxide tension response curve was altered because of the cerebral vasodilatation caused by halothane. (Alexander, S. C., and others: *Cerebrovascular Response to  $P_{aCO_2}$  During Halothane Anesthesia in Man*, J. Appl. Physiol. 19: 561 (July) 1964.)

**PULMONARY BLOOD FLOW** When excised dog lungs which had been almost maximally inflated with air were perfused, pulmonary artery pressure was less than alveolar pressure with flows up to 50 ml./minute. Pulmonary artery pressure exceeded alveolar pressure at all flows when lungs were inflated to comparable volumes with water. The presence of an air-liquid interface facilitates perfusion in lungs due to lowering of pericapillary pressure caused by effects of surface tension of the fluid film which lines alveoli. (Bruderman, I., and others: *Effect of Surface Tension on Circulation in the Excised Lungs of Dogs*, J. Appl. Physiol. 19: 707 (July) 1964.)

**THORACIC MECHANICS** A plethysmograph was used to measure relative contribution of chest wall expansion and diaphragmatic descent to inspiration. In 15 normal subjects, one-third of the tidal volume was effected by diaphragmatic descent and two-thirds by rib cage expansion. This partitioning of tidal volume was not altered by changes in breathing pattern or inhalation of various special gas mixtures. Elastic recoil alone was sufficient to move the rib cage at low tidal volumes, so that no work was required for chest expansion until tidal volumes exceeded one liter. (Bergofsky, E. H.: *Relative Contributions of the Rib Cage and the Diaphragm to Ventilation in Man*, J. Appl. Physiol. 19: 698 (July) 1964.)