of some patients to return to more normal exercise tolerance postoperation. Determination of oxygen ventilatory equivalent is thought to provide objective method of classing severity of mitral stenosis and a means of assessing results of surgical therapy. (*MacIntosh, D. J., and others: Some Aspects of Disordered Pulmonary Function in Mitral Stenosis, Ann. Int. Med. 49: 1294 (Dec.) 1958.*)

AIRWAY RESISTANCE The effect of added airway resistance on ventilation and respiratory muscular activity was studied in decerebrate cats before and during anesthesia. Inspiratory air flow resistance initially reduces the tidal volume and frequency of respiration. This reduction in tidal volume is aggravated by pentobarbital, nitrous oxide, or ether. If the flow resistance persists, there is a gradual recovery in the volume of ventilation, but even in the unanesthetized state this is not complete at the end of five minutes. Ventilation is reduced with increased airway resistance in spite of augmented muscular activity. The reduction of ventilation is proportional to the magnitude of the resistance, and the impairment becomes progressively worse with deeper anesthesia. (*Fink, B. R., and others: Effect of Air Flow Resistance on Ventilation and Respiratory Muscle Activity, J.A.M.A. 168: 2245 (Dec. 27) 1958.*)

RESPIRATORY CONTROL The factors involved in the control of respiration during exercise were studied in normal man. Arterial pO_2 does not decline significantly during heavy exercise and thus, cannot be responsible for the hyperpnea. There is no significant increase in arterial pCO_2. Likewise, changes in arterial pH do not bear a predictable relationship to changes in ventilation during exercise and recovery. Changes in mixed venous pCO_2 and in "central" blood volume may be factors involved in the production of hyperpnea during heavy muscular exercise, but these possibilities cannot be designated with assurance as the cause of the phenomenon at the present time. (*Mitchell, J. H., Sproule, B. J., and Chapman, C. B.: Factors Influencing Respiration During Heavy Exercise, J. Clin. Invest. 37: 1693 (Dec.) 1958.*)

RESPIRATORY ACIDOSIS In the rat, the hypochloremia which develops during exposure to carbon dioxide in air is the result of a loss of chloride in excess of sodium in the urine. This increased loss of chloride is associated with an increased excretion of ammonia, potassium and phosphorus. This occurred in animals maintained on a sodium-free diet as well as those ingesting and excreting liberal amounts of sodium. This suggests that expansion and dilution of extracellular fluid with reabsorbed sodium bicarbonate are not responsible for the increased urinary excretion of chloride. It is suggested that an increase in pCO_2 may induce the observed renal losses of chloride by direct interference with the tubular reabsorption of the chloride ion. (*Levitin, H., Branscome, W., and Epstein, F. H.: Pathogenesis of Hypochloremia in Respiratory Acidosis, J. Clin. Invest. 37: 1667 (Dec.) 1958.*)

RESPIRATION IN OBESITY A case history of a 42 year old male weighing 364 pounds is presented. He was admitted in cardio-respiratory failure due to alveolar hypoventilation, explained on the basis of: (a) extra work needed to maintain alveolar ventilation by moving fat-ladened abdomen and thorax; (b) the diaphragm pushed up by masses of fat in the abdomen; and (c) mediastinal fat deposits encroaching on the lungs. A 47 per cent arterial oxygen saturation was found. (*Berlyne, G. M., and Manc, M. B.: Cardiorespiratory Syndrome of Extreme Obesity, Lancet 2: 939 (Nov. 1) 1958.*)

ARTIFICIAL VENTILATION Two methods of ventilation were compared in alternate cases in a series of 21 young male patients with minimal lung disease—the first group being ventilated automatically with positive-negative pressure as delivered by the Jefferson ventilator and the second group was ventilated manually with intermittent positive pressure. The preoperative pulmonary elastic recoil or compliances of the two groups were not significantly different. After lung resection, the compliance generally was lower in both groups. The magnitude of fall, however, was greater in the group ventilated with automatic positive-negative respiration. (*Glied-*

man, M. L., and others: Effect of Manual versus Automatic Ventilation on Elastic Recoil of the Lung, Ann. Surg. 148: 899 (Dec.) 1958.)

RESUSCITATION A neuromuscular stimulator is described which delivers an 8,000 volt 25 microampere pulse of 300 microseconds duration. The skin electrode stimulates the phrenic nerves either where they traverse the thoracic cavity or on the diaphragm. Although ventilation was not measured, rabbits made apneic with thiopental were sustained. With prolonged resuscitation, respiration gradually diminished and finally disappeared. EKG was not monitored, but maximal stimulating intensities often caused a decreased heart rate. No nerve or skin damage was noted in chronic experiments. Its possible application to asphyxia neonatorum was considered. *(Hon, E. H., and Hulme, G. W.: An Electronic Resuscitator for Possible Use in Asphyxia Neonatorum, Yale J. Biol. & Med. 31: 57 (Nov.) 1958.)*

CYCLOPROPANE Controlled ventilation during cyclopropane anesthesia by either manual or mechanical means usually produces hyperventilation and respiratory alkalosis. Oxygenation of arterial blood may be less than 100 per cent even in the presence of hyperventilation if there is uneven ventilation and perfusion of the lungs. Cardiac arrhythmias perceptible by palpation rarely occur when cyclopropane is administered with ventilatory control. Downward displacement of the cardiac pacemaker as seen on the electrocardiogram may represent myocardial depression by cyclopropane superimposed on pre-existing myocardial disease or drug therapy. *(Wester, M. R., and others: Manual and Mechanical Control of Ventilation During Cyclopropane Anesthesia, J.A.M.A. 168: 2249 (Dec. 27) 1958.)*

NEW SOPORIFIC Trimethoxyhenzoylglycine-diethylamide (Trimeglamide) has been demonstrated to produce sleep without deep hypnosis or anesthesia as usually seen with commonly used barbiturates, chloral hydrate, etc. In animals there has been no evidence of ataxia, excessive central nervous system depression, cardiovascular or respiratory depression. The effects are said to be similar to physiological sleep. Dosages of 50 mg./kg. in dogs and cats produced soporific effects lasting 2–6 hours. At larger doses (100 mg./kg.) soporific effect was prolonged in cats and in some dogs evidence of stimulation became apparent at 500 mg./kg. dosages. In man dosage ranges 500–1500 mg. caused sedation, drowziness and side effects in about half of 200 patients. Electroencephalographic recordings showed no drug induced spindling or disturbance of resting alpha rhythm. *(Cronheim, G., Courzis, J. T., and Tockes, I. M.: New Type Sedative and Soporific Drug, Science 128: 1570 (Dec. 19) 1958.)*

ANALEPTIC Micoren, a new analeptic drug, was administered to 436 patients subjected to various types of general anesthesia. The drug was found efficacious in lightening the depth of anesthesia and, postoperatively, in decreasing the depressant effects of morphine. *(Cattaneo, A. D.: Esperienze Cliniche con il Micoren, Um Nuovo Farmaco Analettico, J. Internat. Coll. Surgeons 31: 87 (Jan.) 1959.)*

SYNERGISM IN PAIN RELIEF True synergism in the relief of pathological pain in man is claimed by Macris to be demonstrated for the first time. Papaverine (which has no analgesic power) and morphine were the drugs first used to demonstrate this synergism. *(Editorial: Synergism in Pain Relief, Canad. M.A.J. 79: 848 (Nov. 15) 1958.)*

OXYGEN CONSUMPTION Long term administration of the carbonic anhydrase inhibitor azetazolamide (Diamox) has been shown to lower oxygen consumption in rats. Antithyroid activity is demonstrated by increase in thyroid weight and decrease in blood protein bound iodine in the treated animals. This lowering of metabolism may explain why certain patients with chronic respiratory insufficiency are improved by acetazolamide without demonstrable increase in ventilation. *(Tenney, S. M., and Schetter, A. B.: Decrease in Oxygen Consumption Associated with Prolonged Administration of the Carbonic Anhydrase Inhibitor, Acetazolamide (Diamox), Am. J. M. Sc. 237: 23 (Jan.) 1959.)*