OXYGEN TOXICITY Central nervous system symptoms of oxygen toxicity at four atmospheres pressure in mice are controlled by THAM. No protection by this dose of THAM was noted at six atmospheres of oxygen. Such protection can be reversed by the addition of potassium and sodium chloride. A mixture of THAM and potassium chloride is lethal to mice. (*Gottlieb, S. F., and Jagodzinski, R. V.: Role of THAM in Protecting Mice Against Convulsive Episodes Caused by Exposure to Oxygen Under High Pressure, Proc. Soc. Exp. Biol. Med. 112: 427 (Feb.) 1963.*)

PREDNISONE IN EMPHYSEMA Ten patients were studied in a double-blind controlled manner to determine the effect of prednisone on air flow obstruction. Prednisone did not produce a statistically significant increase in timed vital capacity. Two of the ten patients did show improvement, however. (*Beerel, F., Jick, H., and Tyler, J. M.: A. Controlled Study of the Effect of Prednisone on Air-Flow Obstruction in Severe Pulmonary Emphysema, New Engl. J. Med. 268: 226 (Jan. 31) 1963.*)

DIFFUSING CAPACITY Increases in tidal volume produced significant increases in steady state diffusing capacity in anesthetized, paralyzed, artificially ventilated dogs. Increase in respiratory rate at constant tidal volume decreased diffusing capacity. These findings are interpreted as indicating more uniform distribution of inspired gas and a larger alveolar surface area available for diffusion at large tidal volumes. (*Kilburn, K. H., and others: Effects of Altering Ventilation on Steady State Diffusing Capacity for Carbon Monoxide, J. Appl. Physiol. 18: 89 (Jan.) 1963.*)

NEONATE LUNG COMPLIANCE A marked increase in survival of neonates suffering from tetanus occurred when the inflating pressures in the anesthetized, paralyzed infant were regulated by P_{CO_2}. No deaths could be attributed to overwhelming toxicity, but all were due to technical difficulties or intercurrent infection. At lower inspiratory pressures, pulmonary compliance fell and P_{CO_2} rose. With time static compliance fell and more pressure was required for ventilation. The average compliance was 2.5 ml./cm. water with a range of 4 to 1.7 ml./cm. Necessary initial inspiratory pressures were from 11 to 15 cm. water and rose in many cases to 15 to 18 cm. water in a few days, in one case rising to 30 cm. water in seven days. Tracheotomy was necessary and special care in removing secretions was of paramount importance. Provided efficient ventilation is maintained, intoxication of severe tetanus seems reversible. (*Smythe, P. M.: Studies on Neonatal Tetanus, and on Pulmonary Compliance of the Totally Relaxed Infant, Brit. Med. J. 1: 565 (Mar. 2) 1963.*)

CO_2 EQUILIBRATION Factors determining whether carbon dioxide equilibration will occur in the lung are the rate of the reactions leading to carbon dioxide production and the time the bicarbonate ions spend in contact with the gas exchange surface. Using a body plethysmograph as a manometer to determine the rate and magnitude of carbon dioxide evolution, it was found that the bicarbonate ions spent an average of 2.2 seconds in contact with the gas exchange surfaces in dogs, a much longer time than the erythrocyte transit time through the pulmonary capillaries. This prolongation of bicarbonate transit time is caused by the presence of a large peri-capillary dilution and reaction space for the bicarbonate-carbon dioxide system, and it is suspected that this prolongation plays a role in the completion of carbon dioxide equilibration in the lungs. (*Feisal, K. A., Sackner, M. A., and DuBois, A. B.: Comparison Between the Time Available and the Time Required for CO_2 Equilibration in the Lung, J. Clin. Invest. 42: 24 (Jan.) 1963.*)

RESPIRATORY HUMIDIFIERS Condenser humidifiers consist of a number of flat wire gauzes, stacked in series in poor thermal contact, through which the patient respires. These humidifiers can produce a substantial degree of humidification of inspired air in patients with tracheotomies or endotracheal tubes by returning to the patient water vapor breathed out in expiration. They perform best when the fresh air is cold and damp rather than hot and dry. If the inspired room air is fairly moist and cool such an apparatus can completely substitute for the humidifying properties of the upper respiratory tract. (*Mapleson,*

W. W., Morgan, J. G., and Hillard, E. K.: Assessment of Condenser-Humidifiers with Special Reference to a Multiple-Gauze Model, Brit. Med. J. 1: 300 (Feb. 2) 1963.)

TRACHEOTOMY The performance of tracheotomy in infants may be difficult. General anesthesia is always advisable unless there is an obviously impassable obstruction. The trachea should be intubated as the first step, and as soon as possible thereafter a soft rubber catheter should be passed through the tracheal tube to remove secretions. Apnea may come on immediately after the trachea is opened, and artificial respiration may occasionally be needed at this stage. Mucolytic aerosols may be of help. An anesthetist should be present at the time of closure of the tracheostomy. A common finding at this time is some degree of obstruction of the trachea with inspiratory stridor, caused by partial collapse of the anterior wall around the opening and some sucking in of the neck tissues. The tracheostomy opening almost always closes spontaneously and only rarely needs suturing. *(Fennell, G.: Management of Tracheotomy in Infants, Lancet 2: 808 (Oct. 20) 1962.)*

RESPIRATORY UNIT In a respiratory insufficiency unit, it is essential that the ward be open, that the patients' heads be placed towards the center of the ward and that all the essential nursing services be grouped compactly near the patients' heads. This may be done by means of a retractable overhead beam containing electrical outlets, suction, humidifying apparatus, oxygen outlet, respirator supports, compressed air, nurse call system, radio or television, and containers for instruments, dressings and catheters. *(Hercus, V.: Planning a Respiratory Unit, Brit. Med. J. 2: 1605 (Dec. 15) 1962.)*

GUANETHIDINE In hypertensive and normotensive males, the effects of intravenously and orally administered guanethidine were observed on the systemic and splanchnic circulations, the sympathetic nervous system and vascular catecholamine stores. An immediate transient pressor effect, presumably due to release of stored catecholamines, was followed by a hypotensive effect in hypertensive patients only. This depressor phase was associated with a decrease in cardiac output in compensated cardiacs and an increased output in patients with decompensated hearts, as well as a decrease in the total peripheral resistance in all patients. At the same time there was increased splanchnic resistance and a fall in estimated hepatic-portal blood flow. Sympathetic nervous system activity, as tested by respiratory-vasomotor reflexes, was incompletely inhibited in both acute and chronically medicated patients. Further, catecholamine stores were not severely depleted in either group as evidenced by active responses to both tyramine and ephedrine. Catecholamine depletion is not important in the hypotensive action of guanethidine in man and in addition to a peripheral block of nerve terminals proximal to the site of norepinephrine release, there may be a direct depression of myocardium and vascular smooth muscle. *(Cohn, J. N., Liptak, T. E., and Fries, E. D.: Hemodynamic Effects of Guanethidine in Man, Circ. Res. 12: 298 (Mar.) 1963.)*

GUANETHIDINE Guanethidine (10 mg./kg.) given intravenously to dogs abolished the chronotropic and inotropic responses to supramaximal, post-ganglionic cardioaccelerator nerve stimulation within 30 minutes. The block persisted three hours before reduction of norepinephrine in atrial tissue was evident. Subsequent infusion of norepinephrine failed to reverse the block. Following reserpine (2 mg./kg.) intravenously, adrenergic blockade did not occur for five hours even though atrial norepinephrine fell from control levels. It was not until the level had fallen below 0.3 μg./g. that the block developed which also was unresponsive to infused norepinephrine and dopamine. Guanethidine blockade is independent of depletion of stored adrenergic transmitter. Reserpine blockade, on the other hand, probably depends on depletion of tissue norepinephrine. *(Gaffney, T. E., Chidsey, C. A., and Braunwald, E.: Study of the Relationship Between the Neurotransmitter Store and Adrenergic Block Induced by Reserpine and Guanethidine, Circ. Res. 12: 264 (Mar.) 1963.)*