EXPIRATORY FLOW A remarkable series of papers since 1958 by Fry, Hyatt, and others, has established that over most of the vital capacity range, maximum expiratory flow rates (MEFR) are ordinarily limited not by expiratory effort but by an effort-independent pulmonary mechanism involving dynamic compression of airways. Analysis of events peripheral to the region of airway compression shows that MEFR is related to lung recoil force and to airway conductance. This approach affords a useful understanding of the ventilatory limitations and the sometimes puzzling spirometric, bronchographic and other findings in pulmonary disease. (*Mead, J., and others: Significance of the Relationship Between Lung Recoil and Maximum Expiratory Flow, J. Appl. Physiol. 22: 95 (Jan.) 1967.*)

ASTHMA In a study of asthmatic children in remission, mean forced expiratory volume was found to be significantly lower than in a control group of normal children. Physiologic dead space was significantly larger in the asthmatic group. During exercise, a significant metabolic acidosis developed in one third of the asthmatic children. (*Beaudry, P. H., and others: Respiratory Gas Exchange at Rest and During Exercise in Normal and Asthmatic Children, Amer. Rev. Resp. Dis. 95: 248 (Feb.) 1967.*)

FAT EMBOLISM The syndrome of systemic fat embolism has been recognized for many years. The principle underlying etiology is truma, especially fracture of long bones. There are, however, other much less common predisposing conditions in which the associated fat embolism is usually not recognized. A partial list of these would include severe burns, acute fatty liver of the alcoholic, extracorporeal circulation, corticosteroid therapy, sickle cell and sickle-C crises with bone marrow infarction, following oil lymphography, and pancreatitis. To these can now be added systemic fat embolism following acute primary osteomyelitis. (*Broder, G., and Ruzumna, L.: Systemic Fat Embolism following Acute Primary Osteomyelitis, J.A.M.A. 199: 1004 (March) 1967.*)

RESPIRATORY DISTRESS SYNDROME A full-term newborn baby was judged to have an Apgar 7 rating but failed to exhibit adequate pulmonary ventilation. An endotracheal tube was passed and IPPB begun which produced crepitant but distant breath sounds over both lung fields. Following failure to respond in 15 minutes, subcutaneous emphysema at the root of the neck was noticed and a diagnosis of bilateral pneumothorax was made and confirmed by roentgen-ray. Needle decompression and further resuscitative efforts were to no avail. Injury to the pulmonary parenchyma accompanying vigorous respiratory efforts after the head was delivered was thought to have initiated the process which resulted in air dissection along the mediastinum to the neck. Bilateral pneumothorax should be considered in all cases of respiratory distress syndrome where the findings indicate dullness and distant breath sounds over both lung fields. Needle aspiration is said to be adequate for less than 25 per cent collapse but closed chest drainage is advised for collapse of more than this amount. (*Clinical Anesthesia Conference: Bilateral Pneumothorax in the Newborn, New York J. Med. 67: 274 (Jan.) 1967.*)

CARBON DIOXIDE DISTRIBUTION An electrical analogue was constructed which permitted arterial and venous CO_2 tensions and quantity of readily available CO_2 in body stores to be continuously recorded. Constancy of venous-arterial P_{CO_2} difference under steady-state conditions and some of the reasons for this constancy were demonstrated. Time necessary to develop a new steady arterial P_{CO_2} following step changes in ventilation was also studied. The time was significantly greater following a decrease in ventilation than for a comparable increase, and at least 20 minutes was necessary following changes in ventilation before steady-state conditions were once more attained. (*Whelpton, D., and Thornton, J. A.: An Electronic Analogue of Carbon Dioxide Distribution in the Body, J. Appl. Physiol. 22: 193 (Jan.) 1967.*)