

ASTHMA Arterial oxygen tension in nine asthmatic patients was found to be reduced due to ventilation-perfusion imbalance. The magnitude of the decrease in oxygen tension depended on the severity of the airway obstruction. Bronchodilation with orciprenaline, a drug with little cardiovascular effect, was followed by an increase in arterial oxygen tension. (Meisner, P.: *Pulmonary Function in Bronchial Asthma*, *Brit. Med. J.* 1: 470 (Feb.) 1968.)

ISOPROTERENOL IN ASTHMA Twenty patients in status asthmaticus were treated with isoproterenol aerosol. Airway resistance was reduced and dyspnea was relieved but the hypoxemia often became worse. By intensifying the ventilation-perfusion disturbances in the lung, isoproterenol may worsen gas exchange, and it should be used with caution in patients with status asthmaticus. (Palmer, K. N. V., and Diamant, M. L.: *Effect of Aerosol Isoprenaline on Blood-Gas Tensions in Severe Bronchial Asthma*, *Lancet* 2: 1232 (Dec.) 1967.)

PULMONARY PATHOGENS AND SURFACTANT The effects of various pure strains of bacteria on surface tension of reconstituted rabbit surfactant were determined. Bacteria found to reduce surface tension were *P. aeruginosa*, *A. aerogenes*, *Proteus species*, *K. pneumoniae* and, to a lesser extent, *D. pneumoniae*. Gram-positive bacteria, including *S. aureus*, *S. epidermidis*, *S. pyogenes* (Lancefield group A), *S. viridans* and *S. fecalis*, did not appear to alter the surface tension of the pulmonary surfactant solutions. The data invite speculation as to what extent chronic respiratory infections may be implicated in the histogenesis of chronic respiratory diseases such as emphysema. (Rose, M., and Lindbreg, D. A. B.: *Effect of Pulmonary Pathogens on Surfactant*, *Dis. Chest* 53: 541 (May) 1968.)

ALVEOLAR VOLUME CHANGES Microholes were made in walls of individual alveoli by puncturing the lung surface with ultrasmall needles. Observation of these microholes permitted inferences as to the behavior of individual alveoli during dynamic and

static changes in lung volume. Conclusions of this extremely ingenious study were that alveoli and alveolar ducts change size proportionally with lung volume over the range RV to TLC. When lung volume was altered, changes in diameter of the microholes were proportional to the cube root of the incremental volume change. Alveolar shape did not change significantly during lung inflation and deflation, and alveolar surface area changed as predicted between minimal lung volume and TLC. (Kuno, D., and Staub, N. C.: *Acute Mechanical Effects of Lung Volume Changes on Artificial Microholes in Alveolar Walls*, *J. Appl. Physiol.* 24: 83 (Jan.) 1968.)

PULMONARY EFFECTS OF BED REST Alveolar-arterial oxygen tension difference ($AaDO_2$) was measured in a group of healthy young volunteers before and after ten days of absolute bed rest. $AaDO_2$ increased in every instance. Mean $AaDO_2$ was 9 mm Hg before the study and 19 mm Hg after the ten-day period of recumbency. Mean arterial oxygen tension fell from 103 mm Hg to 94 mm Hg in these healthy individuals. The mechanism of this change was not clear but may be related to analogous increases in $AaDO_2$ previously observed during anesthesia. (Cardus, D.: *Alveolar-Arterial Oxygen Tension Difference After 10 Days Recumbency in Man*, *J. Appl. Physiol.* 23: 934 (Dec.) 1967.)

ALTITUDE AND VENTILATION Respiratory function of young men known to exercise regularly and others known not to exercise was studied at sea level and at 4,300 meters for 28 days. In this study, physical conditioning seemed beneficial since it resulted in an additional increase in maximum breathing capacity during exposure to high altitude. All groups showed significantly increased maximum breathing capacities at 4,300 meters, suggesting a decrease in work of breathing of rarefied air. At altitude, maximal breath-holding times for all groups were significantly decreased, but were decreased less for exercisers. (Consolazio, C. F., and others: *Respiratory Function in Normal Young Adults at Sea Level and 4,300 Meters*, *Milit. Med.* 133: 96 (Feb.) 1968.)