Literature Briefs

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Briefs were submitted by Drs. John Adriani, C. M. Ballinger, Norman Bergman, Peter Bosomworth, Gaylord Buchanan, M. T. Clarke, Deryck Duncalf, Martin Helrich, R. E. Ponath, W. B. Rabenn, Alan Randall, Wallace Ring, H. S. Rottenstein, and P. H. Sechzer. Briefs appearing elsewhere in this issue are a part of this column.

The "Briefs" of Russian Literature were taken from Excerpta Medica's *"Abstracts of Soviet Medicine," which is supplied through the Public Health Service of the National Institutes of Health.*

AIRWAY OBSTRUCTION About one-half of patients with severe obstructive pulmonary emphysema are not benefitted by intermittent positive pressure breathing (IPPB). Many of these patients cannot tolerate such respiratory "assistance." In 5 normal patients with an external obstruction to respiration which simulated that found in emphysema, when IPPB was used, flow rate increased only during inspiration and expiration became greatly prolonged. Expiratory flow resistance was markedly increased owing to very high end-inspiratory alveolar pressure because of increasing machine-made pressure and premature expiratory effort during insufflation. The resulting great transpulmonary pressure, much in excess of the maximal effective pressure, caused airway closure, "trapping," and greatly increased work of expiration. Though more potential energy was built up on inspiration, it only sufficed to accomplish about one half as much of the expiratory work. Minute ventilation increased somewhat, but effective pulmonary aeration was not improved due to the greater gas exchange needed to compensate for the increased expiratory work and the increased physiologic dead space. Low IPPB pressures (7 cm. of water) decreased the work of breathing 15 per cent. High pressures (25 cm. of water) increased the work of breathing 250 per cent and caused marked alveolar hypoventilation and increased functional residual capacity and physiologic dead space greatly. Peak machine flow was insufficient for passive inspiration. These latter findings suggest why some patients cannot tolerate IPPB. *(Jones, R. H., MacNamara, J., and Gaensler, E. A.: Effects of Intermittent Positive Pressure Breathing in Simulated Pulmonary Emphysema, Amer. Rev. Resp. Dis. 82: 164 (Aug.) 1960.)*

EMPHYSEMA Normal and emphysematous lungs were fixed in inflation with formalin vapor, dried and sectioned. In the emphysematous lungs it was possible to show patent bronchioles and alveolar ducts communicating with emphysematous areas. Emphysema is considered to be a disease of alveolar septal tissue rather than the result of mechanical trauma caused by bronchiolar obliteration and air trapping. *(Pratt, P. C.: Correlation of Post Mortem Functions and Structure in Normal and Emphysematous Lungs, Amer. Rev. Resp. Dis. 83: 419 (Mar.) 1961.)*

EMPHYSEMA In emphysematous lungs the elastic fibers are deranged, the bundles about the alveolar ducts and the alveoli being fragmented, frayed or absent. Where broken, the retracted ends are often coiled. Areas of such elastic tissue damage are associated with deformities of the airways and alveoli consisting of overdistended alveolar ducts, effacement of alveoli and fenestration of varying degree up to complete loss of alveolar septa, which all together produce the characteristic large air spaces of emphysema. At the sites of anthracotic pigment deposition in mild emphysema, elastic fibers are seen to be deteriorated or lost, this change occurring most often at the junction of the respiratory bronchioles and the alveolar ducts. *(Wright, R. R.: Pathologic Changes in Elastic Tissue and Bronchi in Chronic Pulmonary Emphysema, Amer. Rev. Resp. Dis. 83: 424 (Mar.) 1961.)*