

DISCUSSIONS OF PAPERS ON RESPIRATORY SYSTEM AND AIRBORNE INFECTION

Chairman: W. BARRY WOOD, JR.

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DISCUSSION OF: "STRUCTURE AND FUNCTION OF RESPIRATORY TRACT IN RELATION TO INFECTION," GEORGE W. WRIGHT

Discussant: Walsh McDermott

Dr. McDermott designated a few of the factors that play a role in control of infection, including: (i) antibody and macrophage activities, (ii) antagonistic surface components such as lysozyme, (iii) antimicrobial tissue factors such as spermine oxidase, and (iv) structure and function of an organ system. These factors, but in particular the morphological mechanisms, may influence location and type of lesion and influence the development of infection into disease. He also suggested that the effects of these factors upon differing microorganisms may vary. For example, it is probable that microorganisms such as pneumococci, streptococci, and staphylococci produce infection only when the factors controlling infection are interfered with, illustrated by the importance of edematous fluid in the respiratory system in predisposing an animal to infection by the pneumococcus. These bacteria, furthermore, are commonly in residence in the upper respiratory tract in the absence of disease but may induce disease when the factors controlling infection are impaired. Other microorganisms, on the other hand, such as brucellae, Q fever, and tubercle bacilli, probably induce infection and disease irrespective of whether or not the factors ordinarily controlling infection are altered. In other words, they are able to induce infection in the unaltered normal animal.

Preoccupation with the airborne route of infection must not blind us to the importance of alteration of host resistance in determining the susceptibility of an animal to infection by microorganisms such as streptococci, pneumococci, and staphylococci. It is possible that the capacity of a microorganism to thrive in a macrophage is an important determinant of its capability as an infectious agent in the normal animal.

Another point refers to the possible influence of site of entry of a microorganism upon the severity and character of the disease produced.

Dr. Tigertt inquired as to the mechanism by which particulate substances travel from pulmonary alveoli to the lymphatics, in view of the fact that the alveoli are devoid of lymphatic channels (2) as confirmed by electron microscopy. Dr. Wood suggested that particles are transferred by macrophages, and Dr. Nelson indicated that Policard, Collet, and Pregermain (3) has shown that the macrophage concentration in the lung is highest at the terminal bronchiole and respiratory bronchiole. Dr. Wright asked whether or not the particle might not penetrate the alveolar cell and be passively transferred to the lymphatic channels. Professor Hatch described a mechanism of lung clearance as envisioned by Gross and Westrick (1), which suggests that the movement of the lung during ventilation tends to extrude material from alveoli toward the respiratory bronchiole.

The importance of the respiratory bronchiole in certain pathological responses of the lung was indicated by Dr. Wright, who described some experiments in his laboratory showing that inhalation of NO₂ results in fluid accumulation in alveoli followed by inflammation at the respiratory bronchiole and polypoid formation on the bronchiole epithelium, succeeded by healing.

Dr. Nelson inquired as to whether or not the mucous membrane of the respiratory tract was penetrable by microorganisms; illustrations of this were suggested by *Haemophilus influenzae* and experimental group C streptococcal infection.

In conclusion, Professor Hatch, in contrast to formerly held opinions, mentioned a current view suggesting that phagocytosis of particles by macrophages may interfere with the passage of the particles into tissues.

DISCUSSION OF: "MUCOCILIARY FUNCTIONS AS A PROTECTIVE MECHANISM IN UPPER RESPIRATORY TRACT," FREDERIK B. BANG

Discussant: John H. Dingle

Dr. Dingle commended the demonstration of the function of the mucociliary mechanism but found it difficult either to relate the results to infections of the upper respiratory tract or to

understand the effects of heat and humidity on the tracheal mucosa. He was puzzled by the effect of the small dose of virus in the model *in vitro* and expressed reservations concerning the applicability of the model to infections in the intact animal.

Dr. Bang agreed that caution must be exercised in interpreting the results of the experiments *in vitro*, but pointed out that the use of the simplified organ culture model had at least yielded preliminary information as to the manner in which the mucociliary blanket functions in the upper respiratory tract.

DISCUSSION OF: "DISTRIBUTION AND DEPOSITION OF INHALED PARTICLES IN RESPIRATORY TRACT," THEODORE F. HATCH

Discussant: Richard L. Riley

Dr. Riley indicated that the aerodynamic principles of ventilation and settling velocity are equally applicable to the events occurring in the respiratory tract and in the air of a room. Furthermore, he suggested that settling velocity might be a better determinant than particle size because settling velocity is more directly related to retention in the respiratory tract. Furthermore, he emphasized that the portal of entry of a

microorganism cannot be equated with the site at which lesions appear.

Considerable discussion ensued regarding the differences, if any, of particles residing in dust as contrasted with droplet nuclei. Particular interest was demonstrated in infections (Q fever, brucellosis, mycoses), probably airborne, in which dust rather than droplet nuclei may be involved.

A question was discussed as to what the evidence is that small particles must get into the lung alveoli to induce infection. Dr. Cluff re-emphasized that it was not necessary for particles to get to the alveoli to produce infection. Either large or small particles may cause infection; the portal of entry, LD₅₀, and pathogenesis, however, will vary with the size of the particles.

LITERATURE CITED

1. GROSS, P., AND M. WESTRICK. 1954. The permeability of lung parenchyma to particulate matter. *Am. J. Pathol.* **30**:195-213.
2. MILLER, WILLIAM S. 1947. *Lung*. 2nd ed. Charles C Thomas, Springfield, Ill. 222 p.
3. POLICARD, A., A. COLLET, AND S. PREGERMAIN. 1957. Structures alvéolaires normales du poumon examinées au microscope électronique. Extrait de *La Semaine des Hôpitaux (Pathologie et Biologie)* No. IV. p. 385-398. *Annales de la Recherche Médicale* No. 2, février 1957, p. 125-138.