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Pecularities of Lesions in Viral and Mycoplasma Infections of the Respiratory Tract

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Summary. The author and his collaborators have studied 250 autopsied cases of acute viral respiratory infections and mycoplasma pneumonia chiefly in young children as well as the experimental effects of the inoculation of 400 animals with certain viruses. The present paper first dwells upon the common characteristics of this whole group of diseases, and then in greater detail, elucidates the morphological patterns seen in the separate diseases. The most typical changes develop in the alveolar and tracheobronchial epithelium. The formation of large uninuclear cells with a sharply increased hyperchromatic nucleus is representative of adenoviral infection. Later these cells, as well as other cells of the exudate, degenerate, showing a finely granular appearance. In RS-infections one observes papillary proliferations of the epithelium, particularly of the small and medium bronchi. In A2 (Hong Kong) influenza very large uninuclear and rather light cells develop. In mycoplasma infection the alveolar epithelium cells greatly increase in size, and the cytoplasm around the microorganisms becomes clear. The morphologic patterns of the different diseases of this group are quite specific and are of help in the differential diagnosis of various respiratory infections.

Zusammenfassung. Zur Untersuchung gelangten der Tracheobronchialbaum und die Lungen bei 250 Sektionsfällen des Säuglingsalters mit akuten Virusinfektionen und Mycoplasma-Pneumonien. Außerdem wurden die experimentelle Untersuchungen nach viraler Infektion der Lungen durchgeführt. Neben den charakteristischen gemeinsamen morphologischen Merkmalen werden die Besonderheiten der einzelnen Erkrankungsformen dargelegt. Besonders kennzeichnend sind die Veränderungen im respiratorischen Epithel der Trachea, Bronchien und Alveolen. Bei den Adeno-Virusinfektionen kommt es zur Bildung von großen einkernigen Zellformen mit hyperchromatischen Kernen, wobei im Infektionsablauf die Kerne einem kleinbröckligen Zerfall unterliegen. Bei der RS-Infektion (respiratory syncitial infection) kommt es zu einer papillomatösen Epithelproliferation, besonders in den kleineren und mittleren Bronchien. Bei der Grippe A_2 (Hong Kong) finden sich große einkernige Zellen mit hellem Cytoplasma. Bei der Mycoplasma-Pneumonie sind die Alveolarepithelien deutlich vergrößert, wobei es um das infektiöse Agens zu einer deutlichen Aufhellung des Cytoplasmas kommt. Die morphologischen Besonderheiten der einzelnen respiratorischen Infektionen werden als ausreichend spezifisch für die Diagnose angesehen und ermöglichen eine Differentialdiagnose der verschiedenen respiratorischen Infektionen.

Until now acute respiratory infections have been important in human pathology, particularly in children of an early age. Successful treatment and prevention of these diseases are impossible without clearly defining the etiology of each specific case. It is especially important to establish the cause when studying autopsy material, and not to limit oneself merely to diagnosis of pneumonia.

Materials and Methods

The results of 250 autopsied cases of acute respiratory infections, chiefly viral and mainly in young children, were used in this study. The special aim of these studies, which were carried out over many years, was a thorough clarification of the etiology of the infection. This was arrived at with the help of a detailed histobacterioscopic study of the preparation, azureosine dyed after the Gram-Weigert method with reagents of Schiff and Seller, and treated with luminescent serum. The data obtained was correlated with the results of bacteriological and virological investigations, and clinical observations. The whole study complex made it possible to establish the etiology of inflammatory organ lesions with sufficient accuracy, and to study the pathologic anatomy of individual diseases of this group. It should be noted that often the lesions found in the respiratory tract of an autopsied patient were not caused by a single agent, but had been brought about by from 2 to 5 agents: viruses, bacteria, fungi and protozoa. To make certain that the data obtained on the specificity of respiratory lesions under definite stimulants was correct, we carried out experimental studies of intranasal infection of 400 golden hamsters and white mice with several viruses.

Results

Acute Viral Respiratory Infection (AVRI)

When studying any uncomplicated acute viral respiratory infection (AVRI), the first features that draw one's attention are the traits common for the whole group of diseases. They pertain since virus propagation takes place chiefly, intracellularly in the epithelium of the respiratory organs (Figs. 3a, b; 4a; 5a; 6a). The infecting agent is located, more often than not, in the cytoplasm, and less common, chiefly in DNA viruses, in the nuclei.

In the affected cells certain changes develop; they can be revealed not only with the help of electronomicroscopic and immunofluorescent studies, but also with the help of light microscopy. Some of these changes manifest themselves in the lesion, arising as a result of the virus activity, and are expressed as degeneration or even necrosis. In addition, particularly under a favourable course of the disease, oxi- or fuchsinophilic inclusions appear in the cell cytoplasm after several days (Fig. 1). They represent reactive changes, whereby proteins of the cytoplasm condense around virus particles; owing to this the viruses are inhibited or quite often, destroyed. Subsequently we observe an extrusion of a part of cell cytoplasm with the inclusions. When the cell parishes the propagated virus is released and soon infects other, previously intact cells. Not seldom the affected epithelium undergoes giant-cell metamorphosis, which differs from infection to infection.

With the virus infection local inflammatory changes of different degree also appear. These changes are more often of a serous nature with exudation of macrophages and sometimes even neutrophil leucocytes.

The disturbances of blood-circulation are particularly characteristic of all AVRI, especially in the lungs. The disturbances consist of congestion, serous exudation and slight hemorrhage. Such changes chiefly occur in those cases (influenza, for instance) where the greatest degree of toxicosis exists and with the latter, where blood-vessel lesions are observed. These vascular lesions manifest themselves by a loosening edema and certain homogeneity of the vessel wall. In young children hyaline membranes quite often form when the disturbances in permeability of vessel walls are severe.

Besides the circulatory disturbances, nearly always small regions of atelectasis develop, quite often incomplete; these foci alternate with regions of acute emphysema (Fig. 2).

On recovery from AVRI the vascular and inflammatory changes disappear. The injured epithelium is replaced. Regeneration takes place as a sheet of flattened cells over the defect in the mucous membrane.

Macroscopic changes of the respiratory organs in AVRI are insignificant; they chiefly consist of a catarrhal inflammation of the respiratory tract and small regions of slight thickening which are dark-red or bluish-red.

With the local changes in the respiratory organs, as the disease progresses, general changes develop in the patient. These changes are associated first with the action of the viral toxins, and manifest themselves in circulatory disturbances, conditioned chiefly by permeability disturbances of the vessels. Circulatory disturbances are hypervolemia and moderate edema, and sometimes may be accompanied by perivascular hemorrhages. Such changes are seen most commonly in cases of influenza. In addition, a moderate alteration of protein and fat metabolism may be revealed in the parenchymatous organs.

In young children with AVRI inflammatory changes quite often arise in a number of internal organs (liver, intestines, etc.).

Influenza

During the first hours of illness the morphological changes are insignificant. Still the RNA-virus causing the disease may be revealed by immunofluorescent methods in the epithelium cells, chiefly in the mucous membrane of the upper respiratory tract (Fig. 3). As the virus multiplies it forms larger aggregates which can be seen with a light-microscope as basophil inclusions. At the same time lesions of the mucous membrane begin to appear: dystrophic changes of the epithelial cells arise. The bonds between them become broken, and they shed away, usually irregularly. In addition, especially when the infection tends to abate, the oxiphil inclusions develop where the virus has accumulated (Fig. 1a).

In the deeper levels of the walls of the respiratory tract a moderate edema, hyperemia and small infiltrates chiefly of round-cells, may be distinguished.

At this stage of disease desquamated epithelial cells, a few macrophages, neutrophil granula, and a small amount of serous fluid and mucus are present in the lumen of the respiratory tract.

In the lungs circulatory disturbances in the form of acute hyperemia, small hemorrhages, and alveolar edema are chiefly revealed (Fig. 3d). These changes, determined by the increased capillary permeability, are observed not only in the lungs, but in other organs as well, including the brain.

Considerably more seldom, chiefly in young children foci of inflammation with a loose exudate develop; the foci consist chiefly of macrophages with an admixture of occasional neutrophil leucocytes and erythrocytes. In A2 (Hong Kong) influenza we observe a greater hyperplasia of the alveolar epithelial cells with their transformation into large mononuclear cells, quite often considerably larger than the usual alveolar macrophage (Fig. 3e). During the early stage of the disease the influenza antigen may be disclosed in such cells. When infecting some experi-

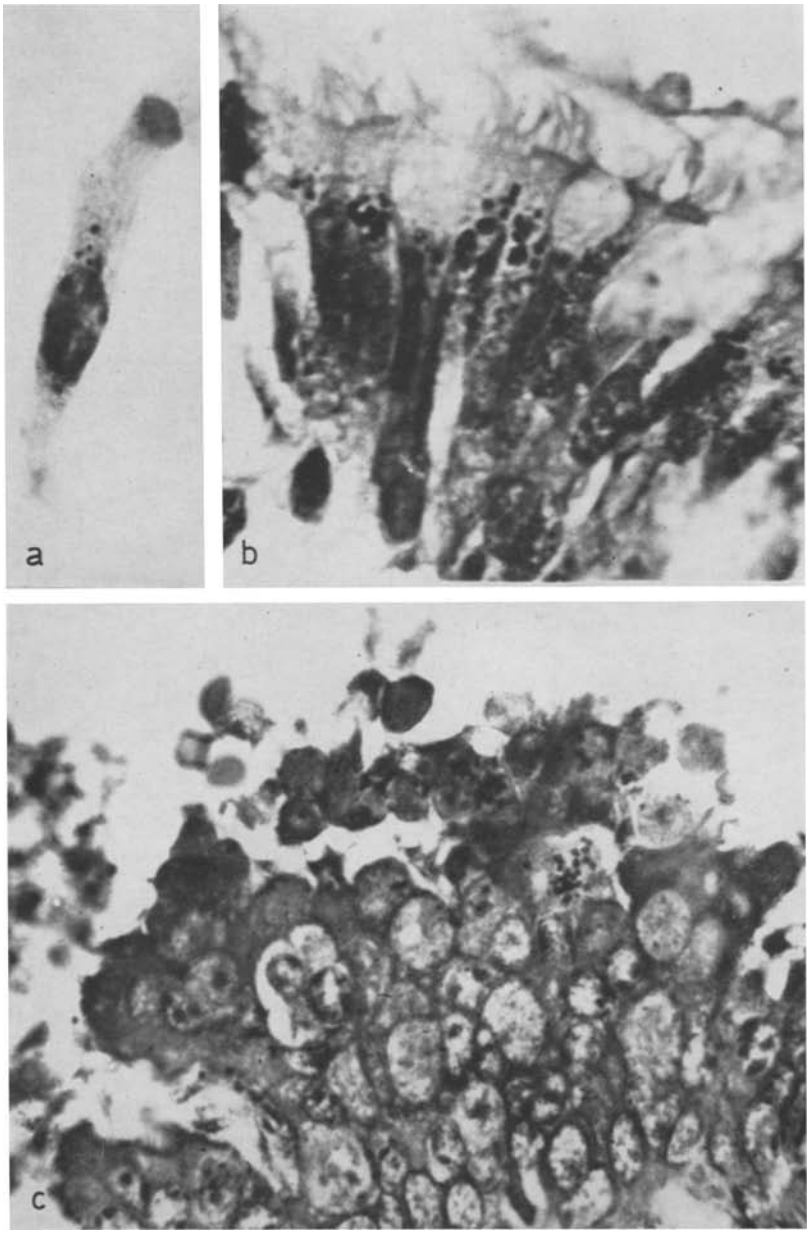

Fig. 1a—c. Fuchsinophyl inclusions in cytoplasma of respiratory epithelium stained by Seller: a Small inclusions in a cell of bronchial epithelium infected with influenza-virus A2 (smear, × 1350). b Numerous inclusions of various sizes in bronchial epithelium infected with paravirus 2 (microprep., × 1350). c Separation of a part of nipple-like growths arising from bronchial epithelium, the cells of which contain inclusions of experimental RS-infection of golden hamsters; the 10th day (microprep., × 1350)

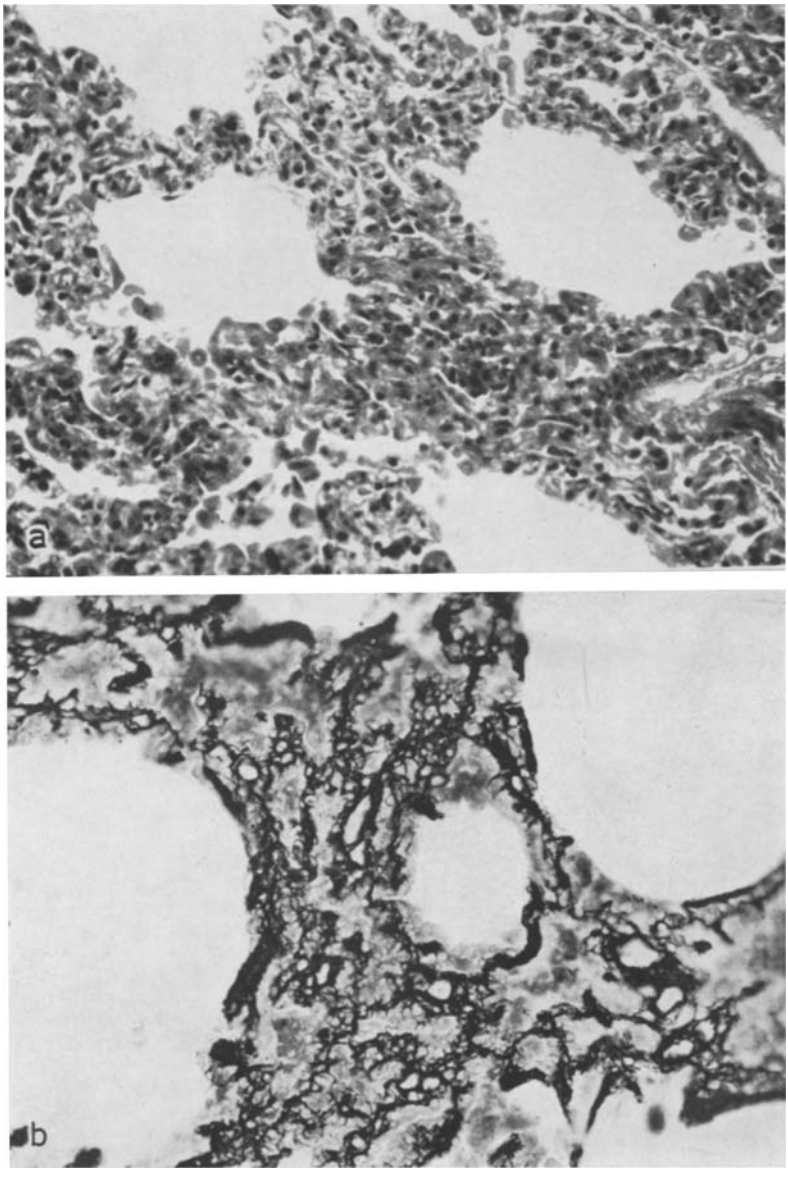

Fig. 2 a and b. Lung after adenovirus infection (remaining alterations): small regions of atelectasis and acute emphysema. Both are simulating interstitial pneumonia: a H.-E., × 240; b Silver-impregnation by Fut, × 240

mental animals with the influenza virus, the golden hamsters for instance, bronchoalveolitis, with a predominantly leucocytic exudate, develops (Fig. 3f). This allows us to regard the changes described during influenza as a viral bronchoalveolitis.

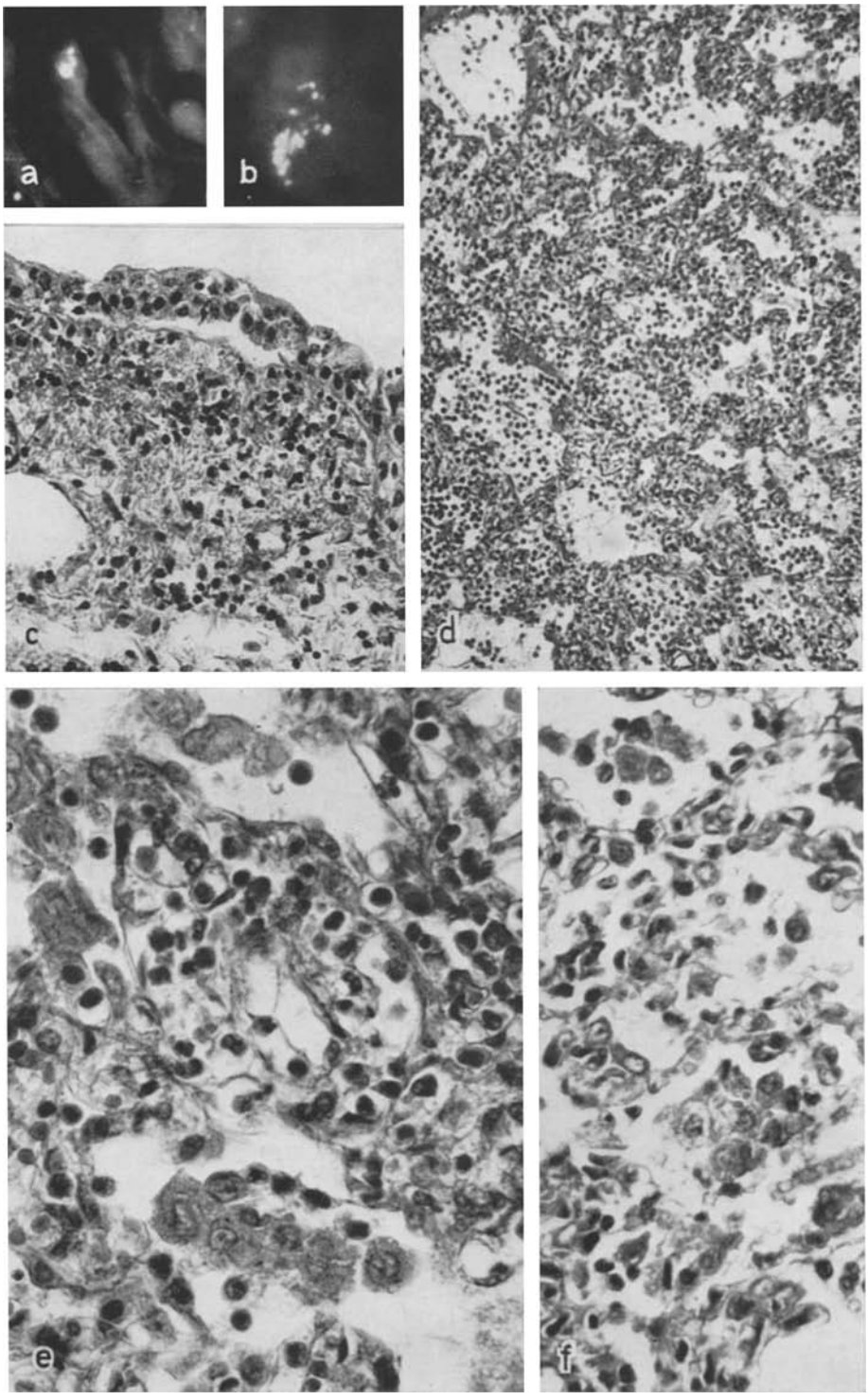

Fig. 3a—f

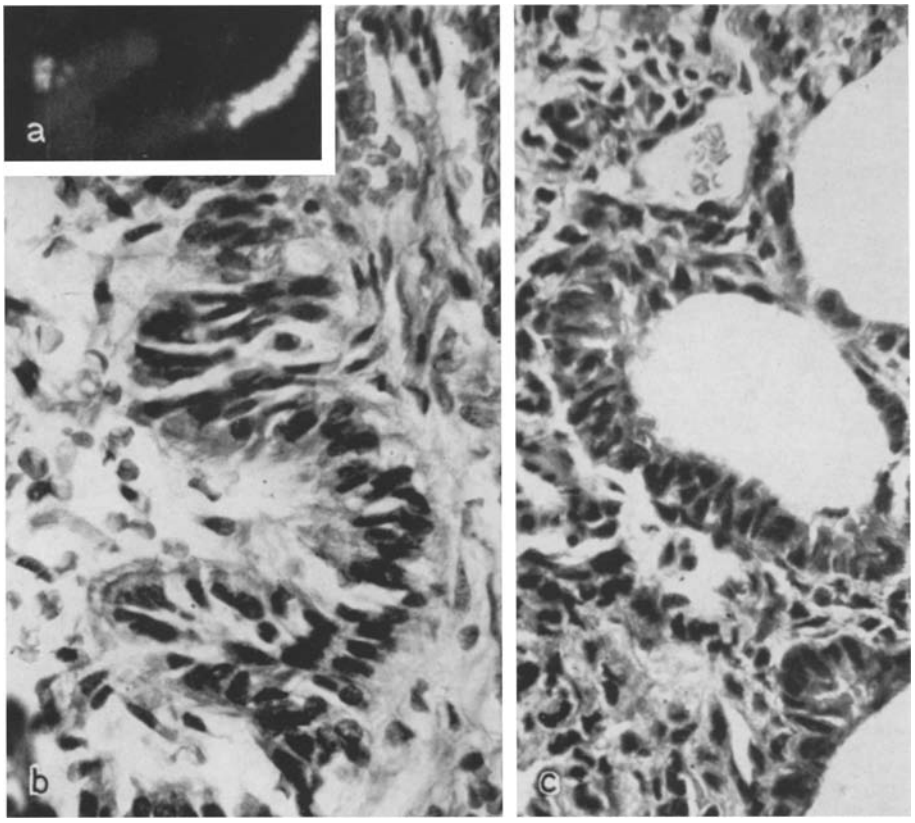

Fig. 4a—c. Parainfluenza serotype 3: a Antigen of the cells of bronchial epithelium (smear) treated with specific fluorescent serum; b bronchitis with hyperplasia of epithelium, H.-E., × 600; c Similar changes in mice on the third day after infection, H.-E., × 600

Fig. 3a—f. Influenza virus A2 (Hong Kong): a, b Antigen of the cell of bronchial (microprep.) and alveolar (smear) epithelium treated with specific fluorescent serum. c Beginning of regeneration of bronchial epithelium, H.-E., × 300. d Acute hyperemia of lung, alveoli contain serum and erythrocytes, macrophages and leucocytes, as well as hyaline membranes, H.-E., × 300. e In alveoli one can see numerous desquamated alveolar cells H.-E., × 600. f Virus bronchopneumonia of golden hamster on the third day after infection with virus A2 (Hong Kong), H.-E., × 600

A few days after the onset of the disease, regeneration of the respiratory epithelium begins. At first poorly differentiated, flattened epithelial cells appear in sections of the injured mucous membrane. They then pile up to form several layers, giving the epithelium a polymorphous appearance or even a multilayer flat cell appearance (Fig. 3c). Subsequently, as a rule, the cells differentiate into an ordinary epithelium with cilia.

Macroscopically in uncomplicated influenza a catarrhal inflammation of the respiratory tract is accompanied by a moderate hyperemia of the mucous membrane with a scanty yellowish coating. The lungs are only slightly changed. In the posterior lower parts of the lung a moderate focal condensation of dark red tissue can sometimes be distinguished from the remaining well-aerated reddish lung. In addition, partly emphysematous anterior sections of the lungs may be noted. Hemorrhagic tracheobronchitis, which is still considered by many to be characteristic of influenza, is met with only when it is complicated by a secondary infection, staphylococcal in particular. With the same complication a "large variegated lung", considered so characteristic of influenza in twenties, may develop.

Parainfluenza

Morphological changes, associated with parainfluenza, are similar to those found with influenza (Figs. 1b, 4). One of its distinctions is its more frequent involvement of the larynx with stenosis (false croup); and quite frequently smal kidney-like growths of the bronchial and bronchiolar epithelium develop (Fig. 4). In the lung there is a moderate hyperplasia of the alveolar epithelium, and in some of the alveoli a serous fluid accumulates, containing occasional macrophages, erythrocytes and sometimes leucocytes.

With the latter edema circulatory disturbance and other changes common for all AVRI come about.

Respiratory-Syncytial Infection

In RS-infection, in contrast to influenza and parainfluenza, the infection is most strongly expressed in the small and medium bronchi (Fig. 5). The epithelium cells affected by the virus undergo rather peculiar changes. Some of them increase in size, become lighter, and then proliferate. As a result multilayer nipple-like outgrowths develop, occupying sometimes a considerable part of the bronchial lumen.

As seen in the histological sections, the alveolar lining cells undergo a moderate hyperplasia; from time to time a tendency for the formation of nipple-like growths may be noted. In addition, small pneumonic foci may develop, which are characterized by an accumulation of cells, chiefly macrophages, in the alveoli.

Apart from such specific changes, circulatory disturbances, mostly insignificant and small focal atelectasis and emphysema may also be noted.

As recovery proceeds the exudate resolves and the hyperplastic epithelium recedes. In the golden hamsters infected with RS-virus, these features of resolution may be observed at the beginning of the second week (Fig. c 1).

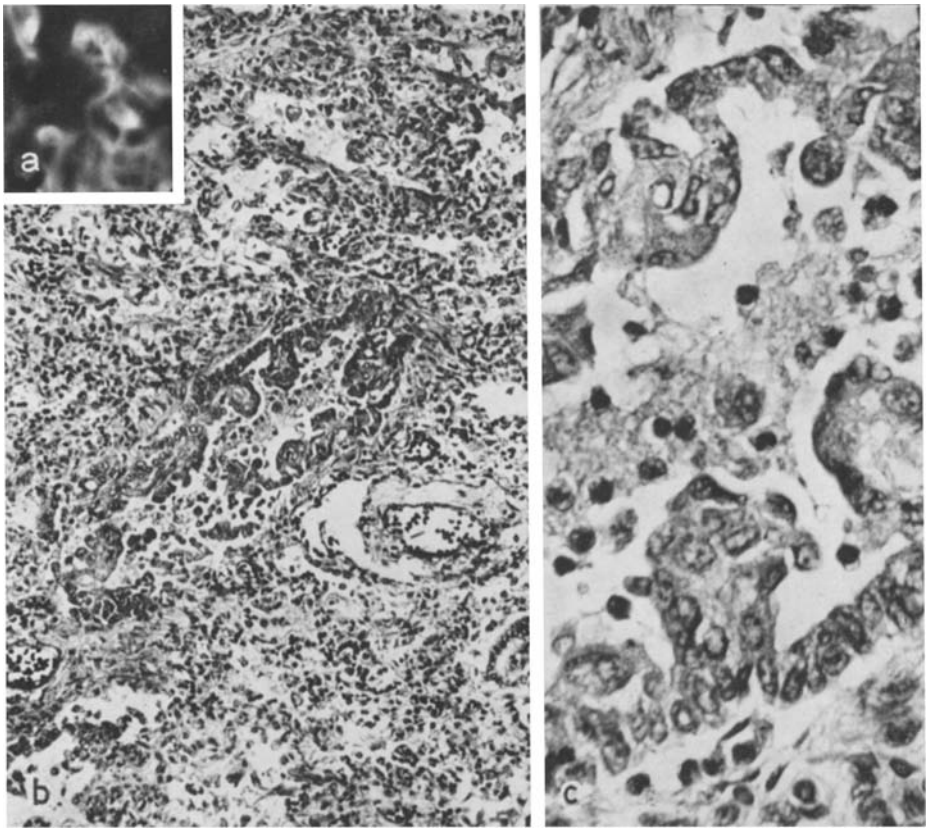

Fig. 5a—c. RS-infection: a Antigen on the cells of bronchial epithelium (microprep.) treated with specific fluorescent serum. b Bronchopneumonia with nipple-like growths of bronchial epithelium, H.-E., × 135. c Detail of the previous picture, H.-E., × 600

Macroscopically, the changes in the respiratory organs are moderate, consisting of catarrhal tracheobronchitis, of small reddish foci of condensation in the posterior parts of the lungs, and of increased inflation of their anterior parts.

Adenoviral Infection

In adenoviral infection the earliest changes are observed in the epithelium of the respiratory tract and alveoli. The DNA-virus first appears in the cytoplasm, and later in the nuclei, it accumulates in greatest quantity in the nuclei, however. Because of the size of the large inclusions, they can be clearly seen even in preparations dyed with hematoxylin-eosin (Fig. 6b, e). They are considerably more basophil than the chromatin. On the whole, the nucleus appears enlarged and hyperchromatic. The epithelial layer quite often becomes loosened and its cells may be shed into the bronchial lumen singly or in layers.

With these specific cells, called adenovirus cells, droplet-like protein masses, flakes and filaments appear in the lumen of the bronchi and alveoli; erythro-

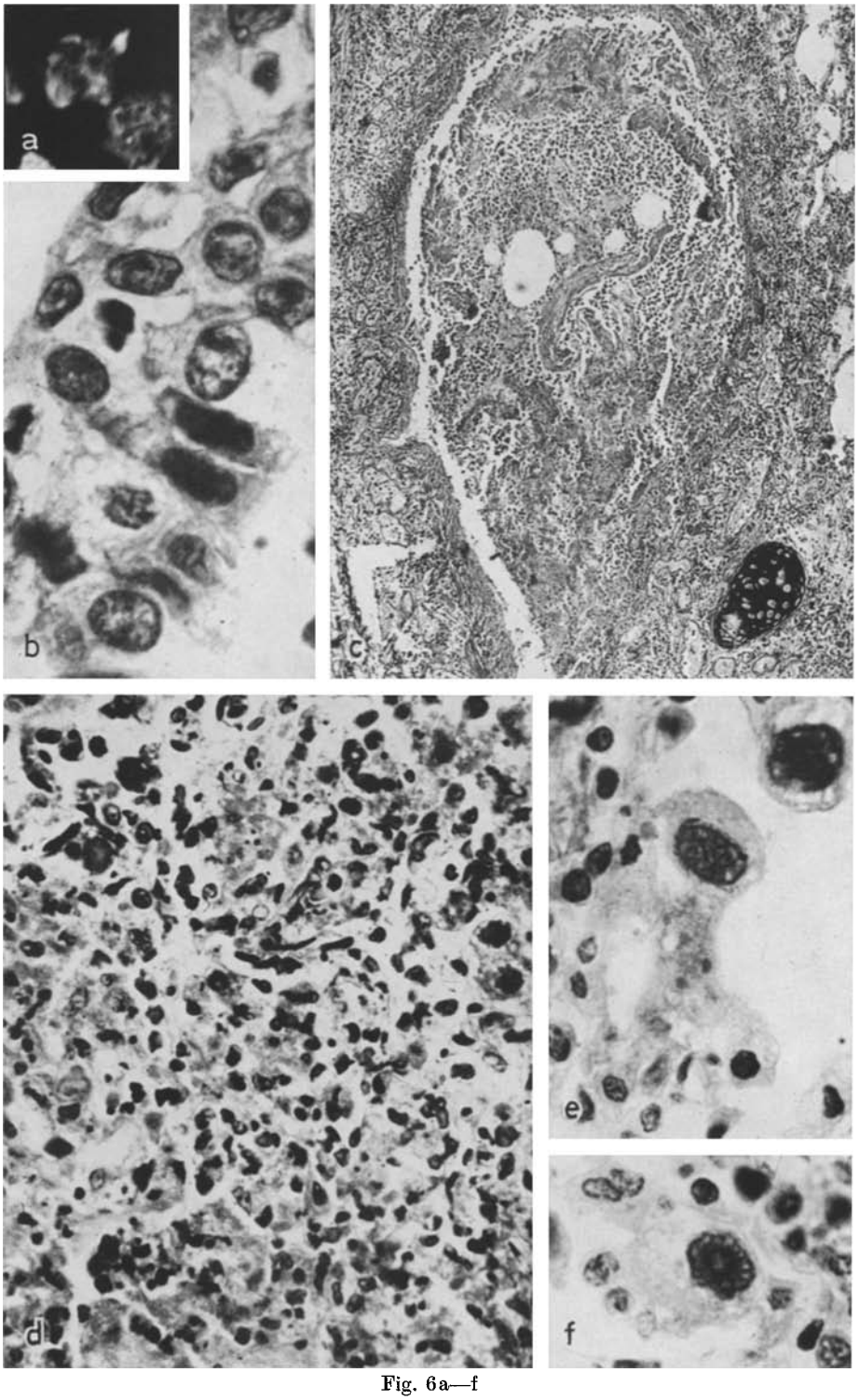

Fig. 6a—f

cytes, macrophages and a small number of neutrophil leucocytes may also be seen (Fig. 6d).

At later stages of the infection the exudate and epithelial cells show a finely granular degeneration that is unusual for other types of infections. The formation of basophil round bodies of different size (Fig. 6c) may be observed.

Quite often the blood-vessels undergo change, showing edema, filament-separation of their walls, and sometimes thrombosis. Dysfunctional changes as atelectasis and emphysema may also develop (Fig. 2).

As the process abates a gradual resolution and regeneration of epithelium take place; dysfunctional changes remain longer.

Macroscopic changes are moderate. A catarrhal laryngotracheobronchitis is present characterized by a slight swelling of the mucous membrane of the respiratory tract and by scanty yellowish deposits on it. Regardless of the duration of the diseased small red or dark-red foci of atelectasis with a smooth moist surface on section are evident.

Mycoplasma Pneumonia

Though mycoplasma are not viruses it is expedient to compare the pneumonia produced by them with viral respiratory infections, since similarities in their manifestations exist which must be considered in carrying out differential diagnosis.

On microscopic study of the lesions of the respiratory organs caused by Mycoplasma pneumoniae, the first change that draws one's attention is the peculiar appearance of the alveolar epithelium (Fig. 7b, c, d). In many sections of the lungs its cells are considerably enlarged. In their cytoplasm, and sometimes in the nucleus, an organism is disclosed lying either singly or clustered as numerous small bodies of different shapes. They are characteristically surrounded by clearly defined lucid cytoplasm. Later, such an altered epithelium desquamates and undergoes lysis. With infection by inhalation some of the cells of the bronchial epithelium undergo similar changes (Fig. 7a).

With a more severe infection bronchopneumonia arises (Fig. 7f, e). Besides the desquamation of greatly altered cells of the alveolar epithelium, the alucoli and bronchi contain ordinary macrophages, serous fluid erythrocytes, either singly or aggregated, and sometimes also a few neutrophyl leucocytes. The alveolar septa are swollen. Quite often there is also a moderate partial atelectasis. In addition, one may find interstitial pneumonia, characterized by a thickening of interalveolar septa and by infiltrates of inflammatory and plasma cells about vessels in small regions of the lungs.

Fig. 6a—f. Adenovirus-infection: a Antigen on the cells of alveolar epithelium (microprep.) treated with specific fluorescent serum. b Some cells of bronchial epithelium are hyperchromatic and enlarged, H.-E., × 1350. c Lumen of the bronchus is filled with exudate, which undergoes a fine-grain decomposition with formation of hyaline membranes. H.-E., × 100. d Alveoli are filled with exudate, consisting of protein substances, leucocytes and large hyperchromatic cells, and undergoning fine-grain decomposition, azur-eosine, × 600. e, f Exudate contains large uninucleated "adenovirus" cells with typical lucid regions between intranucleus inclusion and a remaining part of nucleus, H.-E., × 900

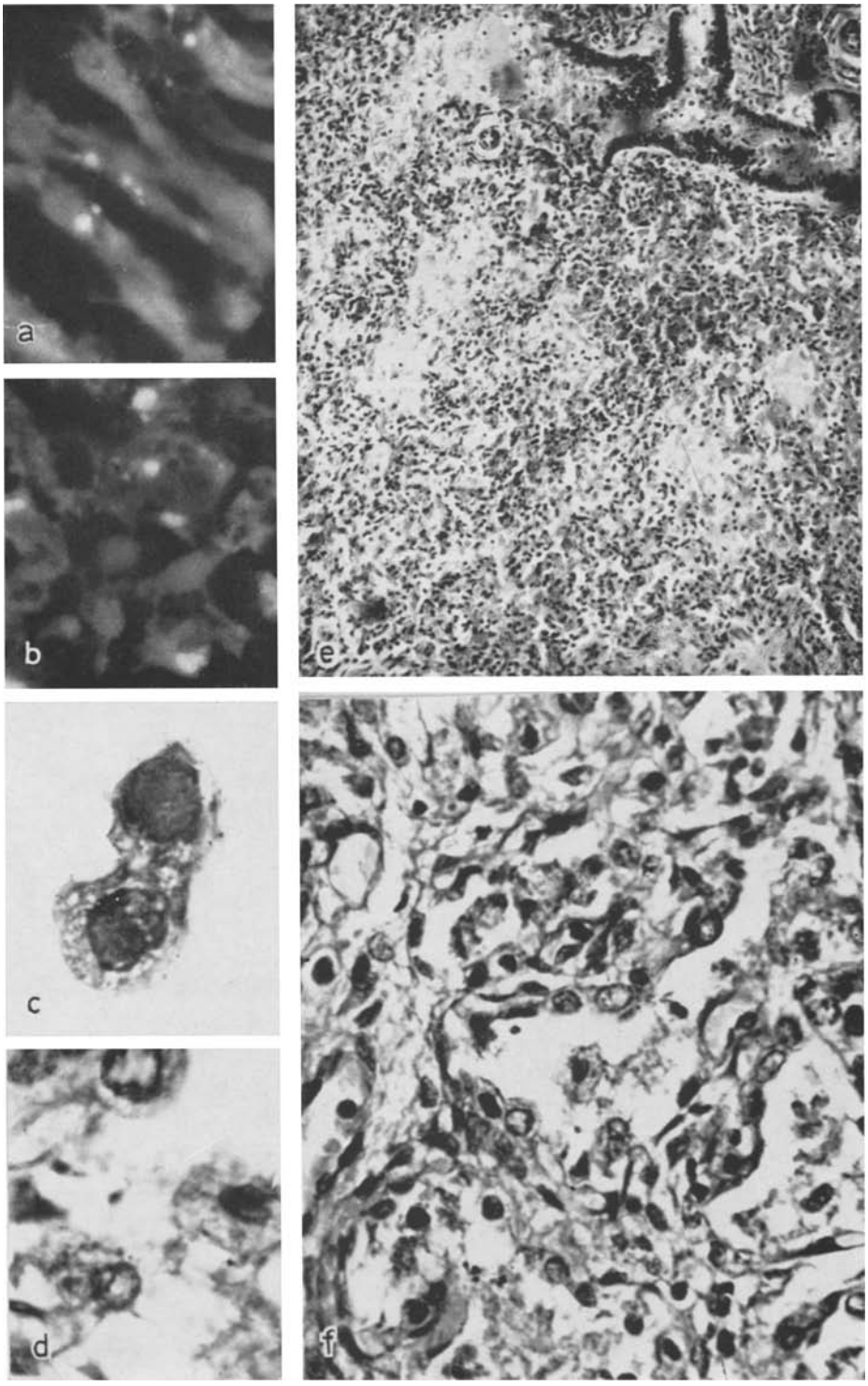

Fig. 7a—f

Macroscopically changes of the respiratory tract are moderate, consisting of a slight swelling of the mucous membrane. In the lumina of the trachea and bronchi the small amount of mucus is whitish-grey or pinkish-red. The lungs, chiefly in the posterior parts put rarely everywhere, are moderately condensed and dark-red. From the fresh surface of these parts a small amount of bloody fluid containing bubbles of air may exude forth.

With involvement of the respiration organs changes in other organs, predominantly kidneys and liver, may be observed in almost 50% of children, especially in the newborn. A hemorrhagic diathesis is manifested quite often.

Discussion

Although respiratory infections are significant in human pathology, until now identification of the etiology of respiratory infections has not generally been carried out, especially in autopsy material. Diagnosing pneumonia without determining its cause is insufficient, since it does not permit one to judge either the epidemiology or the pathogenesis, or the course of diesease, and in addition, it is not possible to evaluate the efficacy of the antibacterial treatment prescribed. To determine the etiology of pneumonia best it is necessary to make use of all possible methods of investigation. Therefore, when studying autopsy data one must not confine oneself merely to bacteriological and virological methods of investigation. Besides these methods, the infecting agents in the tissues must be isolated and studies of their reactions in cells, organs and animals must be carried out.

In some acute respiratory infections it is not difficult to make a diagnosis from morphological changes. That is true especially for adenoviral infections, the pathological anatomy of which has been described to a considerable extent by Lelong *et al.* (1956), Nahmias *et al.* (1956), La Tan Vinh (1961), Vishnevetskaya (1964), Güthert *et al.* (1965), Koroleva *et al.* (1967), Maksimovich *et al.* (1970), and others. The authors of the present paper dwelt upon the question in 1965. In all these papers no distinctions were made in the description of the chief changes; the differences lay only in their evaluation.

The investigations devoted to RS-infection are considerably fewer (Adams *et al.*, 1961; Schneweis *et al.*, 1966; Zinserling *et al.*, 1967; Aherne *et al.*, 1970). However, the descriptions in these papers by different authors are approximately alike.

The few papers devoted to parainfluenza are also analogous (Nods, 1953; Zinserling, 1970; Anisimova, 1970).

Fig. 7a—f. Mycoplasma-infection: a Antigen of the cells of bronchial epithelium treated by specific fluorescent serum. b Fluorescence of antigen on the cells of alveolar epithelium (microprep.), treated with the same serum. c The cells of alveolar epithelium with numerous distinct regions of lucid cytoplasma which contain mycoplasma (smear, stained by Seller, × 1350). d The same picture of alveolar cells (microprep., stained with azur-eosine, × 1350). e Areal "desquamative" pneumonia, H.-E., × 135. f Hypertrophy and desquamation of alveolar epithelium in the lumen of the alveoli. One can also see protein substances, macrophages and single leucocytes, stained by Schiff and Shabadash, × 600

The numerous investigations of influenza are considerably more contradictory. It is to be regretted that in the overwhelming majority of these studies lesions caused by influenza virus were not differentiated from lesions associated with secondary bacterial microflora. In connection with such investigations those of Maksimovich *et al.* (1965) and Zinserling (1970) are of particular interest. In their studies investigated for viruses they confirmed cases of influenza not complicated by bacterial pneumonia. These authors showed that the old concepts are wrong, and that in cases of "pure" influenza, as a rule, hemorrhagic tracheobronchitis does not occur.

Pathological anatomy of mycoplasma infection has been studied considerably less. Evidently there are no investigations published besides the works of the author (Zinserling, 1972), in which a differentiation of respiratory organ lesions caused by mycoplasma and other microorganisms has been carried out. Pulmonary lesions, described in the works of Golden (1944), Parker *et al.* (1947) and Maisel *et al.* (1967), were caused by different agents. The conclusions drawn were reliable only in part, since the evidence presented by the authors that they had studied mycoplasma pneumonia is questionable.

Acute viral and mycoplasma infections affecting both the respiratory tract and lungs, particularly in children of an early age, produce focal viral pneumonia quite often. In cases with pulmonary lesions certain characteristic morphological distinctions are present; in typical cases these permit one to diagnose the diseases and to differentiate them from one another.

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