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HISTOLOGY OF SURGICALLY RESECTED LUNGS

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Evaluation of histological changes in the lungs at necropsy may be interfered with by postmortal autolysis, changes in the distribution of blood and by the effects exerted by the fatal disease. Moreover, at necropsy we see the final stages of the pathological process only, even if some pulmonary condition had been the basic disease. Morphological studies of lungs operated on in different phases of the diseases may allow some insight into the various stages of the pathological process. Before operation the patient is subjected to detailed clinical study and thus the clinical and pathological findings may be analysed together. The lungs removed by surgery and fixed without delay are much more suitable for studying their fine histology than those from cadavers.

The present paper is not intended to discuss the histologic changes occurring in various pulmonary diseases, and only their often closely similar effects on the blood vessels, connective tissue, smooth muscle and bronchi will be dealt with. By presenting in detail the case record, showing X-ray films and describing the gross changes, it would be possible to determine the special mechanical tractile forces and their sequelae. These, however, are different almost from patient to patient and therefore little could be gained from a detailed discussion of a few cases. On the other hand, to deal with many cases in detail would exceed the scope of the paper and for this reason we shall discuss only general laws and conclusions. Some of the changes outlined exceed the limits of the well-known quantitative adaptation process and fulfill the requirements of qualitative adaptation, of neodifferentiation. A more detailed description and analysis of these occurrences has been made possible by the studies of I. KROMPECHER on the experimental influencing of tissue differentiation. Our results concerning the human lung may confirm his data obtained in animal experiments.

Material and methods

The material subjected to study was obtained from the 3rd Department of Surgery, University Medical School, Budapest. Of the lungs operated on in the period from January 1, 1954, to June, 1958, 285 were studied in detail.

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In the hilar area of the resected lungs the branch of the pulmonary artery was identified and was injected with 10 per cent formaldehyde until the lung was completely expanded. The preparation was then submerged in a 10 per cent formaldehyde bath. Histological sections were made from the wall of cavities, from small and large bronchi, from the junction of the pathological and intact areas, as well as from the hilar and peripheral areas. Haematoxylin-eosin, van Gieson's, resorcin-fuchsin, Giemsa's and Heidenhain's azan were the stains used. In addition, lungs from 39 patients who had died of various pulmonary diseases were studied; these specimens originated from the same period.

The material was divided into 3 groups, *viz.*

Group 1: tuberculous lungs.

Group 2: bronchiectatic lungs.

Group 3: lungs removed because of chronic inflammation (chronic abscess, gangrene, pneumosclerosis, mycosis, echinococcus).

It was sometimes difficult to differentiate group 2 from group 3, because bronchiectasis was often associated with abscess or pneumosclerosis and *vice versa*. The material was analysed group by group and *in toto* according to sex, age and localisation. These data are presented in Tables I, II and III. In view of the small number of autopsied cases it is not really justified to present percentage data; they are nevertheless given, in order to facilitate evaluation and to point out the essentials.

As shown in the Tables, 59 per cent of the operated cases were males and 41 per cent were females. Of the cases subjected to autopsy, 66 per cent were males and 33 were females. Thus, both groups contained significantly more males than females. Eighty per cent of those operated on because of tuberculosis belonged to the age group of 20 to 40 years. Sixty-six per cent of the patients operated on because of chronic inflammation were 40 to 60 years old, while 55 per cent of those operated on because of bronchiectasis were 30 to 50 years of age. Most of the patients with tuberculosis were 30 to 50 years old; 63 per cent of the cases autopsied because of tuberculosis originated from those age groups. Seventy-seven per cent of the patients who had died of chronic inflammation were 40 to 60 years of age. Likewise, bronchiectasis killed 87 per cent of the patients in the age group 40 to 60. For tuberculosis the right upper lobe was resected in 34 per cent and the left upper lobe in 21 per cent. Segmental resection was performed in 36 per cent of the cases. Because of chronic inflammation the middle lobe was resected in 21 per cent and the left lung also in 21 per cent of the cases. Because of bronchiectasis the left lower lobe was resected in 27 per cent, the right lower one in 20 per cent and the left upper one in 22 per cent of the cases.

In the following, the effects of the various pathological processes on the vascular system, bronchi lymphatic tissue, collagen and elastic fibres will be dealt with.

1. Vascular changes

In each of the three groups vascular changes were common and extensive. The large and medium arteries near the hilum showed a thickening of the elastic internal membrane (Fig. 1). The lumen was markedly narrowed by the proliferating intima. Changes of the media were less common and less severe. Extreme hypertrophy of the media was found particularly in the atelectatic indurated pulmonary areas. The muscle fibres were thickened and increased in number (Fig. 2). Here, too, the lumen was narrowed by the granulation tissue in the intima. In the media of other blood vessels diffuse or scattered fibrosis with destruction of the muscular fibres was visible, the lumen was narrowed or obliterated by thrombi, fresh or showing different grades of organization. Some areas were so severely affected that remnants of the vascular wall could only be found. Similar changes occurred in the adventitia. The smallest arteries exhibited the most severe changes.

Vascular lesions occurred not only in the diseased areas, but sometimes also in the collapsed or relatively normal tissue. They were especially frequent

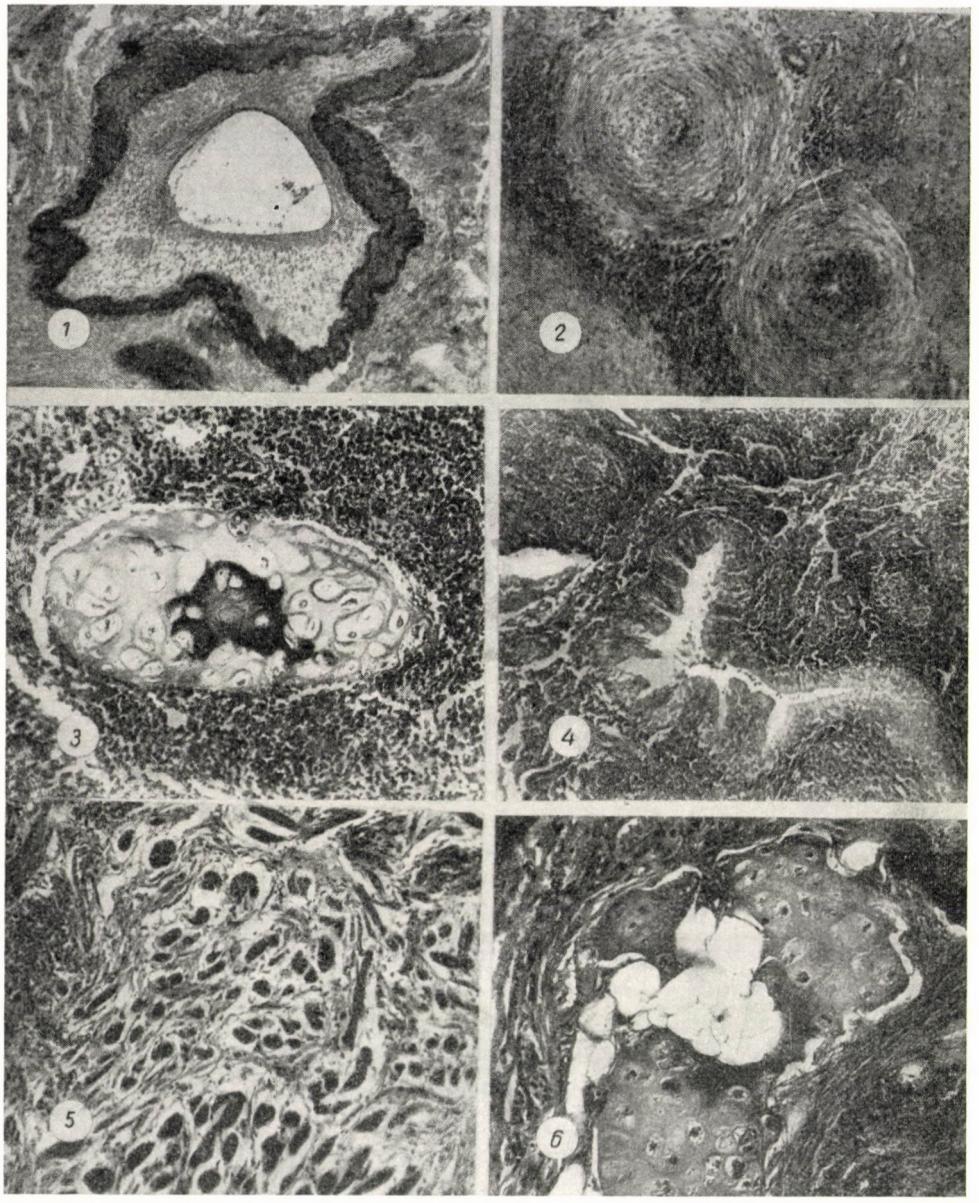

Fig. 1. Vascular lumen narrowed by tissue proliferating in the intima. Thickened internal lamina elastica. H. E. ×48

Fig. 2. Marked increase in the number and volume of muscle fibres in the media. Narrowing of vascular lumina. H. E. ×48

Fig. 3. Degeneration and central calcification of the cartilage ground substance, surrounded by inflammatory elements. H. E. ×48

Fig. 4. Lymph tissue hyperplasia with germinative centres around a small bronchus. H. E. ×48

Fig. 5. Muscular hyperplasia and hypertrophy of bronchial muscle. H. E. ×48

Fig. 6. Cyst formation in cartilage, showing mucous degeneration and calcification in the right upper portion. H. E. ×96

in the cases treated previously by pneumothorax. In the peripheral areas changes in the arterioles and precapillaries dominated the picture. In some cases they bore resemblance to the changes occurring in hypertensive vascular disease. The subendothelial tissue was imbibed and thickened circularly by a homogeneous material staining red with eosin and pink with azan and staining also with Sudan, similar to the lipo-hyalinosis occurring in the kidney. Changes in the bronchial arteries (similar to those outlined above) were found chiefly in the bronchial vessels next to the hilum.

Similar, though less common and less severe changes were found in the veins.

2. Bronchial changes

We do not intend to deal with the various types of bronchial inflammation and the secondary changes of the bronchial mucous glands. From our point of view it was of great importance that in some areas the severe inflammation had completely destroyed the bronchial wall and all that could be recognized between the inflammatory elements were a few isolated islets of cartilage (Fig. 3). The granulation tissue had attacked also the cartilage; its ground substance underwent degeneration, with calcification of the central areas. Cartilage changes of such severity occurred in 3 of our cases only. There was also degeneration and atrophy of the perichondrium, often with round cell infiltration. It has to be emphasized, however, that it is not these inflammatory changes leading to destruction of the cartilage that play a role in the ossification of the bronchial cartilage, a process to be discussed later.

Hypertrophy of the lymphatic tissue was especially common in cases of chronic bronchiectasis, in the carnifications around chronic abscesses. Germinative centres had formed around small bronchi (Fig. 4), in other cases independently of them.

Hyperplasia and hypertrophy of the bronchial muscles was extreme in some cases: even the smallest bronchioles were embraced by thick bundles of muscle. In one case there was extensive smooth muscle hypertrophy (Fig. 5). Some hypertrophied bundles were connected with the bronchial muscle, most of them, however, proliferated independently. The single bundles of muscle were surrounded by connective tissue fibres; Van Gieson's stain revealed their connection with the peribronchial connective tissue bundles.

In some cases the peribronchial connective tissue was replaced by fatty tissue. Like the above-described hyperplasic muscle, this sometimes showed excessive proliferation. It contained blood vessels and nerves, with islets composed of lipoblasts.

The carnified and indurated areas, the perifocal, peribronchial proliferations around the cavities showed on staining with azan great masses of wavy

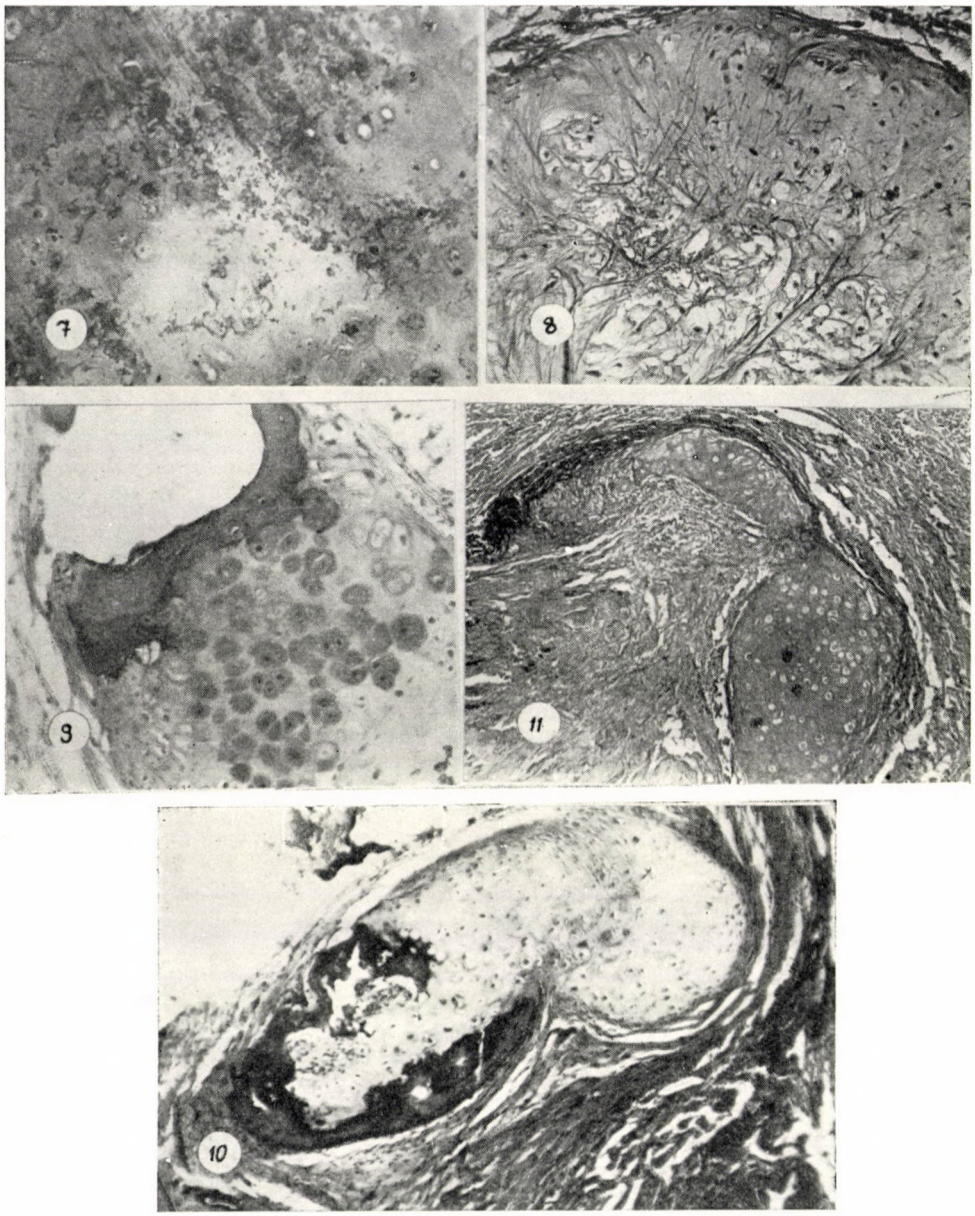

Fig. 7. Fine calcium granules in degenerated and necrosed cartilage. H. E. ×48
Fig. 8. Gelatinous undifferentiated connective tissue containing fine fibres, replacing destroyed cartilaginous tissue. H. E. ×48
Fig. 9. Upper peripheral part of bronchial cartilage converted to bone tissue with fatty marrow. Calcification of adjacent cartilage ground substance. H. E. ×48
Fig. 10. Lacunar chondrolysis and bone formation from undifferentiated granulation tissue, side by side in the cartilaginous tissue calcified in the shape of a two-tooth fork. H. E. ×48
Fig. 11. Elastic fibres increased in number and volume embracing necrosed cartilage with beginning calcification. H. E. ×48

collagen fibres in a haphazard distribution, and in the interspaces elastic fibres exhibiting ramifications and forming net-like plexuses, staining dark brown with resorcin-fuchsin. In some areas the elastic network of the lung was destroyed, in others it was increased, forming thick bundles.

The changes of the bronchial cartilage in the areas free of inflammation were characterized in some areas (especially at the periphery) by the formation of true bone marrow. The cells and ground substance of the cartilage exhibited severe degeneration. Pycnosis, karyorrhexis, kariolysis, fatty degeneration, calcification, mucous degeneration of the ground substance, vacuolization leading to the formation of cysts appeared side by side (Fig. 6). The acidophilic halo around the cartilage cells turned basophilic, was calcified and stained red with azan. Fine granules of calcium appeared in the destroyed cartilage cells and degenerating ground substance (Fig. 7). The place of the destroyed cartilage was occupied by an undifferentiated jelly-like connective tissue with fine fibres (Fig. 8), with positive mucus stainings, but no elastic fibre staining. In the peripheral areas true lamellar and spongious bone tissue appeared, with partly haematopietic and partly fatty bone marrow. In Fig. 9 it is clearly visible that the peripheral part of the bronchial cartilage has been transformed into bone containing fatty marrow and the adjacent cartilage ground substance is calcified. Ossification is restricted almost exclusively to the peripheral areas surrounding fork-wise the central, still intact cartilage. Chondrolysis and bone formation go side by side (Fig. 10).

This inflammation-free, reaction-free bone tissue metaplasia occurred exclusively in the cases exhibiting extensive pleural adhesions. Bone formation was localised everywhere to the cartilage, ossification of the mucous membrane similar to that observed in osteoplastic tracheobronchitis did not occur. Most of the cartilages of the perihilar bronchi of large and medium size were ossified. The bone trabecules were thin, with a few osteoblasts and no osteoclasts. Elastic staining demonstrated clearly how the elastic fibres had increased in number and thickened around the calcified areas (Fig. 11), embracing the cartilaginous and ossifying parts. In many areas the proliferating elastic fibres had invaded the ossifying focus and the ground substance, branching off in various directions in the adjacent lung tissue. This ossification of the bronchial cartilage occurred in 25 per cent of the cases operated on for tuberculosis, with 80 per cent frequency in the 20 to 40 years age group. It was present in 22 per cent of all the autopsied cases, in 22 per cent of cases autopsied because of tuberculosis and in 25 per cent of the lungs removed because of chronic inflammation. The age distribution was similar. Ossification was revealed in 40 per cent of the lungs removed because of bronchiectasis (in 75 per cent of the patients aged 20 to 40), in 50 per cent of the autopsied cases with bronchiectasis, in 27 per cent of all the operated, and 30 per cent of all the autopsied

cases. Thus it was more common in association with bronchiectasis than with tuberculosis or chronic, non-specific inflammation.

Discussion

SCHMIDTMANN et al. [37] were the first to publish a detailed description of the pathological changes in surgically removed lungs. They laid particular emphasis on a comparison of the changes occurring in subjects treated with various antituberculotic drugs and those in untreated subjects. OLSON et al. [32] examined 602 resected pulmonary specimens and studied the various types of bronchitis in some detail. ANDREW et al. [2] reported on the pathological changes in 100 resected lungs (with emphasis on the bronchi) in the period 1951—55. SWEANNY et al. [39] examined 34 resected preparations, ABELLO [1] published a detailed histologic study on the bronchi of the lobectomized lung. All these authors presented a detailed analysis of the changes caused by the basic illness.

Numerous reports have dealt with the vascular changes in the tuberculous lung [21, 8, 4, 5, 15, 34, 40, etc.]. FÖLDES [13] found fragmentation and total destruction of elastic fibres in the blood vessels in areas adjacent to or distant from the tuberculous focus. DENST et al. [9] described vascular changes in lungs resected because of tuberculosis. According to TERPLAN, the vascular changes need not be of tuberculous origin. They may be reactive, resulting from the increasing collapse, induration and chronic local circulatory overstrain. PAGEL et al. emphasized the importance of chronic local tissue anoxia, a chief factor in secondary infections. Similar vascular lesions were described in acute and chronic atypical pneumonia by GREVER et al. [14].

The mechanical factor also has a role of considerable importance. Studying the resected lungs from patients with therapeutical pneumothorax, DENST et al. found the pulmonary small arteries frequently affected.

COURNAND [6], COURNAND et al. [7], MENDELSOHN et al. [30], DRESSLER et al. [11], HUST et al. [17] tried to find a correlation between the pathological vascular changes and the evidence yielded by cardiac catheterization. Cardiac catheterization was made by HURST et al. before and after lobectomy and pulmonectomy in 85 cases. Their data make it clear that there is little relationship between the vascular changes and the catheterization findings. In some cases pressure was high and the blood vessels were normal or minimally affected, and *vice versa*. MENDELSOHN et al. studied pulmonary haemodynamics during lung resection and following the ligation of major blood vessels found that after a transient rise pulmonary arterial pressure was soon normalized. The pulmonary blood vessels are extremely elastic; they can adapt themselves to a threefold increase of blood volume without any appreciable elevation of pul-

monary arterial pressure. According to EULER et al. [12] anoxia leads to vasoconstriction. MOTLEY et al. [31] induced pulmonary hypertension by the inhalation of a gas mixture containing (10—12 per cent O_2—N). The above observations indicate the great importance of anoxia; this means that the vascular lesions revealed at autopsy do not allow conclusions as to the conditions of pulmonary circulation.

Our observations, too, have proven that at least three types of vascular lesion may develop in the blood vessels of the lung. 1. Inflammation of the parenchyma may directly spread to the blood vessels in the affected region: in such cases the inflammatory (acute or chronic), infiltrative or proliferative process is in the foreground; such arteritis or phlebitis is often associated with thrombosis. This type is frequently encountered in tuberculosis, especially near to the cavities, but it may occur also with bronchiectasic cavities, and with other conditions. 2. Diffuse productive arteritis, presenting itself mainly in the form of intimal fibrosis and fibroelastosis. In its development a spread of inflammation from adjacent tissues may be ruled out. Mechanical factors, bacterial toxins, local tissue anoxia may play a role; allergic causes have also been mentioned in the literature. 3. The arteriolo-hyalinosis, fibrosis, the lamellar elastosis of small arteries, the consequent muscular hypertrophy appear to be due to hypertension; in our opinion, pulmonary hypertension caused by anoxia might be involved in their development.

The increase of bronchial and bronchiolar musculoelastic tissue may be considered to be a hypertrophy of effort; it is due to an interference with breathing and expectoration by the extensive pulmonary and pleural process.

Peribronchial lipomatosis is an uncommon phenomenon, but it invariably accompanies atrophy of the muscles of non-functioning bronchi and thus may be looked upon as being an increase of non-functioning tissue, in analogy to pancreatic lipomatosis, or to the lipomatous pseudohypertrophy of the muscles.

The often quite extensive intrapulmonary fibrosis due to various causes replacing the destroyed lung tissue is a result of cicatrization in the granulation tissue.

In addition, lymph congestion may play a role, giving after some time rise to acellular fibrosis, especially in perivascular, peribronchial and interlobular processes. The orientation of fibres and the genesis of the newly produced often very dense network of elastic fibres is governed partly by the tractile-tensile forces of the respiratory movements modified by the presence of pleural callus, rigidity of lung tissue and fixation of the hilum. According to the investigations of KURUCZ [27], in part of the cases pleural fibrosis is closely correlated with the perilobular fibrosis of the parenchyma. Pleural adhesions are responsible for the widest variety of tractile forces in the lung and the compact, islet-like fibrosis next the cavities is irregular, whereas in farther regions the fibres are running parallel and are oriented radially.

According to BRAUN et al. [3], ossification of the bronchial cartilage is a very rare occurrence in normal subjects; in HAYEK's large material there was only one single case of enchondral ossification of the bronchial cartilage in the otherwise normal lung of a young male. LOESCH [29] could not find a single bronchial cartilage ossification among 120 autopsies of subjects from 22 to 82 years of age, though calcification was common. True ossification of the bronchial cartilage was observed for the first time in bronchiectasis by SIPPEL [38] and in a case of chronic pulmonary abscess by SATINSKY and KRON [36]. In spite of the text-books' mentioning bronchial cartilage ossification as a change frequently met with in chronic pulmonary processes, few such cases have been reported in the literature. According to TEUFL [41] ossification occurs when there is (i) young, more or less undifferentiated connective tissue; (ii) increased calcium content and calcium avidity and (iii) an adequate forming stimulus (this may be the most important factor). To these, KERNAU [19] added mechanical stimulation: traction and pressure. The mechanics of the breathing lung, the distant action of the various pathological changes, pleural adhesions, scar formations on the intact parenchyma were studied in great detail by PARODI [33], DUMAREST and LEFÈVRE [10]. Among the internal forces active within the lung, the traction exerted by the respiratory movements, the weight, fixation and own weight of the lung are important. These forces act differently in the various directions, because the lung is composed of small parts and contains elements of different elasticity. These patterns are even more complicated when the lung is filled, for example with inflammatory exudate. Complex tractile forces originate from pleural adhesions of which there are three determinants, *viz.* the lower vertical dominant, which is the downward traction by the movements of the diaphragm; the upper vertical dominant, a traction acting upwards as a result of the respiratory movements of the superior thoracic muscles; and the lateral vertical dominant, starting from the area of maximum costal excursion during respiraton. We found ossification of the bronchial cartilage exclusively in such pulmonary areas in which pleural adhesions were present. The site of the ossification seems to be governed by certain laws. The cartilage facing the apex in the bronchi of the upper lobe fixed by a thick pleural callus was the commonest site. In such cases the lower vertical dominant, downward traction by the diaphragm, acts strongly on the lung fixed by the pleural callus and suspended at the uppermost part of the chest. In our opinion, ossification in the bronchial cartilage takes place in the centre of gravity. The bronchial cartilage is fixed perilobularly and peribronchially by thick scars to the pleural callus, so that instead of its physiological role it acts as a fix point of suspension, exposed to strong tractile-tensile forces. This task is too much for the cartilage; just as the muscles have their site of insertion not in cartilage but on bone, and on the most massive part of the bone, on the tuberosity, the bronchial cartilage is also transformed into

a firm, hard bone tissue to meet the requirements of its new function. From the site of ossification we could often reconstruct the mechanism of the various main vectorial forces, vertical dominants. In most cases ossification resulted in the part of the bronchial cartilage facing the pleural callus; for example, in a bronchiectasic lower lobe mostly in the area facing the adhesion. In other instances the direction of ossification differed from that of the main vertical dominants. In such cases the extensive intrapulmonary fibrosis made the pattern of forces even more complex. It would take lengthy calculations to analyse the vectorial components involved in such processes. It is known from ROUX' [35] doctrines concerning functional adaptation that the shape and function of the tissues are closely interrelated in the gross, microscopic and molecular dimensions alike. The structure (shape) of the tissues corresponds to the mechanical forces to which they are exposed: function and an increase of function carries with it an adaptative increase of the shape. The change in shape associated with functional adaptation is quantitative in nature, consisting of an increase or decrease in the amount of the tissue elements in response to an intensification or weakening of the usual stimulation, without involvement of a new kind of effect. If a different new biological action acts on the tissue, this will differentiate in a new direction, in a direction formerly unexistent in that area of the organism. This process, the existence of which in animals has been proved in numerous experiments by KROMPECHER [22, 23, 24, 25, 26], is termed neodifferentiation. KROMPECHER experimented first with connective tissue elements and by means of stimuli succeeded in inducing formation of locally new collagen fibrous connective tissue, hyaline cartilage, tendon, fibrous cartilage, elastic fibres, articular surface, completely new joints, synovial tufts, special epithelial differentiation, etc. On the basis of these results have been outlined the process of new tissue formation and the causal relationships presumably involved. In the case of cartilage formation the importance of bradytrophism has been emphasized.

In our material severe vascular changes were demonstrable in the bronchial wall. The increasing vascular obstruction and the bacterium toxins interfered with the metabolism of the bronchial wall and the cartilage in it. Fine granules of calcium had been precipitated into the hypoxic, bradytrophic, degenerating cartilage. The calcified, perishing cartilaginous tissue was then replaced by non-differentiated connective tissue (Fig. 8). Corresponding to the new type of exposure and by using up the precipitated calcium, lamellar and spongious bone is then formed, with fatty bone marrow in some and haematopoietic marrow in other areas. Ossification is restricted almost exclusively to those areas of the bronchial cartilage in which the mechanical stimulation — traction — takes the strongest effect. It is clearly visible in Fig. 11 that the elastic fibres have increased in number and diameter around the ossified areas. According to KROMPECHER, LELKES and KARMAZSIN [28], the above described

elastic fibre formation is also a result of exposure to a new formative stimulus. A proof of the importance of mechanical stimuli in bronchial cartilage ossification was in our material the frequency of ossification in the bronchiectasic lungs (40 to 50 per cent). Intrapulmonary cicatrization (tuberculous scars, carnification, induration) enhance the development of bronchiectasis; they may give rise to new tractile and tensile forces within the lung.

The clinical significance of bronchial cartilage ossification is that it further deteriorates bronchiectasis, since the ossified cartilage is no longer capable of fulfilling the task allotted to it. These late complications call for an up-to-date and effective treatment of bronchitis, lest the condition become chronic and remain a permanent source of peribronchitis, bronchopneumonia and accompanying pleuritis. Peribronchial scars, the induration following bronchopneumonia and the pleural adhesions may produce bronchiectasis. Thus arises a vicious circle giving ultimately rise to cavities, acute and chronic abscesses.

The above observations may prove helpful for the thoracic surgeon in judging his cases. In our opinion it were for example justified to resect carnified lung areas also because their presence might lead to bronchiectasis in the adjacent lung tissue. Finally our observations may offer an explanation for the development of postoperative bronchiectasis in cases in which recovery was associated with the formation of extensive pleural adhesions.

Summary

A histological examination of 285 surgically resected lung specimens and the lungs from cadavers of 38 subjects has been made in order to establish the effects of various pulmonary diseases on the blood vessels, connective tissue, smooth muscle and bronchi of the lungs. It has been found that the different pathological processes usually produce identical changes in the pulmonary elements. Among these, ossification of bronchial cartilage exceeds the concept of quantitative adaptation and meets the requirements of neodifferentiation. Ossification of the bronchial cartilage was observed in 27 per cent of all the cases and in 40 to 50 per cent of the cases with bronchiectasis. New mechanical forces active on the area in question as a result of adhesions are thought to be responsible for the ossification. The course and assumed relationships of bronchial cartilage ossification have been outlined in the light of KROMPECHER's experiments for influencing tissue differentiation.

REFERENCES

1. ABELLO, J.: 1956 Los bronquios de los lobulos resecados en enfermedades tuberculosos. Estudio histopoatologico. (Enfermedades del Torax 519, 353.) — 2. ANDREWS, N. C., PRATT, P. C., GILMON, R. A., KLASSEN, K. P.: 1956. The Correlation of the Pathology in the Resected Bronchus with Postoperative Complications in Pulmonary Tuberculosis. (Amer. Rev. Tuberc. 74, 874.) — 3. BRAUN, H., DHOM, G.: 1954. Verknöcherung des Tracheo-Bronchialskelets mit tödlichem Ausgang.) (klin. Med. 151, 355).— 4. BRENNE, O.: 1935. Pathology of the Vessels of the Pulmonary Circulation. (Arch. intern. Med. 56, 1189.) — 5. CHARR, R., SAVACOOL, J. W.: 1940. Changes in the Walls of Tuberculous Pulmonary Cavities. (Arch. Path. [Chicago] 30, 1159.) — 6. COURNAND, A.: 1950. Some Aspects of Pulmonary Circulation in Normal Man and in Chronic Cardiopulmonary Diseases. (Circulation, [N. Y.] 2, 641.) — 7. COURNAND, A., RILEY, R. L., HUMMELSTEIN, A., AUSTRIAN, R.: 1950. Pulmonary Circulation

and Alveolar Ventilation Perfusion After Pneumectomy. (J. thorac. Surg. 19, 80.) — 8. DAVIES, H. M.: 1933. Pulmonary Tuberculosis: Medical and Surgical Treatment (Cassell, London 1933). — 9. DENST, J., HURST, A., DRESSLER, S. H.: 1951. A Histologic Study of the Blood Vessels in Surgically Resected Tuberculosis of the Lungs. (Amer. Rev. Tuberc. 64, 489). — 10. DUBAREST, F., LEFÈVRE, P.: 1933. Intérêt de l'étude mécanique du poumon en physiologie. (Presse méd. 41, 839.) — 11. DRESSLER, S. H., SLOUNIM, N. B., BALCHUM, O. J., BRONFIN, G. J., RAVIN, A.: 1952. The Effect of Breathing 100 per cent Oxygen on the Pulmonary Arterial Pressure in Patients with Pulmonary Tuberculosis and Mitral Stenosis. (J. clin. Invest. 31, 807.) — 12. VON EULER, U. S., LILJESTRAND, G.: 1946. Observations on the Pulmonary Arterial Pressure in Cat (Acta physiol. scand. 12, 301). — 13. FÖLDES, I.: 1954. Vascular Changes in Experimental Tuberculosis of Guinea Pigs. (Acta morph. hung. Suppl. 4, 15.) — 14. GREVER, E. F., NEUBERGER, K. T., RUTLEDGE, E. K.: 1951. Atypical Pulmonary Inflammatory Reactions. (Dis. Chest 19, 325.) — 15. GOLDBERG, B.: 1942. Clinical Tuberculosis. (Davis, Philadelphia 1942. Vol. 1.) — 16. HAYEK, H. V.: 1953. Die menschliche Lunge (Springer, Wien). — 17. HURST, A., DRESSLER, S. J., DENST, J.: 1953. The Relationship between Pathological Changes in Blood Vessels in Resected Lobes and Lungs as Correlated with Pulmonary Pressure Changes Recorded during Cardiac Catheterisation. (Dis. Chest 24/1, 41.) — 18. KAYNE, G. G., PAGEL, W., O'SHAUGHNESSY, L.: 1948. Pulmonary Tuberculosis. (Oxford Medical Publications.) — 19. KERNAU, T.: 1936. Beitrag zur Kenntnis vielfacher Knochenbildungen in der Lunge. (Frankfurt. Z. Path. 50, 213). — 20. KITAMURE, E.: 1907. Über secundäre Knochenbildung in der Lunge. (Virchows Arch. path. Anat. 190, 163.) — 21. KLEBS, A. C.: 1909. Tuberculosis. (Appleton, New York.) — 22. KROMPECHER, ST.: 1928. Die Entwicklung der elastischen Elemente der Arterienwand. (Z. Anat. Entw. Gesch. 85, 704.) — 23. KROMPECHER, ST.: 1935. Experimentelle Beeinflussung der Art der Knochenbildung durch mechanische Einwirkungen. (Verh. dtsch. anat. Ges. 138). — 24. KROMPECHER, ST.: 1947. The Effect of Adequate Physical Influences on Cell Differentiation in Mammals and in Man during Regeneration. (Proceedings of the 6th International Congress of Experimental Cytology, p. 522.) — 25. KROMPECHER, ST.: 1954. Development of the Connective Tissue and its Capacity of Transformation. (Acta morph. hung. 4, 113.) — 26. KROMPECHER, ST.: 1958. Die qualitative Adaptation der Gewebe. (Z. mikr.-anat. Forsch. 64, 50.) — 27. KURUCZ, J.: 1959. Adatok a kaverna környezetének pathologiájához. Thesis, Budapest. — 28. LELKES, GY., KARMAZSIN, L.: 1955. Development of Elastic Elements in Tissue Cultures. (Acta morph. hung. 5, 149.) — 29. LOESCH, W.: 1936. Über Verkalkung in den Knorpeln der kleinen Bronchien. (Beitr. path. Anat. 97, 192.) — 30. MENDELSOHN, H. J., ZIMMERMANN, H. A.: 1950. A Study of Pulmonary Hemodynamics During Pulmonary Resection. (J. thorac. Surg. 20, 366.) — 31. MOTLEY, H. L., COURNAND, A., WERKO, L., HUMMELSTEIN, A., DRESDALE, D.: 1947. The Influence of Short Periods of Induced Acute Anoxia Upon Pulmonary Artery Pressure in Man. (Amer. J. Physiol. 1950, 315.) — 32. OLSON, D. F., JONES, F. S., ANGEVINE, D. M.: 1953. Bronchial Diseases in Lungs Resected for Pulmonary Tuberculosis (Amer. Rev. Tuberc. 68, 657). — 33. PARODI, F.: 1933. La mécanique pulmonaire (Masson, Paris). — 34. PINNER, M.: 1945. Pulmonary Tuberculosis in the Adult: its Fundamental Aspects. (Thomas, Springfield.) — 35. ROUX, W.: 1895. Gesammelte Abhandlungen über Entwicklungsmechanik der Organismen. (Engelman, Leipzig.) — 36. SATINSKY, V. P., KRON, S. D.: 1955. Heteroplastic Ossification in Chronic Lung Abscess. (dis. Chest 27, 196). — 37. SCHMIDTMANN, M., SAUER, R., LIEBALDT, G., DICK, H.: 1952. Pathologische-anatomische Studien an der operativ entfernten Lunge. (Virchows Arch. path. Anat. 322, 601.) — 38. SIPPEL, P.: 1911. Über Knochenbildung in Bronchiektasen. (Frankfurt, Z. Path. 6, 461.) — 39. SWEANNY, H. C., SEILER, H. H.: 1956. The Pathology and Bacteriology of Resected Lesions in Pulmonary Tuberculosis. (Dis. Chest 29, 3.) — 40. TERPLAN, K.: 1945. Anatomical Studies on Human Tuberculosis. (Amer. Rev. Tuberc. 51, 321.) — 41. TEULF, R.: 1936. Intraalveoläre verästelte Knochenbildungen in der Lunge. (Frankfurt. Z. Path. 50, 326.) — 42. VAN VLEET, M. E., EDWARDS, J. E.: 1954. Histologic Study of Intrapulmonary Vessels in Tuberculosis. (Arch. Path. [Chicago] 58, 168.)

ДАННЫЕ К ПАТОЛОГИЧЕСКОЙ АНАТОМИИ ОПЕРАТИВНО УДАЛЕННЫХ ЛЕГКИХ

Ф. ТОТ

На 285 иссеченных частях легких и на легких лиц, умерших вследствие различных легочных заболеваний, проводились патологоанатомическое исследования. Исследовалось влияние различных легочных заболеваний на кровеносные сосуды, соединительную ткань, гладкие мышцы и на бронхи легких. Было установлено, что различные

патологические процессы в большинстве случаев вызывают на вышеуказанных элементах легочной ткани одинаковую реакцию, одинаковые изменения. Среди этих изменений окостенение бронхиального хряща выходит за пределы понятия количественной адаптации, и исчерпывает критерии новой дифференциации. В 27% случаев удалось выявить окостенение бронхиального хряща, а при расширении бронхов даже в значительно большем проценте, а именно в 40 или же 50% случаев. За окостенение бронхиального хряща ответственным следует считат ь новые на данном месте, обусловленные сращениями механические воздействия. Течение и предположительные взаимосвязи окостенения бронхиального хряща описываются на основании результатов, достигнутых Кромпехером в области экспериментального влияния на дифференциацию тканей.

BEITRÄGE ZUR PATHOHISTOLOGIE DER OPERATIV ENTFERNTEN LUNGENTEILE

F. TÓTH

An 285 resezierten Lungenteilen und der Lunge von 39 infolge verschiedener Lungenerkrankungen verstorbenen Personen wurden pathohistologische Untersuchungen zur Klärung der Frage vorgenommen, welche Wirkung die verschiedenen Lungenerkrankungen auf die Gefäße, das Bindegewebe, die glatte Muskulatur und die Bronchien ausüben. Es wurde festgestellt, daß die verschiedenen Krankheitsprozesse in den meisten Fällen die gleichen Reaktionen, die gleichen Veränderungen der obenangeführten Lungengewebselemente hervorgerufen hatten. Unter diesen Veränderungen überschreitet die Verknöcherung des Bronchenknorpels den Begriff der quantitativen Adaptation und erschöpft die Kriterien der Neodifferezierung. In 27% der Fälle gelang es eine Verknöcherung des Bronchusknorpels nachzuweisen. Bei Bronchiektasie sogar in einem viel höheren Prozentsatz, und zwar in 40, bzw. 50% der Fälle. Für die Verknöcherung des Bronchusknorpels sind an der betreffenden Stelle neue, durch Verwachsungen bedingte mechanische Einwirkungen verantwortlich. Der Verlauf und die vermutlichen Zusammenhänge der Verknöcherung des Bronchusknorpels werden auf Grund der von KROMPECHER bei der experimentellen Beeinflussung der Gewebsdifferenzierung erzielten Ergebnisse skizziert.

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