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RELATIONSHIP OF HAEMODYNAMICS AND CHANGES OF PULMONARY VESSELS IN MITRAL STENOSIS

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It has been known in the 18th century already (ROKITANSKY, ANDRAL) that, independent of an arteriosclerosis in the greater circulation, signs of sclerosis may be detectable in pulmonary vessels. As early as 1851, BAMBERGER pointed out the frequent association of pulmonary sclerosis and mitral stenosis. According to POSSELT, the two conditions would occur together in about 40 per cent of cases.

It was however, only by the end of the 19th century that primary and secondary pulmonary sclerosis have been distinguished. While there are no detectable causes either in the lung, or in any other organs in primary pulmonary sclerosis, the secondary sclerosis of pulmonary vessels always arises in consequence of some other pathological condition. KLOB, WOLFRAM, ROMBERG and AUST are the authors usually credited with the description of primary pulmonary sclerosis. The grave form of the condition, with cyanosis and dyspnoea, was described in 1901 by AYERZA. Some authors attributed a significant role to syphilis in the aetiology of „AYERZA'S disease", while others (PAPPENHEIMER and VON GLAHN, CHIARI and others) were in favour of a rheumatic aetiology. Detailed studies of pathological changes in pulmonary vessels have been made by Ageitsenko and Maximovich in connection with rheumatic pneumonia, and by BALOGH and CORTEN in pneumonia due to grippe.

The pathogenesis of secondary pulmonary sclerosis has been ascribed to pulmonary hypertension (BRÜNNING, TORHORST), to a congenital weakness of pulmonary vessels (Mönckeberg), while others have suggested infectious or toxic factors. BRENNER has found sclerotic changes in the vessels of the lung not only in connection with heart affections but also in lung diseases (emphysema, bronchiectasis, fibrosis, tuberculosis, chronic abscess, tumour, etc.), and even in genuine hypertension and chronic glomerulonephritis. BRENNER also pointed out the similarity of pathological changes in the pulmonary artery to the arteriosclerosis of the great circulation. Rakov's combined mitral valvular disease in which there was ulcerative atheromatosis in the pulmonary artery, has confirmed BRENNER'S theory.

Pulmonary sclerosis may be produced experimentally by various methods, such as increasing the pressure in the pulmonary artery (MÜLLER, DAMMAN and HEAD), introducing microemboli into the pulmonary circulation, by thromboembolism of the small branches of the pulmonary artery (HARRISON, MUIRHEAD and MONTGOMERY, HEAR, GREEN, and STONER, LOEB and MYERS) by an allergic mechanism (RICH and GREGORY, CRAWFORD and NASSIM), and by administration of protein (BEREGI and FÖLDES).

Evidence obtained in recent years at heart operations have opened new ways for elucidating the pathogenesis of pulmonary sclerosis. Similarly to WELCH, JONSON and ZINNSER, ENTICKNAP, CLOWES, et al., we have also examined auricular appendages and specimens of lung removed at heart operation. Comparative studies have been made of haemodynamical relations and morphological changes detectable in pulmonary vessels.

Methods

At commissurotomy performed in patients with mitral stenosis at the Post-Graduate Surgical Clinic, the left auricular appendage and a piece of the size of a small bean from the lingula of the left lung were removed surgically. The specimens were then subjected to histological studies at the Institut of Pathological Anatomy and Experimental Cancer Research. The data relative to function, drived from clinical but particularly from haemodynamical examinations of the patient, as well as the histological appearance of the left auricular appendages, were compared with the morphological changes detectable in the pulmonary vessels of the lingula. There were a great number of controls to show that this procedure was justified and the histological pattern in the left pulmonary lingula truly reflects the morphological changes demonstrable in other areas of the lung. This view has been confirmed by a number of authors *(Enticknap*, etc.). The histology of the left auricular appendage has been studied by BIÖRCK et al. as well as by DECKER et al., and the morphological changes found were stated to be characteristic of the whole heart.

Haemodynamical studies

These were done in a specially equipped room, which could be darkened. The rate of oxygen intake and CO_2 output, as well as the respiratory quotient were estimated electrically, using a NOYONS diapherometer. The respiratory and pulse rates were recorded. By means of electrocardiography, and with a reasonable degree of accuracy, the diastolic rate per minute was determined.

One of the superficial antecubital veins was prepared under local anaesthesia and in intravascular catheter introduced. The catheter was attached to a manometer of the WIGGERS type, modified by us. (41) Under direct visual control facilitated by X-ray screening the catheter was introduced into the superior vena cava, then into the right atrium. From here, it was pushed into the right ventricle and, from there, into the pulmonary artery. The pressures prevailing in the pulmonary artery and in the cardiac cavities were determined, together with the so-called „pulmonary capillary pressure". Samples

of blood were examined for oxygen content in a VAN SLYKE apparatus and the values were related to 100 ml. of blood. The oxygen capacity of blood, as well as the oxygen saturation of arterial and mixed venous blood were also estimated.

The minute volume per 1 m^2. body surface, the so-called minute volume index, was calculated according to the FICK principle,

$$Mi = \frac{\left(\frac{O_2/min}{A - V} \cdot 100\right)}{S}$$

where Mi = minute volume index, litre/m^2
O_2 = oxygen uptake per minute
A—V = arterio-venous oxygen difference
S = body surface, in square meter.

Pulmonary vascular resistance, characteristic of the condition of the vessels in the lung was calculated according to the formula (Riley),

$$Pvr = \frac{Pm - Pcm}{Pvs} \cdot 1{,}332,$$

where Pvr = pulmonary vascular resistance dyn. sec. cm^{-5}
Pm = mean pressure in pulmonary artery mm. mercury
Pcm = mean pulmonary „capillary" pressure, mm. mercury
Pvs = second volume, litre.

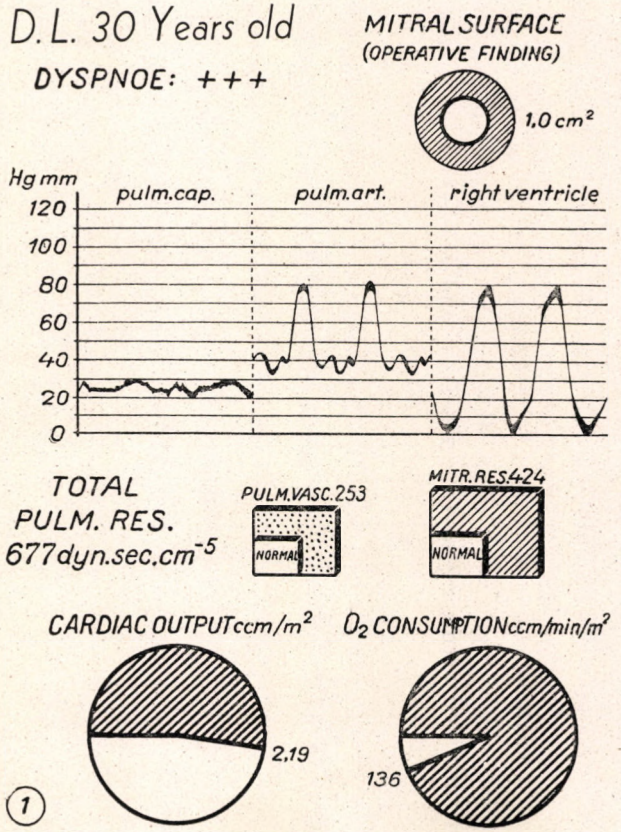
The work of the right ventricle agains the pressure of the pulmonary artery was calculated according to DEXTER, DOW and HAYNES,

$$WRV = \frac{(Mi.\ 1{,}055) \cdot (Pm.\ 13{,}6)}{1000}$$

where WRV = work of the right ventricle, mkg/hour.

Histological studies

After removal at operation, the excised parts of the heart and lung mentioned were fixed without delay in 4 per cent formaldehyde. The paraffine-imbedded sections were stained with haematoxylin-eosin or according to VAN GIESON, WEIGERT (for elastic elements) or MALLORY (for connective tissue elements). The lumen of each lung vessel in the range of 50 to 400 micron was measured according to KERNOHAN, ANDERSON and KEITH, related to the thickness of the vascular wall. Six measurements were made on each vessel and the mean value of 36 measurements derived from 6 vessels was calculated.



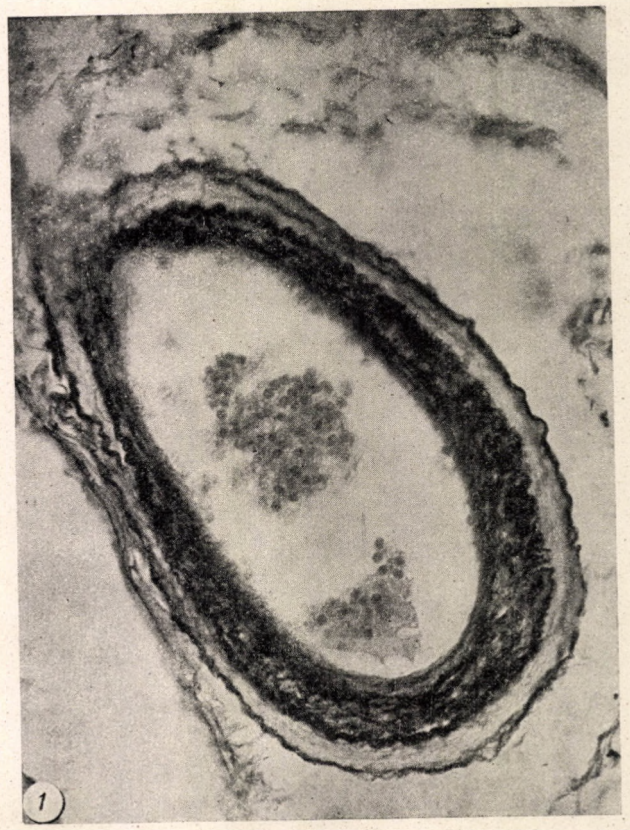
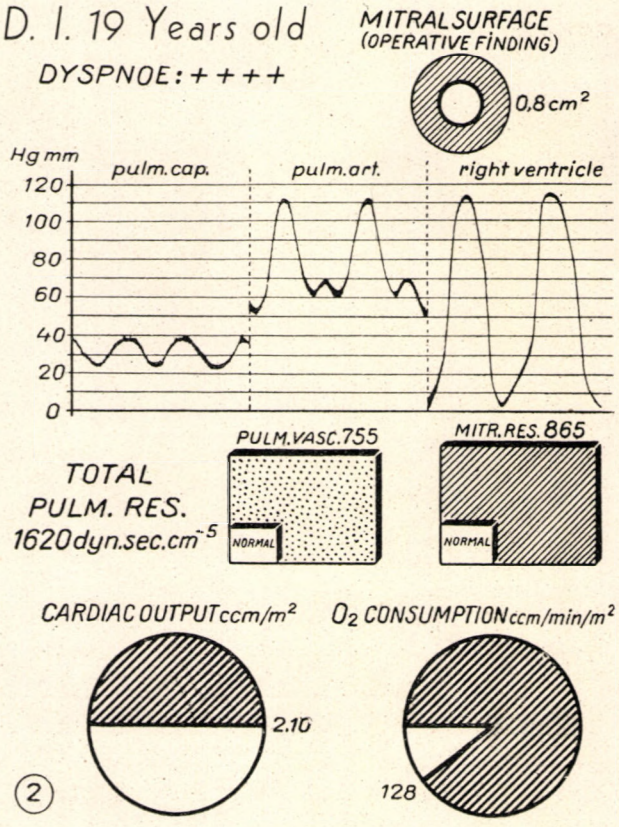


Fig. 1. D. L. 30 yrs. old, male. Hemodynamical relations are shown in table on the left. On the right: a small pulmonary vessel of the elastica type. There are moderate intima proliferation and elastosis. W/L index: 0,7 (Weigert's resorcin-fuchsin stain)



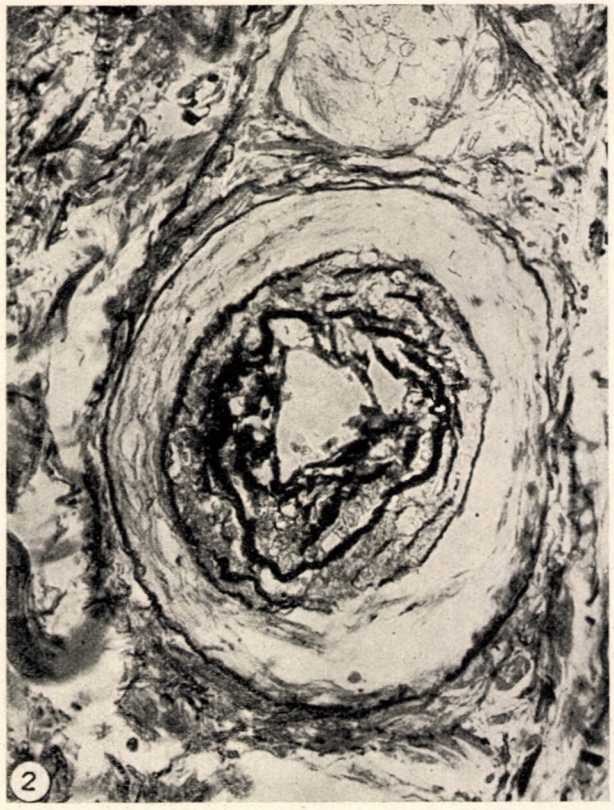
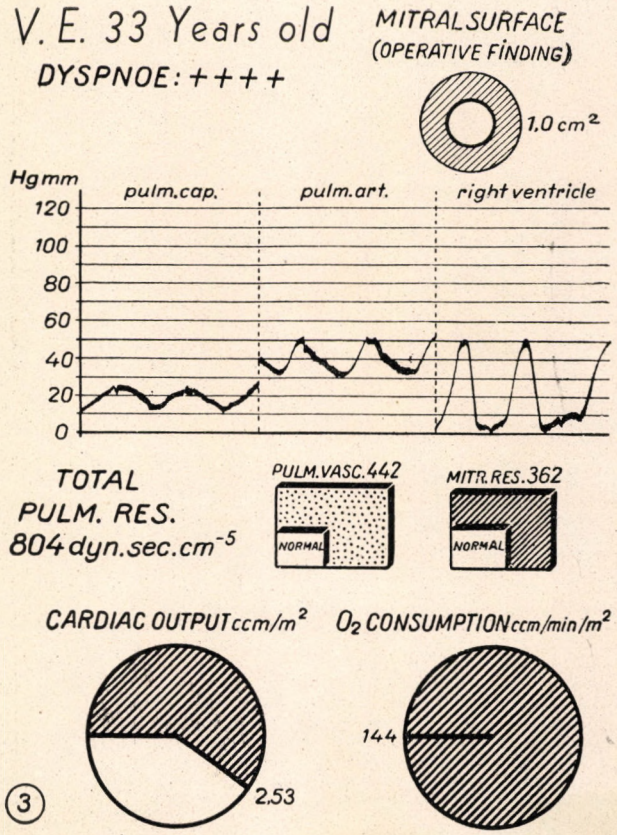


Fig. 2. D. I., 19 yrs., male. The hemodynamics are shown in the tabel on the left, distincly indicating the considerable rise in pulmonary pressure. On the right a small lung vessel with thickened wall can be seen. In the markedly widened intima elastica hypertrophy similar to lamellar elastosis is visible: the lumen of the artery is narrow. W/L index: 0,9 (Weigert's resorcin fuchsin stain)



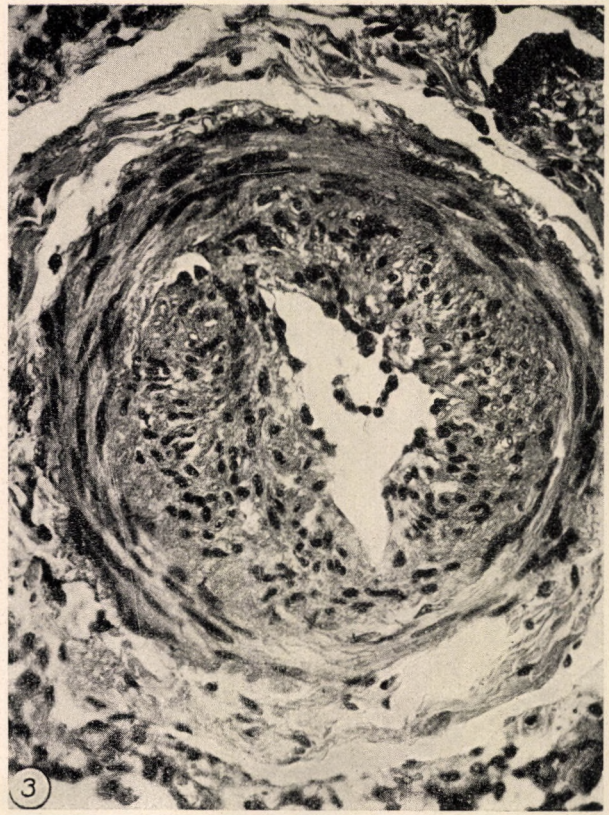


Fig. 3. V. E., 33 yrs., female. Combined vitium of 6 years duration. On the left: relations of pulmonary pressure. On the right small lung vessel with marked proliferation of intima. Media thinned W/L index: 1,3

Considering, that the severity of sclerosis in small vessels is directly proportional to tickness of the vascular wall and inversely to the diameter of the lumen, the vascular wall/lumen ratio appeared to be a reliable index of the severity of pulmonary sclerosis.

In 15 control cases thus index was below 0,3, thus each case with an index of 0,3 or above could be considered as pulmonary sclerosis. In grave cases the value of the index was above 1, in 1 case it was as high as 3, indicative of a considerable thickening of the vascular well and of narrowing of the lumen. The age of the patients was in the range of 15 to 50 years ; none of them had a blood pressure higher than 120 mm. Hg. The average duration of the illness was 7,3 years.

Changes in the branches of the pulmonary artery

These were most grave in the small pulmonary arteries of the muscular type. Connective tissue proliferation of the intima frequently narrowed the lumen. WEIGERT'S stain revealed the presence at sites in the intima of a circular network of fibres, resembling lamellar elastosis. In the vessels with marked intima-proliferation the media was thinned but vessels with excentrically

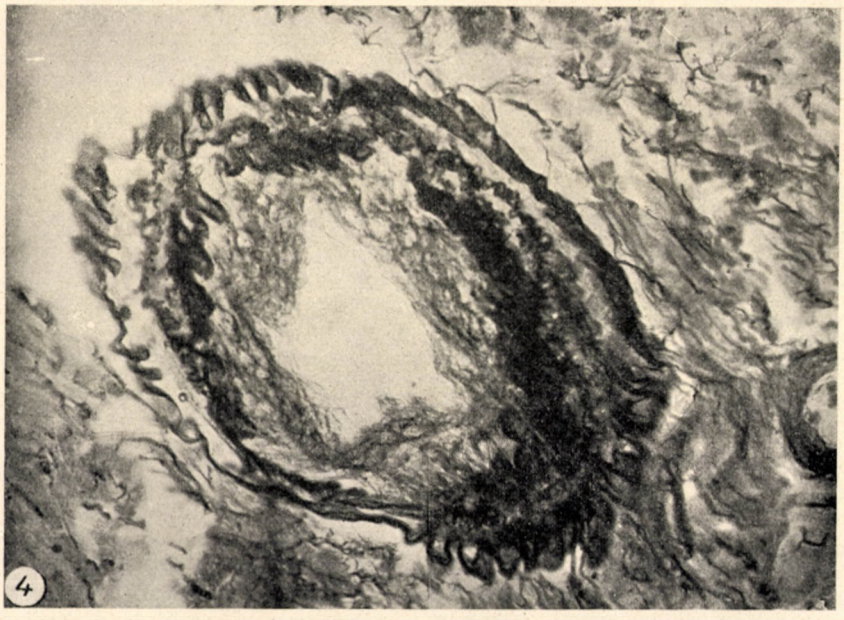


Fig. 4. 31 yrs old female with pulmonary hypertension. Proliferation of intima and hyperplasia of elastica in small pulmonary vessels. W/L index : 1,3

thickened wall showing muscular hyperplasia were also frequently found. A homogeneous staining and hyaline transformation of the media of vessels of the muscular type was a common finding. The wall of the larger, elastica-type vessels was also unusually thick. Marked accumulation of elastic fibres could be detected in the widened intima, as well as in the media. A similarly common finding was the proliferation of adventitial connective tissue. In some cases the veins were markedly distended, had thickened walls and stained more intensively with elastic tissue stains. In 15 of the 85 cases examined the capillary endothelium proliferated to such an extent that the lumina were practically obliterated ; in such cases the capillaries appeared as minute hyaline globules in the lung tissue. Of the cases examined, in 56 (66 per cent) there was grave pulmonary sclerosis, with an index higher than 0,5 (Figs 1 to 4).

The histological changes detectable in the specimens from F. M., a 18 years old girl, were different (Fig. 5). The lumen of the arterioles was practically obstructed by an excessive proliferation of intima. Both the intima and the thinned media showed invasion by large numbers of leucocytes and mononuclear cells. Also the perivascular connective tissue showed inflammatory infiltration. Fibrin staining revealed fibrin imbibition of the vascular walls, while Mallory's connective tissue stain resulted in the appearance of a bright red discoloration. There was extensive destruction of elastic fibres. On the whole, the histological appearance of the vessels resembled closely the changes usually encountered in rheumatic arteriitis.

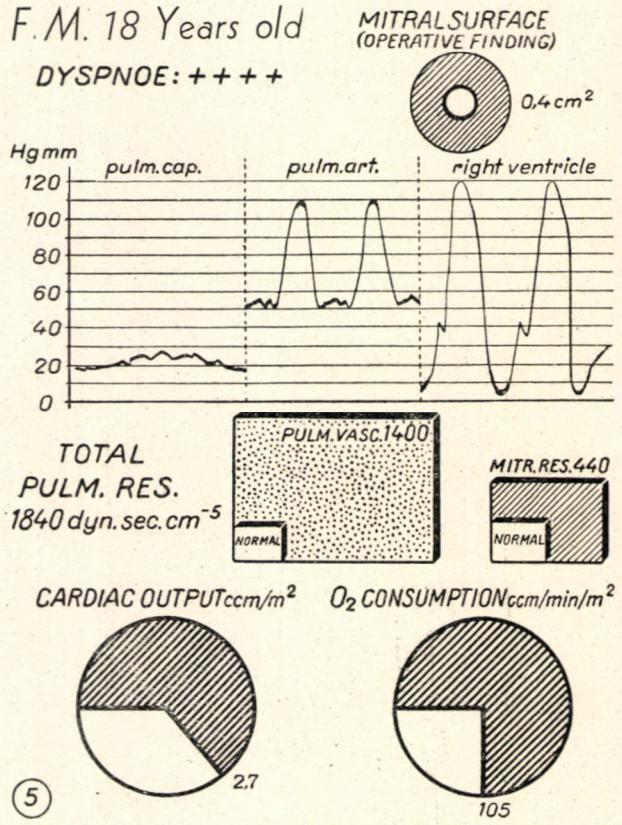
Other changes in the lungs

In 70 out the 85 cases was there histological evidence of emphysema. In 61 cases numerous heart failure cells were visible in the alveoli (induratio brunea pulmonis). Leucocytic-lymphocytic infiltration of the alveolar wall, interstitium, and, not infrequently, of peribronchial tissue, was detectable in 42 instances. Thickening and fibrosis of alveolar septa were found also in 42 cases. There was anthracosis in 21, haemorrhage in 5 cases.

Discussion

The haemodynamical and histological evidence derived from the above investigations have been used for making comparisons between functional data and morphological changes in the pulmonary vessels.

Considerable importance has been attributed to elevated blood pressure in the pathogenesis of arteriosclerosis. The presence of pulmonary sclerosis is important for both the cardiologist and the cardiac surgeon, since it must



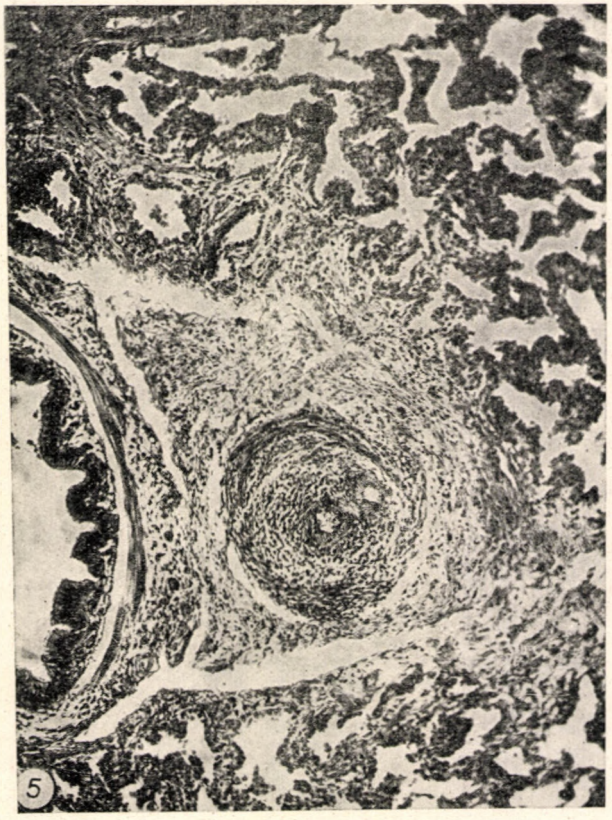


Fig. 5. F. M., 18 yrs., female. Rheuma of 10 years standing in history, operated for mitral stenosis. Relations of pulmonary pressure in table on the left. On the right: arteriitis rheumatica in small pulmonary vessels. W/L index: 4,1

be always considered when surgery is to be decided upon and may, to some extent, influence the outcome of a cardiac operation (LITTMANN).

We hawe compared the mean pulmonary pressure, as determined by intravascular catheterization, with the severity of histological changes detectable in minor branches of pulmonary artery. Fig. 6. illustrates the correlation of these two data; the mean pulmonary pressure in mm. Hg in shown on the abscissa, and the degree of pulmonary sclerosis on the ordinate. In order to exclude the modifying effect on the shape of the curve of the variability in the extent of mitral stenosis, only those cases have been considered in which the

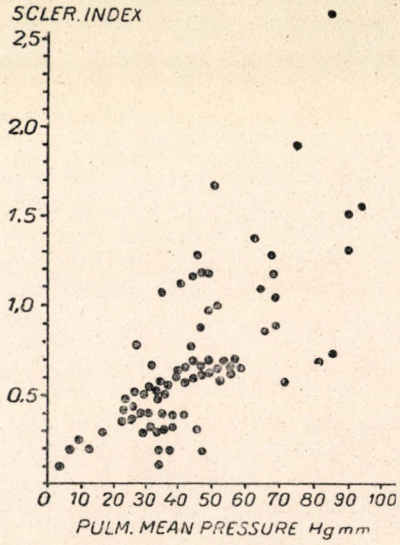


Fig. 6. It can be clearly seen that with the rise of mean pulmonary pressure (abscissa) there is a proportional increase in the degree of pulmonary arteriosclerosis (ordinate)

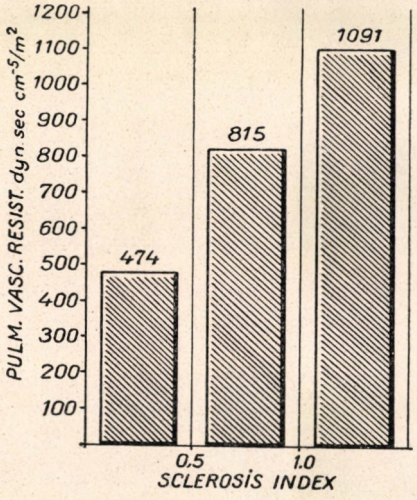


Fig. 7. The progression of pulmonary sclerosis is accompained by a simultaneous considerable increase of pulmonary vascular resistance. Abscissa: pulmonary changes grouped according to severity of changes: mildest: 0 to 0,5, moderate: 0,5 to 1, grave: 1. Ordinate: Mean pulmonary vascular resistance

diameter of the mitral ostium was in the range of 0,5 to 1,0 cm. As seen, along with the rise in pulmonary pressure there was a proportional increase in the severity of pulmonary sclerosis. In none of our patients was there a systemic pressure higher than 120 mm. Hg; this appears to indicate that the systemic pressure had no bearing on the development of pulmonary sclerosis, with the decisive role being played by pulmonary hypertension.

Comparing the index of pulmonary vascular resistance with the severity of pulmonary arteriosclerosis, it has been found that pulmonary vascular resist-

ance was the lowest in the group where vascular changes were the least marked, while it was the highest where the changes were the most severs (Fig. 7). Thus, aggravation of pulmonary sclerosis is accompanied by a considerable rise of pulmonary vascular resistance. About 10 per cent of the cases were exceptions to this rule, inasmuch as the increase in pulmonary vascular resistance was not associated with pulmonary arteriosclerosis of the anticipated severity. It is assumed as supported by data in the literature (Halmágyi et al.), that apart from the organic pulmonary vascular changes also other, functional factors may be involved.

The evidence obtained corfirm BRÜNNING'S theory dating back to 1901, and are in accordance with similar observations by ENTICKNAP, WELCH and associates. On the basis of our observations we are unable to accept the view put forward by CLOWES, HACKEL, MUELLER and GILLESPIE that the thicknes of the wall of pulmonary vessels would be independent from the pressure

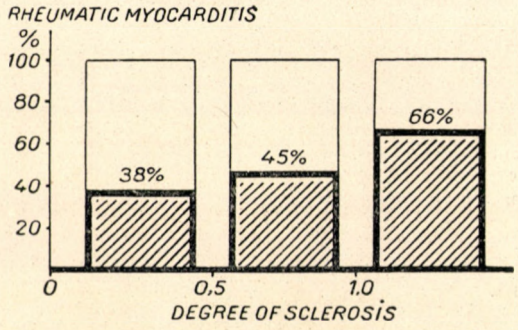


Fig. 8. The more severe the vascular changes, the higher the incidence of rheumatic myocarditis. (Abscissa : pulmonary vascular changes, grouped according to severity. Ordinate number of cases of rheumatic myocarditis)

prevailing in the pulmonary artery. We agree with ENTICKNAP, und are of the opinion that the histological changes of the pulmonary are consequential to pulmonary hypertension and that the changes, once developed, are greatly responsible for the increase in pulmonary vascular resistance.

In further studies the relationship between right ventricular plus-work and the degree of pulmonary sclerosis was examined in patients with mitral stenosis. It was found that the burden on the right ventricle owing to mitral stenosis was further increased by sclerosis.

Histological examination of the left auricular appendages removed at operation brought the following auricular finding. In the muscle and subendocardially, ASCHOFF—TALALAYEV nodes, or infiltrations strongly resembling them, could be detected in 40(50 per cent) of the 80 cases (in 5 cases no examination was made), although on the basis of careful clinical examinations they were all thought to be completely inactive from the point of view of

rheumatic infection on the basis of meticulous. As shown in Fig. 8. rheumatic myocarditis could be detected in 38 per cent of cases with minimal vascular changes and in 60 per cent of cases where pulmonary sclerosis was of marked severity. This parallelism appears to indicate, as it is emphasized by Soviet authors, that in the pathogenesis of pulmonary vascular changes rheumatic inflammation may also play some role.

We were unable to detect any relationship between pulmonary blood flow, oxigen uptake and severity of pulmonary sclerosis. As to the duration of illness, it could be stated that there are two form of pulmonary sclerosis associated with mitral stenosis : one is benign and develops slowly, while the other is malignant and progresses rapidly. In the latter case, very grave vascular lesions, or, in a few instances, even vascular necrosis, may develop within a very short period of time.

Summary

The left auricular appendage and a small part of the lingula of the left lung were removed from 85 patients operated upon for mitral stenosis (commissurotomy). Histological examination revealed the presence of pulmonary sclerosis in 66 per cent, and signs of a pathological condition most probably identical with rheumatic myocarditis in 50 per cent, of the specimens examined.

It has been found that neither the age of the patient, nor the systemic pressure, nor the duration of illness bear influence on the development of pulmonary arteriosclerosis. The severity of a developing pulmonary sclerosis is proportional to the rise of blood pressure in the pulmonary artery.

With the increase in severity of pulmonary vascular sclerosis, pulmonary vascular resistance increases in direct proportion. The disproportionately high vascular resistance found in some of our cases is attributed to functional factors.

In mitral stenosis the strain on the already overburdened right ventricle is considerably increased by the presence of pulmonary arteriosclerosis.

Pulmonary sclerosis is a disease of multiple aetiology ; it appears that rheumatic inflammation is also involved in the genesis of this condition.

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ВЗАИМОСВЯЗЬ МЕЖДУ ГЕМОДИНАМИЧЕСКИМИ УСЛОВИЯМИ И ИЗМЕНЕНИЯМИ ЛЕГОЧНЫХ СОСУДОВ ПРИ МИТРАЛЬНОМ СТЕНОЗЕ

Я. ЛАСЛО, И. ЛИТТМАНН, Ф. РОБИЧЕК и А. ТЕМЕШВАРИ

Авторы проводили коммисуротомию над 85 больными, страдающими митральным стенозом. Они удалили при этом кусок языка левого легкого, как и придаток левого предсердия, и обнаружили при гистологическом исследовании в легочных сосудах в 64% случаев легочный склероз, а в придатке предсердия в 50% всех случаев по всей вероятности ревматический миокардит.

Согласно исследованиям авторов, развивающийся в легочной артерии артериосклероз не зависит ни от возраста больного, ни от кровяного давления в большом кругу

кровообращения, и не пропорционален длительности заболевания. Развивающийся легочный склероз пропорционален повышению кровяного давления в легочной артерии.

По мере прогрессии артериосклероза легочных сосудов повышается сопротивление в малом кругу кровообращения; в одной части случаев авторы сводят непропорциональное по сравнению с изменениями сосудов большое сопротивление в большом кругу кровообращения к функциональным факторам.

При митральном стенозе в развитии перенагрузки правого желудочка склероз легких играет также значительную роль.

Склероз легких является полиэтиологическим заболеванием: в его патогенезе, по всей вероятности, играет роль также и ревматическое воспаление.

ZUSAMMENHANG ZWISCHEN DEN HÄMODYNAMISCHEN VERHÄLTNISSEN UND DEN VERÄNDERUNGEN DER LUNGENGEFÄSSE BEI MITRALSTENOSE

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Bei 85 an Mitralstenose Erkrankten wurden nach Entfernung des linken Herzohres und eines Teiles der Lingula der linken Lunge durch Commissurotomie, bei 64% histologisch in den Lungengefässen pulmonale Sklerose und bei 50% im Herzohr eine, allem Anschein nach rheumatische Myocarditis gefunden.

Nach den Untersuchungen der Autoren hängt die in der Arteria pulmonalis sich entwickelnde Arteriosklerose weder mit dem Alter des Patienten, noch mit dem Blutdruck im grossen Kreislauf zusammen und sie ist nicht proportional zur Dauer des Bestehens der Erkrankung. Das Ausmass der sich entwickelnden pulmonalen Sklerose ist proportional der Steigerung des Blutdrucks in der Arteria pulmonalis.

Proportional mit der Zunahme der Arteriosklerose der Lungengefässe, wächst der Widerstand im kleinen Blutkreislauf: bei einem Teil der beobachteten Fälle halten die Autoren für dem im Verhältnis zu den Blutgefässveränderungen unverhältnismässig grossen Widerstand im grossen Kreislauf funktionelle Faktoren für verantwortlich.

Bei Mitralstenose spielt in der Belastung der rechten Herzkammer die pulmonale Sklerose eine bedeutende Rolle.

Die pulmonale Sklerose ist eine polyätiologische Erkrankung: wahrscheinlich spielt in ihrer Pathogenese die rheumatische Entzündung gleichfalls eine Rolle.

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