Tuberculosis and Cardiopulmonary Failure*

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Clinical observations on the effect of chronic tuberculosis on the pulmonary circulation are of no recent origin. The Prague School of Physicians was well aware of its significance in clinical practice over 50 years ago. Professor Jaroslav Jedlicka, one of the few direct pupils of Thomayer, can remember the emphasis placed by this great physician on the signs of impending cardiac failure in phthisis. He considered an accentuated second sound over the pulmonary area in chronic tuberculosis to be a bad omen. In a contribution to the discussion on the paper of Thomayer at the Congress of Naturalists and Czech Doctors in 1914, Professor Maixner spoke of an accentuated diastolic sound over the pulmonary artery as a sign of dilatation and rigidity of this artery. The pupils of Thomayer and Maixner developed this observation still further.

Recently, Herles and Widimsky pointed to the increased incidence of chronic and acute cor pulmonale in the post-war years in the face of a continuous fall in mortality from tuberculosis. An analysis of the cause of death in cases of tuberculosis found at necropsy at Bulovka and Thomayer Hospitals in Prague, revealed a definite relationship between cardiopulmonary complications and the new methods of treating pulmonary tuberculosis. The decrease in mortality from tuberculosis as a result of the use of bacteriostatic drugs has, as a consequence, an increase in chronic forms, which may lead to the development of cor pulmonale. Radical surgery is also a major factor in the increased incidence of acute right-sided cardiac failure over that which was recorded in the era of treatment by pneumothorax and thoracoplasty.

Zak and Stejskal studied the pathologic picture on necropsy of 1357 cases of tuberculosis in persons over 40 years of age.

On the basis of these findings, pathologists have requested that cor pulmonale be considered a complication of extensive cavernous and fibrotic pulmonary tuberculosis.

We have made an investigation of cardiopulmonary failure in tuberculosis at the University Clinic for Tuberculosis in Prague over the past 12 years (1946-1957).

Figure 1 shows that duration of tuberculosis before admission to the clinic has increased during the past ten years from 2.8 years in 1947 to 7.5 years in 1957. The duration of the disease has, therefore, increased almost threefold. This factor, in concurrence with the observation of Herles and Widimsky, is of no small importance in the increase in cardiopulmonary failure. Chronic cases are admitted repeatedly to the clinic and there is a decrease in patients admitted for the first time and newly diagnosed.

From the total of 6,854 patients with tuberculosis admitted over the past 12 years, 201 died at the clinic, i.e. 2.9 per cent as against 5 per cent in the last three years of the war.

*University Clinic for Tuberculosis, Director, Prof. Jaroslav Jedlicka; dedicated to Prof. Jedlicka on his seventieth birthday, July 30, 1961.
Figure 2 shows the average age of patients who died at the clinic. This has increased from 37 years in 1947 to 57.5 years in 1957. Death from tuberculosis at the clinic, therefore, occurs mainly in older people. This is the second factor increasing the incidence of cardiopulmonary failure. This has also been confirmed by Söderholm.

Figure 3 gives a survey of the forms of pulmonary tuberculosis in patients who died at the clinic. Chronic cavernous tuberculosis was present in 75 per cent. This is a greater percentage than during the war, when the figure was 70 per cent. Acute pneumonic tuberculosis, miliary tuberculosis and meningitis appear only sporadically in recent years as against an increased incidence in the years 1948 to 1950. From 1954, however, there has been an increase in retractive fibrotic tuberculosis. This is the third factor which may contribute to the increased incidence of cardiopulmonary failure.

Chronic cavernous tuberculosis, however, remains a basic question in the problem of tuberculosis and also holds first place in the causes of cardiopulmonary failure as may be seen from Figure 3. Spread by aspiration and the substitution of the tissue, defects by scarring take place. Retraction occurs, for the most part, in the presence of persistent cavities and the remaining areas of the lung undergo emphysematous changes.

Unilateral post-tuberculous pneumosclerosis with displacement of the intrathoracic organs in a horizontal direction is rare in our series. Furthermore, it does not profoundly affect the pulmonary circulation, in contradistinction to cranial displacement of the root of the lung. The latter change was present in more than 10 per cent of cases in this series. In this so-called subclavicular hilum of the lung, distortion of the main branches of the pulmonary artery is found. They become deranged and

**Duration of tuberculosis in patients admitted to the Chest Clinic, Prague in 1947 and 1957.**

![Figure 1](image-url)
narrowed, causing considerable resistance to blood flow, and are a source of vascular murmurs, which are now being studied at the clinic.\textsuperscript{9} The existence of these murmurs has also been confirmed by Curti et al. in 1955, according to Mira.\textsuperscript{10}

Prolonged treatment with bacteriostatic drugs leads to the cleaning up of even large cavities. The thickness of the internal necrotic layer of the cavity is gradually reduced until only a thin-walled sac is left, which looks like a cyst or emphysematous bulla. The cavities are then spoken of as "open healed cavities." In the era before chemotherapy they were observed by Auerbach\textsuperscript{11} in only 0.2 per cent of necropsies. On examining excised parts of lung in recent years, they have been found in open healed cavities up to 10 per cent.

Thus, there is an increase in the group of patients with clinically healed tuberculosis with persistent tissue defects, which often, because of their considerable size embarrass the pulmonary circulation, at least to the same extent as cavities healed by scarring.

Figure 3 also shows the number of cases in which clinical signs of cardiopulmonary failure were found and, in the last column, cardiopulmonary failure confirmed at necropsy as the cause of death.

More than half of the patients had clinical signs of cardiac failure before death. Cor pulmonale has been found to be the cause of death at necropsy with increasing frequency in the last four years. It was the cause of death in 23 per cent (46 patients).

Figure 4 gives an analysis of the individual clinical signs of cardiopulmonary failure and the corresponding necropsy findings in 100 cases

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\caption{Average Age of Patients Dying from Tuberculosis and its Complications at the Chest Clinic, Prague, 1946-67}
\end{figure}
with adequate clinical, radiologic and pathologic documentation. All had chronic cavernous pulmonary tuberculosis.

Two-thirds of the patients had a resting pulse rate of over 100. Cyanosis, increased liver dullness and edema of the legs were present in a third of the patients.

In the postero-anterior x-ray film, enlargement of the heart shadow, particularly of the right half, was considered to be present in more than half of the patients.

Cranial displacement of the hilum with dilatation of the main branches of the pulmonary artery, confirmed by tomography, were present in almost a third of the patients. This finding was one of the most reliable radiologic criteria of hypertension of the lesser circulation, which leads finally to right-sided failure.

In the pathologic findings, kindly placed at our disposal by Professor J. Jedlicka, signs of chronic cor pulmonale were, of course, present much more frequently. Hypertrophy of the right ventricle was found in 65
per cent, dilatation in 85 and venous congestion of the liver and other organs in 84 per cent.

It is thus evident from the last figure that in two-thirds of the patients where cor pulmonale was found with certainty at necropsy, it was missed on routine clinical examination.

It remains to be seen to what extent incipient or obvious but hitherto compensated cor pulmonale, which Van Loo according to Söderholm considers to be present in 45 per cent of patients with advanced pulmonary tuberculosis, can be diagnosed clinically.

The diagnosis is dependent upon the finding of hypertension of the pulmonary artery. This can be reliably demonstrated by cardiac catheterization, if necessary with occlusion of the main branches of the pulmonary artery for a short period at rest and after exercise, according to the method of Carlens et al., in conjunction with hemodynamic investigation and the estimation of blood gases. These complicated investigations have been made in selected cases of pulmonary tuberculosis (Söderholm, Uggla, Widimsky et al.). They cannot be made routinely, but such investigations are a necessary starting point for elaboration of simple criteria for the early diagnosis of pulmonary hypertension.

Widimsky et al. have established the following simple criteria for the diagnosis of compensated chronic cor pulmonale.

III. Physical examination:

A. Signs of hypertension in the lesser circulation:

1) Palpable second sound over the pulmonary artery.

2) Accentuated second sound over the pulmonary artery with a first sound of equal intensity over the aorta and pulmonary artery.

3) Split second sound over the pulmonary artery with accentuation of its second (pulmonary) part.
4) Diastolic murmur over the pulmonary artery as an expression of functional insufficiency of the pulmonary valves in the presence of dilatation of the pulmonary artery (only in very advanced conditions).

5) Slapping first sound over the pulmonary artery.

6) High "a" wave in the phlebogram of the jugular vein.

B. Signs of hypertrophy of the right ventricle:

1) Expansive precordial pulsation.

2) Pulsion of the right ventricle below the xiphoid process.

II. Electrocardiogram:

Tracings must be taken from all unipolar chest and limb leads, where necessary with the addition of a lead from the right precordium. Both the direct and indirect signs of cor pulmonale must be taken into consideration.

III. Radiography of the heart:

It is necessary to make a comprehensive evaluation of several x-ray signs.

1) Heart shadow of normal or decreased size in the postero-anterior projection.

2) Relative increase in the curve of the right ventricle in the second oblique position.

3) Dilatation of the common trunk of the pulmonary artery in the first oblique position.

4) Dilatation of the main branches of the pulmonary artery in dorsoventral position.

5) Increased difference between width of shadow of main, segmental and subsegmental branches of the pulmonary artery.

6) Active pulsation of the hilar sector of the pulmonary arteries.

The radiologic findings are considered to be positive if at least three of the above signs are present.

IV. Auxiliary investigations:

Degree of hypoxemia, hematocrit, erythrocyte count, estimation of alkali reserve in the blood.

The prevention of cardiopulmonary failure in pulmonary tuberculosis consists of obviating the development of tuberculous cavities, or at least, of their rapid healing. As long as this aim is not attained, it is advisable to keep patients with chronic pulmonary tuberculosis under close supervision. This makes a correct assessment of their working capacity possible and prevents their being completely excluded from employment by overlooking signs of impending cardiopulmonary failure.

It is worth noting that the exact study of pulmonary hemodynamics has confirmed the value of simple examination methods in early diagnosis.

The dictum of Thomayer, according to Pelnar," with regard to cardiac cirrhosis of the liver, also holds true in this condition: "That it would be possible to avoid the development of such gross changes if we always made a thorough examination of our patients as a whole, without reference to what they are complaining of—and if the patients themselves comprehended the gravity of their symptoms in time."
SUMMARY

We have made an investigation of cardiopulmonary failure in tuberculosis over the years 1946-1957. Our experience demonstrates the increased incidence of cardio-pulmonary failure in the past war years in the face of a continuous fall in mortality from tuberculosis. The factors increasing this incidence are:

1. The duration of tuberculosis before admission to the clinic has increased almost three-fold in the past ten years—from 2.8 years in 1947 to 7.5 years in 1957.
2. The average age of patients who died at the clinic has increased from 37 to 57.5 years from 1947 to 1957.
3. There has been an increase in retractive fibrotic tuberculosis.
4. In two-thirds of the patients where cor pulmonale was found at necropsy, it was missed by routine clinical examination.

RESUMEN

Hemos llevado a cabo una investigación de la relación de la tuberculosis con la insuficiencia cardíaca durante los años 1946-1957. Nuestra experiencia demuestra que la incidencia de la insuficiencia cardíaca ha aumentado en los años de la postguerra frente a un descenso de la mortalidad por tuberculosis:

Los factores que hacen aumentar esta incidencia son:
1. La duración de la tuberculosis antes de la admisión a las clínicas ha aumentado casi tres veces en los pasados 10 años de 2.8 años en 1947 a 7.5 años en 1957.
2. La edad media de los enfermos que murieron en la clínica ha aumentado de 37 años a 57.5 años de 1947 a 1957.
3. Ha habido un aumento en la tuberculosis retractí.
4. En dos tercios de los enfermos de quienes se encontró cor pulmonale al examen postmortem, este padecimiento no se había diagnosticado por el examen clínico de rutina.

REFERENCES

Important Announcements

Notice of Annual Meeting

The 28th Annual Meeting of the American College of Chest Physicians will be held at the Morrison Hotel, Chicago, June 21-25, 1962. The American Medical Association will hold its annual meeting in Chicago, June 25-29.

Scientific Program

Preparations for the 1962 scientific program are now under way. Abstracts of papers for consideration must be received by the program committee prior to January 1, 1962. Please forward abstracts, in duplicate, 200 words or less, directly to the chairman of the committee, as follows:

JOSEPH W. PEBODY, JR., M.D., Chairman
Committee on Scientific Program, A.C.C.P.
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