Right Heart Failure After Thoracoplasty*

ALLAN HURST, M.D., F.C.C.P., and H. M. MAIER, M.D.

Denver, Colorado

From a study of the available literature it would appear that little attention has been paid to the question of cardio-pulmonary insufficiency after long-standing thoracoplasty. In the Scandinavian literature two cases are reported in which death occurred three and four years respectively after thoracoplasty with necropsy findings of distinct right cardiac hypertrophy and dilatation. In a recent article on chronic cor pulmonale one case is mentioned in which marked right ventricular hypertrophy was found at necropsy, nine years after a thoracoplasty. The authors state "undoubtedly, the question of whether or not long-standing thoracoplasty may result in a strain on the right side of the heart is a subject which deserves further study." We recently had occasion to observe two cases of cardio-pulmonary insufficiency after long-standing thoracoplasty.

CASE REPORTS

Case 1: I. C., White, female, 39 years old. Diagnosis of pulmonary tuberculosis in 1934. Pneumothorax on the right instituted in June 1936. Permanent phrenic paralysis performed in July 1936. Tuberculous empyema with bronchopleural fistula developed in 1939. Patient was admitted to the National Jewish Hospital and a thoracotomy followed by a three stage ten rib thoracoplasty and Schede procedure was performed. Vital capacity which was found to be 36 per cent before the operation was 26 per cent after surgical procedure. After discharge from the institution patient was seen in the Out-Patient Department at regular intervals. She presented no complaints except for slowly increasing dyspnea on exertion. In the last week of May 1946 she developed what seemed to be an upper respiratory infection with slight temperature elevation and nonproductive cough. There was severe increasing dyspnea despite routine treatment of the upper respiratory infection and she was re-admitted to the hospital. On examination she was found to be dyspneic, orthopneic and cyanotic. Tachycardia with a pulse rate up to 140 was present. Blood pressure which had been normal previously was elevated to 170 systolic and 108 diastolic. Slight distension of the neck veins was noted and tenderness to pressure was found over the right upper quadrant. X-ray film showed cardiac enlargement by comparison with previous films. EKG gave evidence of slight right axis deviation. Venous pressure studies were done and showed elevation to 21 and 24.5 cm. respectively on the right and left side with positive response to right upper quadrant pressure (Pasteur-Rondot test). She was placed on bedrest and given a full course of digitalization with a purified digitals

---

*From the National Jewish Hospital at Denver.
product. Her dyspnea subsided gradually with disappearance of the other chemical findings and she was discharged after three weeks. At the time of discharge, x-ray film showed reduction of her heart size, venous pressures were in normal range (R.A. 5 and L.A. 9) and the modified Pasteur-Rondot test was negative. She was given a maintenance dose of digitalis and advised to move to a lower altitude. According to reports received from her since, she has had no further episodes of cardiac decompensation and her exertional dyspnea has been reduced.

Case 2: P. M., White, female, 46 years old. Onset of pulmonary tuberculosis in 1922 with hemoptysis. Left pneumothorax was induced and permanent left phrenic paralysis performed in 1930. Because of evidence of cavity in the left upper lobe and positive sputum she was re-admitted in 1935 and a four rib thoracoplasty on the left was performed. She was followed in the Out-Patient Department and presented no complaints except gradually increasing dyspnea on exertion. In February 1946 she was seen in the Out-Patient Department with indefinite gastro-intestinal symptoms. Gall bladder and gastro-intestinal x-ray series were negative. Chest x-ray film showed definite cardiac dilatation; EKG, right axis deviation with signs of right ventricular strain. She was not seen again until August 1946 when she was first admitted to a general hospital in full congestive failure. She was digitalized and after a short stay was re-admitted to the National Jewish Hospital. On admission she was markedly dyspneic and cyanotic, with moist rales over the bases, liver enlargement and ankle edema. Venous pressures were: R.A. 12, L.A. 16½; right upper quadrant pressure resulted in venous pressures of R.A. 18, L.A. 23. EKG showed right ventricular strain and digitalis effect, x-ray film gave evidence of marked cardiac enlargement. Her digitalization was continued and oxygen was given by nasal catheter. Her condition improved with reduction of edema and weight. Venous pressures returned to normal, R.A. 6 and L.A. 9.5. When oxygen was discontinued, her edema recurred and despite low sodium-acid ash diet and intensive use of mercurial diuretics she showed increasing fluid retention. Oxygen was again begun and after a time she was discharged home where she continued on the same routine with no improvement in her condition. She died in congestive failure in October 1946. No necropsy was done.

Discussion and Comment

From a clinical point of view the two above described cases illustrate the importance of early recognition of the cardiac factor in patients developing cardio-pulmonary insufficiency in chronic pulmonary tuberculosis with permanent collapse procedures. The anoxemia caused by the ventilatory-respiratory insufficiency, results in dyspnea and cyanosis and the superimposed cardiac involvement can be recognized “early” only if attention is paid to “specific” cardiac signs of right heart failure. If only suggestive evidence is found, the patient should then be subjected to a thorough diagnostic work-up, including x-ray, electrocardiogram, and venous pressure studies. Our observations on these two and other cases of cardio-circulatory failure in chronic pulmonary disease have led us to endorse Spain and Handler’s² impression that routine cardiac treatment, especially digitalization, seems to
be of value only if applied early. We also believe that oxygen should be administered early so as to prevent irreversible changes in the myocardium as well as pulmonary edema.

As to the causes of right heart failure after thoracoplasty there is a wide field for speculation. Bruce\(^3\) feels that the marked kyphoscoliosis developing in some cases of thoracoplasty may be an important factor. He states “the plastic patient often shows a striking gross anatomic resemblance to the kyphoscoliotic patient.” As attractive as this theory is, in neither of our cases was there sufficient deformity to consider it of etiological significance.

Studies of the respiratory function of lungs before and after thoracoplasty by bronchospirometry as those by Pinner, Leiner, and Zavod\(^4\) tend to prove that respiratory function is not unfavorably influenced by thoracoplasty. However, they state that this does not apply to thoracoplasties with pre-existing or added diaphragmatic paralysis. This was present in our two cases and probably contributed to the pulmonary insufficiency. It is necessary to point out that even temporary phrenic crush may result in permanent paralysis or at least partial palsy. From our own experience we feel that this measure contributes greatly to impairment of cardiopulmonary reserve.

Kaltreider, Fray and Phillips\(^5\) in an interesting study of pulmonary-ventilatory and respiratory function as well as function of the cardiovascular apparatus came to the conclusion that surgical interference with the chest-cage results in an anoxic anoxemia. The average value for the oxygen content of the arterial blood was below the average normal value. However, frank signs of right ventricular failure were not noted in their series and they state “this is not surprising when one realized that the vascular bed of the lungs must be reduced by approximately 60 per cent before the pressure is increased in the lesser circulation.” They make the additional statement: “there are however, other factors, such as anoxemia of the myocardium which may put an additional load on the right ventricle in patients with thoracoplasty.”

Cardiac failure in chronic pulmonary tuberculosis with permanent collapse procedures is obviously due to a combination of factors. The collapsed pulmonary area is usually not large enough to cause persistent pulmonary hypertension and its sequel, cor pulmonale. Additional factors which have been considered such as rigidity of the chest wall which interferes with normal respiratory-circulatory cycle, compensatory emphysema of the uncollapsed lung parenchyma of the thoracoplasty lung, capillary congestion and emphysema of the contralateral lung may each be contributory. The result of combining any of these factors appears to be anoxic anoxemia resulting in an added burden to
the myocardium. This chronic anoxemia as well as obliteration of part of the pulmonary capillary bed plus additional factors of age, coronary artery disease, or intercurrent upper respiratory infection will result in heart failure.

Obviously no definite causal relationship can be claimed to exist between the permanent collapse procedure performed in the two presented cases and the occurrence of right heart failure. However, by prolonging the life expectancy of individuals with chronic pulmonary tuberculosis by thoracoplasty, we may be presented with an increasing incidence of cardiac failure. It is definitely felt that patients with chronic pulmonary disease with or without collapse procedure who are dyspnoeic should be enjoined to live at low altitudes to avoid any additional strain upon the myocardium.

SUMMARY

1) We have presented two cases of right heart failure with chronic pulmonary tuberculosis with associated thoracoplasty and permanent phrenic paralysis.

2) While the theoretical considerations for such an association have been given, no definite causal relationship could be postulated.

3) Regular cardiovascular examinations were stressed for patients with increasing dyspnoea who had undergone permanent collapse procedures. Early treatment in cases of right heart failure appeared of definite value in restoring compensation as opposed to late cases.

RESUMEN

1) Hemos presentado dos casos de insuficiencia del corazón derecho con tuberculosis pulmonar crónica, asociada con toracoplastía y parálisis permanente del nervio frénico.

2) Aunque se han dado las razones teóricas de tal asociación, no se pudo enunciar ninguna relación causal bien definida.

3) Se recaló la importancia de llevar a cabo exámenes cardiovasculares metodicos en pacientes con disnea creciente que habían sido sometidos a procedimientos permanentes de colapso. El tratamiento temprano de los casos de insuficiencia del corazón derecho pareció ser de valor bien definido para restablecer la función cardíaca, en contraste a los casos tratados tardíamente.

REFERENCES