Compression of the Pulmonary Artery by an Aneurysm of the Ascending Aorta

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ANEURYSMS OF THE ASCENDING AORTA have been called "aneurysms of physical signs." In their growth, they may compress the right bronchus and lung, the sternum and ribs, the superior vena cava, the right and left atrium, the right ventricle and the pulmonary artery. Compression of the pulmonary artery occurs when the aneurysm enlarges anteriorly and to the left. Chronic cor pulmonale with its associated physical findings will result.

Perforation into the pulmonary artery is the usual result of such compression leading in turn to a left-to-right shunt with physical signs suggesting a patent ductus arteriosus. There are, however, some rare exceptions. It is the purpose of this paper to record an additional case of compression of the pulmonary artery without perforation by a syphilitic aneurysm.

CASE REPORT
A 66-year-old white man was admitted to the Bronx Veterans Administration Hospital on September 28, 1960, because of an abnormal chest x-ray film taken as part of a pension evaluation. He had been aware of an occasional cough with scanty mucoid sputum for three years. Hoarseness and fleeting sharp bilateral anterior chest pain had been present for one year. His collar size had increased from 15½ to 17 inches in a three-year period. Incidental discovery of a positive serology at the age of 42 resulted in a series of injections with bismuth and arsenic.

The blood pressure was 140/100 bilaterally. The radial pulses were equal and normal in volume. The pupils and eye grounds were normal. A tracheal tug was present. Prominent distended superficial veins of the arms, face, neck and chest were present. The lungs were clear. The point of maximal impulse was in the sixth intercostal space at the midclavicular line. The heart rate

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FIGURE 1: Roentgenogram of the chest showing marked aneurysmal widening of the mediastinal shadow. The arrow is pointing to the calcific deposits.

FIGURE 2: Angiocardiogram showing the pronounced distortion of the superior vena cava.
The aneurysms of the aorta were well visualized (Fig. 2). Cardiac catheterization: A No. 7 French cardiac catheter was introduced into the left basilic vein and passed successfully under fluoroscopic guidance into the superior vena cava and the right atrium. The right ventricle and the pulmonary artery could not be entered. An abrupt rise in pressure was noted as the catheter was being withdrawn from the right atrium toward the superior vena cava. This maneuver was repeated several times and the same pressure change occurred constantly (Fig. 3). Data obtained by cardiac catheterization have been tabulated in Table 1.

These studies confirmed the diagnosis of a superior vena cava syndrome due to compression of an aortic aneurysm. The patient received 12 million units of penicillin and was discharged on November 11, 1960. However, on December 3, 1960, he developed dyspnea on exertion. This increased in severity and necessitated his readmission on December 17, 1960. The blood pressure was now 100/70, and the pulse 100 per minute and regular. Rales were present at the right base. A holosystolic murmur could now be auscultated in the pulmonic area. A protodiastolic gallop was present. The liver was palpated four fingerbreadths below the right costal margin. There was moderate peripheral edema as well as cyanosis of the hands. The chest roentgenogram revealed a right pleural effusion. The electrocardiogram showed that the frontal plane axis had shifted further to the right. The impression was that the aneurysm had compressed the pulmonary artery.

Table 1—Cardiac Catheterization Data

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<th>Pressure in mm Hg</th>
<th>Saturation %</th>
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<tbody>
<tr>
<td>Superior Vena Cava (mean)</td>
<td>16</td>
<td>44.6</td>
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<tr>
<td>Right atrium (mean)</td>
<td>3</td>
<td>59.1</td>
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<tr>
<td>Brachial artery</td>
<td>118/84</td>
<td>88.9</td>
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was 96 beats per minute with a regular rhythm. There were no thrills, rubs, murmurs or gallops. The examination of the abdomen was negative. There was mild edema of the right forearm. No neurologic abnormalities were demonstrable. Hemoglobin was 12 gm per 100 ml. There were 7,800 leukocytes per cubic millimeter. Urinalysis was normal. The Wasserman was strongly positive. Spinal fluid, serology and colloidal gold were negative; protein was 38 mg per 100 ml.

The venous pressure in the right arm was 400 mm water and, in the right femoral vein, 100 mm water. The circulation time from the arm to the tongue was 16 seconds. Roentgenogram of the chest showed an aneurysm of the ascending aorta and arch. In the inferior margin of the aneurysm, calcific deposits could be seen (Fig. 1). The electrocardiogram showed right ventricular hypertrophy.

Angiocardiogram: With the patient in the supine position, 50 ml of 90 per cent Hypaque was rapidly injected into the right and left basilar veins using Robb-Steinberg needles. The dye entered all chambers of the heart. An adequate view of the pulmonary artery was not obtained.

**Figure 3:** A withdrawal tracing from the right atrium (left side of middle curve) to the superior vena cavae (right side of middle curve) shows the marked gradient between the two chambers. The upper curve is the brachial artery pressure.

**Figure 4:** Schematic drawing showing the aneurysm's compression of the superior vena cava and the pulmonary artery.
Treatment with digoxin and diuretics was of little benefit. The patient deteriorated progressively and expired on January 11, 1961.

Necropsy revealed that the heart weighed 400 grams. The left atrium was markedly dilated and the right atrium was moderately dilated. The right ventricular wall was thicker than normal and measured 5 cm. The left ventricular wall measured 1.5 cm. The cardiac valves showed no abnormalities. The major coronary arteries and ostia were widely patent. Four cm above the aortic valve a massive saccular aneurysm was observed (Fig. 4). It occupied approximately 75 per cent of the thoracic cavity and measured 26 x 15 x 8 cm. The wall of the aneurysm was partly calcified. There was a thick layer of organized thrombus in the aneurysm which measured approximately 4 cm in thickness. In its growth, the aneurysm had pushed anteriorly and to the left compressing the pulmonary artery. At the point of compression, the pulmonary artery contained two thrombi each 3 cm in diameter and the wall of the vessel was markedly thickened. At the site of union of the two innominate veins, the superior vena cava was compressed anteriorly by the aortic aneurysm. The aortic aneurysm had also distorted the atria. A small area of old pulmonary infarction was seen in the right lower lobe. This was most likely due to an embolus from the pulmonary artery thrombus. The microscopic sections of the ascending aorta showed typical mesoartitis luetica. The pathologic changes in the liver, spleen and kidney were those of chronic passive congestion.

**DISCUSSION**

Compression of the pulmonary artery by an aneurysm of the thoracic aorta is a rare occurrence. Including our report, there are now 36 proved cases. An analysis of these cases reveals that dyspnea and chest pain are the most prominent initial complaints. Dyspnea may be an expression of reflexes generated by aortic dilatation or may be related to beginning cardiac failure. Distention of the pulmonary artery, with activation of the stretch reflexes, probably also plays a role in the production of dyspnea. In some cases, chest pain is due to concomitant aortic insufficiency. However, pain can be produced by the stretching of the intrinsic nerve fibers within the aortic wall. Experimentally, obstruction and distention of the main pulmonary artery can lead to the production of pressing substernal chest pain presumably due to the stimulation of the afferent nerve endings scattered throughout the pulmonary arteriolar bed and the pulmonary artery.

When a critical reduction in the caliber of the pulmonary artery is present, right heart failure regularly occurs. Hepatomegaly, cyanosis, peripheral edema and/or ascites are the sequelae of this obstruction. In animal experiments, constriction of the pulmonary artery of at least 40 per cent is needed to raise the systolic pressure appreciably in the right ventricle, and still greater degrees of constriction are needed to produce right ventricular failure.

The presence of a systolic murmur at the pulmonic area is a universal finding. The pulmonic component of the second sound is of low intensity. Occasionally, a basal diastolic murmur can be heard even though the aortic valve is competent. In these cases, the murmur is due to the distortion of the pulmonic valve by the aneurysm resulting in valvular incompetence and pulmonic regurgitation.

Right ventricular hypertrophy or right bundle branch block are present on the electrocardiogram. A shift of the frontal plane axis to the right indicating right ventricular hypertrophy, proved to be a helpful diagnostic sign in our case.

Chest x-ray films show enlargement of the cardiac silhouette in the transverse diameter. The aneurysm extends anteriorly and to the left, forming part of the left border of the heart. Its appearance can simulate a pulmonary artery aneurysm. The peripheral lung fields often appear to be oligemic. Demonstration of calcification in the aneurysm is a useful diagnostic sign.

Cardiac catheterization of the right heart will reveal a systolic gradient across the compressed pulmonary artery. Angiography which delineates the ascending aorta and pulmonary artery is the definitive diagnostic study.

The average duration of life from the time of onset of symptoms is 9.2 months. Thus, surgical excision of the aneurysm by means of cardiac bypass offers the only possible cure of this condition.
REFERENCES


ALTERATION OF BLOOD COAGULATION DURING THORACIC SURGERY

During studies of blood coagulation and anticogulation systems in patients before, during and after surgical interventions on the lungs, it was revealed that during the operation there occurred disturbances in the coagulation system which was substantiated by intensification of the fibrinolytic and fibrinolytic activity, and an augmented discharge of heparin into the blood. On the 5th-11th postoperative days, the fibrinolytic function decreases, the level of free heparin normalizes and the fibrinogen content rises, this being conducive to thrombogenesis.


CARDIOVERSION FOLLOWING OPEN-HEART VALVULAR SURGERY

Twenty-six patients underwent cardioversion for atrial fibrillation and flutter following open-heart valvular surgery. In 11 of these, a Starr-Edwards valve prosthesis was inserted. The procedure was successful in 23 (88 per cent) and unsuccessful in three (12 per cent). There were eight recurrences over a period of ten months. Three failures and three recurrences occurred in those who had a prosthetic valve. In this series, prosthetic valves were employed in patients with mitral regurgitation (four), aortic regurgitation (one), and mitral stenosis with heavily calcified mitral valve (six).

It is felt that factors associated with the underlying lesion rather than with the valve itself were responsible for these results. Relative frequency of systemic embolism following the insertion of the prosthetic valves, especially in cases with a history of previous embolization, strongly justifies the attempt at cardioversion for atrial fibrillation and flutter.