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Cardio-circulatory changes associated with pulmonary emphysema

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CARDIO-CIRCULATORY CHANGES ASSOCIATED WITH PULMONARY EMPHYSEMA

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INTRODUCTION

Pulmonary emphysema is a disease which has long been known, but only recently recognized as having much clinical significance. A great deal has been written concerning the pulmonary function in this disease. However, less has been published concerning the associated cardio-circulatory changes. It is the purpose of this paper to review the changes occurring in the cardio-circulatory system in association with pulmonary emphysema, with special emphasis on the cardiac changes shown by x-ray studies.

GENERAL PATHOLOGY OF PULMONARY EMPHYSEMA

The mechanics of obstructive emphysema have not been adequately explained. The following explanation has been offered by Gunn (13). During inspiration, dilatation and elongation of the small bronchi and bronchioles occur. As mucus in these passages becomes dried and adherent to the walls, a partial obstruction developes, especially as the bronchi become shortened with expiration. Thus the air, trapped in the alveoli and respiratory bronchioles beyond the obstruction, cannot be absorbed by the blood as rapidly as it is replenished by inspiration. This leads to a constant

distension of the alveoli.

The additional probability of "interstitial alveolitis" was proposed by Liebow (20). This leads to focal necrosis with a loss of substance of the walls of the distal air spaces and ultimate fibrosis of the alveoli. Variable consequences may result, all leading to a disturbance in the normal respiratory function of the lung.

With the increasing expiratory obstruction, the lung becomes more or less permanently inflated. The patient develops an increased antero-posterior chest diameter, the diaphragms become depressed, and the lung less efficient. This results in an abnormal timed vital capacity, small inspiratory capacity, enlarged functional residual capacity and residual volume, slightly increased total lung capacity, and an increased residual volume: total lung capacity. Ventilation studies show increased resting ventilation, decreased maximal breathing capacity, diminished ventilatory reserve, and an elevated index of intrapulmonary mixing (38). As a result of these various degrees of ventilatory insufficiency, the first symptoms noted are coughing, wheezing, and shortness of breath. As repeated episodes of infection further impair gas

exchange, varying degrees of arterial hypoxia and hypercapnia develope, and respiratory acidosis and pulmonocardiac changes follow.

RESULTING CHANGES IN CARDIO-CIRCULATORY FUNCTION

Vascular Changes:

As the disease process of pulmonary emphysema progresses and bullae develope, thinning and rupture of the alveolar septa occur. This results in a reduction of the number, calibre, and expansibility of the smaller vessels (15). However, the loss of alveolar vasculature has never been adequately explained. McLean (23) felt that acute occlusive bronchiolitis leads to this disruption of alveolar walls. In his further study (24), he noted that inflammation of air passages produces some degree of damage to adjoining arterial walls. This precipitates the formation of arterial thrombi, which subsequently become organized. With these thrombi, sclerosis of the small arteries results. In early generalized emphysema, this arterial sclerosis is confined predominantly in the small arteries adjoining terminal and respiratory bronchioles. In addition to these findings, Reid (34) concluded that the most fundamental change in the blood vessels is ulceration, which, added

to the fibrosis and scarring often found in the lungs of patients with chronic bronchitis, results in a loss of the capillary bed.

As the capillary fields are obliterated, a muscular transformation occurs in the larger vessels which formerly supplied them (20). The reason for this muscular hyperplasia is not known. There is debate at the present time as to whether obliteration of these vessels is the cause or the effect of the pulmonary lesion, but at present most evidence points to the latter. In any event, these vessels persist as cords of muscular tissue. Whether this is actual muscular hyperplasia or the result of a state of musculature spasticity has not been determined. There appears to be little doubt that, in other instances of severe pulmonary hypertension, actual hyperplasia and hypertrophy occur.

It has long been known that arteriosclerotic changes occur in "normal" aging of lungs. Benner (5), as early as 1935, found that sclerotic changes, though slight, were seen in some part of the pulmonary vascular bed in 97 per cent of an unselected series of 100 autopsies. The atheromatous and fibrosing pulmonary artery changes commonly occurring in pulmonary emphysema were studied by McKeown (22). He concluded that emphysema

accentuates only slightly the normal age changes found in the pulmonary circulation. These bear no relationship to sclerosis of the aorta, coronary arteries, or other systemic vessels. This conclusion was shared by Parker (31) who felt that arteriosclerotic changes of the pulmonary vessels are probably secondary to pulmonary hypertension produced by obstruction of the capillary bed.

Blood Gas Changes:

Harvey (15) concluded that in chronic pulmonary emphysema anoxia is the most important abnormality. It is believed directly or indirectly responsible for the circulatory complications which result. Hypoxemia occurs in any condition in which there is interference with the normal transfer of oxygen from the alveoli to the capillary blood. This may be present even though alveolar ventilation is normal (27). A decrease in pulmonary blood flow, resulting from a reduced number of patent capillaries or a decrease in the number of capillaries in contact with functioning alveoli, decreases the diffusion capacity of the lung as readily as does an increased resistance at the pulmonary membrane.

Several studies have shown variable levels of

arterial blood oxygen saturation at rest in patients with pulmonary emphysema. West and others (42) found a reduced oxygen diffusing capacity in many of their patients, but concluded that this has little effect upon gas exchange in conditions of rest. They also concluded that this reduction was due to diminished total vascular bed rather than any change in the character of the alveolar capillary membrane. Motley (27), on the other hand, found a significant decrease in the arterial blood saturation at rest, with even more severe hypoxemia with exercise. He agreed that this is due to the presence of poorly ventilated alveoli perfused with blood, and to the perfusion of blood through non-ventilated areas. The increased cardiac output of exercise opens more capillaries in areas of extensive ventilation perfusion abnormalities. His studies, using intermittent positive pressure breathing and various concentrations of air and oxygen, have demonstrated the presence of poorly ventilated alveoli, and a significant amount of perfusion of blood through non-ventilated alveoli even at rest. He also demonstrated that an alveolar-capillary membrane block was not a significant factor in hypoxemia associated with pulmonary emphysema (27).

In the final stages of the disease there is also

a rise in the partial pressure of carbon dioxide in the blood (38). The arterial carbon dioxide tension may rise above 48 mm. of mercury with progression of the disease. Hyperventilation is the mechanism which attempts to compensate for the derangement in oxygen and carbon dioxide tension. With exercise this mechanism becomes inadequate. The onset of cor pulmonale is felt by some to be dependent on the degree of disturbance of blood gas tensions (17).

Hematologic Changes:

Hammarsten (14) studied 16 patients with pulmonary emphysema in whom the arterial oxygen saturation was less than 92 per cent. However, in none of these was there any significant difference from normal subjects in blood volume, erythrocyte volume, reticulocyte count, hemoglobin, or iron turnover. This was in agreement with the findings of Harvey (15) in patients with mild emphysema. However, he found a slight elevation of total blood volume and hematocrit in patients with an oxygen saturation of less than 90 per cent at rest.

Patients with chronic cor pulmonale in congestive failure exhibited very different findings. In these patients, in whom the oxygen saturation ranged from

61 to 53 per cent, there was a marked polycythemia and hypervolemia. The hemoglobin levels noted varied from normal or low in Harvey's series to significant elevations reported by Mounsey and others (28). The increase in total blood volume is almost entirely a result of increased numbers of red cells.

Hemodynamic Changes:

Bronchopulmonary Circulation:

The elastic fibers of the lung are attached to the visceral pleura and to the interstitial tissue in which are imbedded the vessels and bronchi (20). With inspiration a pull is exerted on the bronchi and the vessels. The vessels therefore enlarge, and in so doing acquire a larger volume of blood. In this way the blood-pumping action of respiration is initiated. The capillaries, on the other hand, are enclosed within the elastic alveolar walls. As these walls stretch and become thin with inspiration, the capillaries become flattened. If a critical point is exceeded, an impediment to inflow of blood results.

In areas of early emphysema, with abnormal destruction of alveolar septa and dilatation of alveolar ducts, there is a disruption of the tension balance (30). As a result, abnormally high alveolar pressures cause the

blood to be shunted away from the involved area. This semi-shunt vascular structure in the periphery of the lung lobes is believed to be related to rapid changes in blood flow requirements.

The collateral bronchial blood supply does not appear to be involved significantly in emphysema unless an associated fibrosis, bronchial disease, or bronchiectasis is present (20). Thus Nakamura and others (29) found no increase in bronchial blood flow in 7 of 8 cases of pulmonary emphysema. When the disease is complicated by fibrosis, the collateral blood supply gains in importance. Liebow (19) felt this to be a result in part of obliteration of the pulmonary arterial and capillary bed and marked expansion of the bronchial artery system. Simultaneously, large precapillary anastamoses appear between the bronchial and pulmonary arteries. In this way the high pressure systemic circulation is in direct communication with the low pressure pulmonary circulation. Because of increased resistance in areas of diseased parenchyma, desaturated blood in the pulmonary artery is shunted away from the points of anastamosis. Gray and others (12) felt that these collateral channels in part overcome the anoxemic effect of venous blood being shunted into the pulmonary veins through poorly aerated lung tissue. In postmortem examination of 18

cases of chronic bronchitis and emphysema, Cudkowicz and Armstrong (7) found profuse anastamoses between the vasa vasorum of the pulmonary arteries (derived from the bronchial arteries) and the pulmonary arteries themselves. They found this was associated with obliteration of the normal peripheral bronchial artery bed, and changes in the wall of the larger pulmonary arteries.

The expansion of the venous collateral flow is much more marked in pulmonary emphysema than in other types of pulmonary disease (19) (20). This expansion occurs even in the absence of right heart failure. Normally the true bronchial veins connect freely distally with the hilar pulmonary veins and proximally join plexuses of mediastinal vessels which empty into the azygos system. In advanced emphysema the bronchial veins expand greatly. With this expansion the valves become incompetent, and blood is allowed to flow in either direction. With the development of cardiac failure, right atrial pressure may exceed that of the left atrium and desaturated blood may flow in reverse to the usual direction.

Pulmonary Artery Pressure:

The development of pulmonary hypertension appears to be dependent on an increase in pulmonary resistance (9).

Borden and others (4), in an attempt to correlate pulmonary arterial pressure with loss of pulmonary function, found a significant elevation of pulmonary arterial diastolic pressure in all cases studied. No correlation was found between the degree of pulmonary hypertension and the severity of the emphysema, as estimated by increased ratio of residual air to total lung volume. In 8 cases with evident right heart failure, the pulmonary arterial pressure was higher and the oxygen saturation of the arterial blood lower than in uncomplicated cases. Mounsey and others (28) found that right ventricular pressures returned toward normal as patients recovered from failure. Therefore they concluded that the pulmonary hypertension in emphysema heart failure is reversible and cannot be due entirely to anatomical changes in the vascular bed of the lung. Considerable evidence exists that reduction in oxygen tension in the alveoli can result in pulmonary arterial spasm (10).

In a catheterization study of 18 patients with emphysema, Yu and others (45) measured the "pulmonary capillary" pressure, which corresponds to pulmonary venous pressure, and the pulmonary artery pressure simultaneously. While the "pulmonary capillary"

wedge pressure was within normal limits in all 18 cases, the mean pulmonary arterial pressure exceeded 15 mm. of mercury in 14 cases. The total pulmonary resistance and pulmonary arterial resistance exceeded normal levels in all instances of pulmonary hypertension. They also observed that the mean pulmonary arterial pressure varied inversely with the oxygen saturation of the arterial blood. Similiar findings have been reported by other authors (3) (41).

Cardiac Output:

In normal persons there is an increase in both cardiac output and arteriovenous oxygen difference with exercise, but the increase in cardiac output is predominant. If the heart cannot increase it's output sufficiently, the arteriovenous difference is increased. Hickman and Cargill (16) found, in their study of emphysema patients, that the arteriovenous difference remained within the normal limits, indicating an adequate rise in cardiac output. In the study by Blount and Reeves (3) the resting cardiac output was in the low normal range. With exercise, the cardiac output tended to be lower than normal for the work performed and the oxygen taken up.

By increased work, the right ventricle appears to

compensate for the increased vascular resistance in the pulmonary bed, thus preventing a decrease in volume flow (36). When an increase in cardiac output occurs, it is felt to be the result of an increase in oxygen consumption (35), polycythemia, and increased blood volume.

Renal Flow:

Saltzman (37), in a study of 17 cases of severe emphysema, found that the renal plasma flow can fall significantly in pulmonary insufficiency prior to the onset of heart failure. From this he concluded that cardiac insufficiency may occur early in cor pulmonale. This may result in an alteration in renal blood flow and clearances, which in turn may precede and contribute to pulmonary heart failure.

Cardiac Changes:

Changes in the Myocardium:

The sequence of events leading to myocardial failure is described by Harvey and others (15). Contributing factors include chronic hypoxia, arterial or arteriolar constriction, polycythemia, hypervolemia, and increased cardiac output. All of these contribute to pulmonary hypertension, which in turn leads to some degree of right ventricular hypertrophy and/or dilatation.

As the optimal stretch of the myocardial fibers is exceeded, right ventricular failure occurs. As this point is reached, the findings are those of impaired emptying of the ventricle, reduced stroke volume, increased residual diastolic blood volume, and an elevated end diastolic filling pressure. The intrinsic myocardial damage is not as extensive as that found in coronary disease or rheumatic myocarditis.

Not all patients with chronic pulmonary emphysema develope right heart failure. When it does develope, it is often reversible, or perhaps even preventable, with proper treatment. If, however, the disease has been untreated, inadequately treated, or has reached the end stage in which relief from anoxia is impossible, deterioration of the myocardial fibers occurs. Saltzman (37) believed coronary insufficiency may be a contributing factor in this developmental pattern.

Electrocardiographic Changes:

Littmann (21) observed that pulmonary emphysema causes characteristic electrocardiographic changes, probably due in part to altered extracardiac conductivity. The findings suggest backward displacement of the apex and clockwise rotation, but this is not confirmed by x-ray. Abnormal P wave changes also occur

which cannot be due to conduction disturbances. If altered chest conductivity were responsible for all of the electrocardiographic changes, the P wave would be expected to migrate in the same direction and to an equal degree as the QRS. This does not occur.

Phillips (33) studied the electrocardiographic evolution in cor pulmonale secondary to pulmonary emphysema. The earliest tracings showed no deviation from normal patterns. In these patients the autopsy findings revealed the least evidence of right ventricular hypertrophy. The second stage was the development of a rightward posterior direction of the major electromotive force (EMF). The third and final stage was characterized by anterior as well as rightward direction of the major EMF. At autopsy these patients had the heaviest hearts with the thickest right ventricular walls.

These findings were confirmed by Johnson and others (25) who studied pulmonary arterial pressure and electrocardiographic findings. Of 14 patients with a mean resting pulmonary arterial pressure exceeding 30 mm. of mercury, 8 showed a pattern of right ventricular hypertrophy. An opposing conclusion was expressed by Nakamura and others (29) who found no abnormal electrocardiographic findings in spite of considerable

heart enlargement.

X-ray Changes:

As early as 1937, Parkinson and Hoyle (32) described the radiological findings of emphysema with special reference to the cardiovascular system. They found that the cardiac factor in emphysema alone is seldom pronounced except late in the disease, and even then may not be significant. They found the most frequent cardio-circulatory change noted was enlargement of the pulmonary artery at the hila. The second most frequent evidence was localized enlargement of the heart, which was seen in about 40 per cent of cases studied. Most often involved was the conus pulmonalis of the right ventricle. In about half of these cases the body of the right ventricle was also enlarged. This was most readily seen in the left and right oblique positions respectively. Enlargement of the right atrium occurred in only about 14 per cent of the cases. Enlargement of the left ventricle and left atrium occurred only with coexisting cardiovascular disease.

The heart as a whole was not found enlarged in uncomplicated cases of emphysema. These findings have been confirmed by several authors. Nakamura and others (29) found that enlargement of the heart was least in

pulmonary emphysema among various chronic pulmonary diseases. Meschan (25) felt that the heart shadow is usually small and narrow, so that the heart appears to be smaller than it actually is. This is undoubtedly responsible for the description of the heart in emphysema as being small and "drop-like."

THERAPY

Anoxia appears to be the most important abnormality concerning the circulatory complications of pulmonary emphysema (15). Consequently the basic therapy is directed at alleviating the bronchiolar spasm and obstruction which are in large part responsible for the anoxia. As outlined by Woolf (44), the therapeutic management of an uncomplicated case of emphysema has three main objectives: 1. Eliminate smoking entirely. He stated that respiratory function tests show a decrease in pulmonary function after only one cigarette in emphysema patients. 2. Eliminate infection and prevent future infections if possible. The most commonly used antibiotics used are penicillin and streptomycin, tetracycline, or chloramphenicol. If a patient is subject to repeated infections, prophylactic antibiotics during the season of peak incidence is advocated. 3. Treat

bronchospasm vigorously at all times. This may be present without auscultatory findings. The continuous use of oral bronchodilator agents is recommended, plus the use of rectal suppositories at bed time and bronchodilator spray four times daily.

The prevention of cardiac failure in the emphysema patient depends on the relief of anoxia, plus the restriction of physical activity as indicated by the individual. When heart failure occurs, it is treated with salt restriction, diuretics, and digitalis glycosides. If oxygen is used, one must use care not to remove the only respiratory stimulus left to the patient. Gould (11) found, during the initial period of therapy with Digoxin, that cardiac output and pulmonary artery pressure increased slightly and diastolic pressure fell. After prolonged treatment, dyspnea and cyanosis disappeared and the heart size, pulmonary arterial pressure, right ventricular initial pressure and cardiac output had almost returned to normal and the arterial oxygen saturation had risen. Mounsey and others (28) also noted that full doses of cardiac glycosides increased peripheral arterial pulse pressure with reduced diastolic filling pressure, a finding which they attributed to an improvement in myocardial function.

Other drugs have been tried in the treatment of emphysema. Barach and Pons (1) reported on the use of anti-inflammatory steroids in pulmonary emphysema and cor pulmonale. A prompt weight loss was noted, probably due to increased glomerular filtration rate via maintainance of the structural integrity of capillaries to prevent fluid loss. The drug is also thought to inhibit the action of pituitary ADH, reduce edema of the bronchial mucous membrane, and relieve bronchospasm. Progesterone was also tried by Cullen and others (8) who found it produced an increase in both total ventilation and alveolar ventilation, accompanied by a fall in carbon dioxide tension in about half of the patients. The mechanism of action of the drug is not known.

CASE PRESENTATIONS AND DISCUSSION

Two cases of pulmonary emphysema are presented. These are intended to show the variable degree of cardio-circulatory involvement observed in the disease.

Case I:

This 29 year old single white male was admitted to University Hospital for the first time on September 19 with a chief complaint of dyspnea of 8 months duration, and pedal edema of 2 weeks duration. The patient, a

farmer, reportedly had "milk allergy" in the form of eczema during the first year of life. At age 6 he developed asthma. This had become progressively more severe and the patient was forced to drop out of school after the seventh grade because of frequent respiratory disease. Since that time he had farmed when he was physically able to do so. He stated he did not smoke except for perhaps one cigar a week. For the four months prior to admission the patient reported severe dyspnea with a dull, non-radiating mid-chest pain, accompanied by chronic cough. This cough was productive of two cups of sputum daily, occasionally blood-streaked. For one month prior to admission he had been unable to sleep except in a sitting position, and during this period had awakened several times with profuse sweating. For two weeks he had noted bilateral ankle edema and some intermittent, sharp precordial pain which was non-radiating.

Physical examination revealed a poorly developed, poorly nourished patient who appeared chronically ill. The patient was 5 feet, 9 inches in height, and weighed 111 pounds. The temperature reported on admission was 37.5° C. and the heart rate was noted to be 128 per minute. The patient's fingernails and lips appeared

somewhat cyanotic. Examination of the chest showed a marked increase in the antero-posterior diameter with use of the accessory muscles of respiration. On percussion the chest was noted to be hyperresonant throughout. Coarse rhonchi and wheezes were heard throughout the chest on both inspiration and expiration. Occasional premature ventricular contractions were noted on auscultation of the heart, but no murmurs or thrills were present. The liver was palpable 6 to 8 centimeters below the right costal margin and there was 2 + pitting edema of the feet and legs.

On admission the patient was noted to have a white blood cell count of 10,600 per cubic millimeter. Laboratory studies showed the patient to be in respiratory acidosis. An electrocardiogram on admission was reported as showing sinus tachycardia, frequent ventricular premature beats and aberrant conduction, a wandering pacemaker, right atrial hypertrophy, and marked right ventricular hypertrophy. This was considered consistent with cor pulmonale. Pulmonary function studies were also done, with marked impairment of function observed. (See Table I) Repeated chest x-rays were reported as showing marked bilateral pulmonary emphysema with chronic fibrosis and chronic bronchiectasis of both lung

bases. The heart was markedly enlarged, consistent with cor pulmonale. (See Table II)

Case II:

This 39 year old married white male was admitted to Douglas County Hospital for the second time on January 22. The chief complaint upon admission was shortness of breath and a "tight feeling" in the chest. The patient had apparently been well until 5 years prior to admission at which time he was diagnosed as having pulmonary tuberculosis. A segmental resection of the left upper lobe was performed. At that time the patient was told that he had emphysema. Prior to and following surgery, the patient received a total of two years of therapy with anti-tuberculous drugs. One year following thoracotomy, the patient developed a Friedlander's Bacillus pneumonia of the right lung and was hospitalized for approximately 6 weeks. Since that time he had experienced considerable shortness of breath with minimal activity. The patient had been seen in the emergency room on several occasions prior to the present admission for episodes of extreme dyspnea and shortness of breath. At one time, 6 months prior to admission, the patient was hospitalized for 10 days. Since that time he had been confined to bed most of the time. Two days prior

to admission the patient's dyspnea became much more severe and he noted a "tightness" in the right side of the chest. He denied any history of chest pain. Past history revealed that the patient had smoked two packs of cigarettes daily for years. He denied any history of allergy, and family history was non-contributory.

On physical examination, the patient appeared poorly nourished. He was 5 feet, 5 inches tall, and weighed 97 pounds. On admission the patient was in acute respiratory distress, and spoke with difficulty because of gasping breaths. The face appeared somewhat ashen, but no cyanosis was evident. The antero-posterior diameter of the chest was markedly increased, and the accessory muscles of respiration were employed. There was poor chest expansion with inspiration, and little movement of the diaphragm was noted. The entire thorax was hyperresonant to percussion. Breath sounds were distant and bilateral expiratory wheezes were heard. The heart sounds were distant. No murmurs were audible. The liver was not enlarged to palpation and no peripheral edema was observed.

The white cell count was observed to be 10,600 per cubic millimeter at the time of admission. Respiratory acidosis was not prominent as determined by laboratory

values. Sputum smears and cultures for acid fast organisms were negative. An electrocardiogram was reported as showing mild cor pulmonale and pulmonary emphysema. Pulmonary function studies after a period of hospitalization showed marked impairment of respiratory function. (See Table I) A chest x-ray was reported to show large emphysematous bullae filling the lower lung field and a portion of the left upper lung. (See Table II)

Case Discussion:

Both patients presented exhibited severe pulmonary disease as noted by the clinical findings described. The extent of the disease is probably best evaluated by the pulmonary function studies reported in Table I.

The first case represents a patient in whom respiratory function was markedly impaired, and in whom the clinical findings of pulmonary emphysema were evident. This was corroborated by the laboratory findings and the x-ray studies. The x-ray picture was also that of chronic fibrosis and chronic bronchiectasis. It is of interest to note that in this patient the heart was markedly enlarged (See Table II), and the electrocardiogram revealed right atrial hypertrophy and marked right ventricular hypertrophy. In addition, the physical

TABLE I. PULMONARY FUNCTION STUDIES

	Observed	Predicted
Case I:		
Vital Capacity:		
Total	750 cc.	4130 cc.
1 Second	30 %	75 %
Maximum Breathing Capacity	11 L.	114 L.
Case II:		
Vital Capacity:		
Total	1400 cc.	3760 cc.
1 Second	27 %	75 %
Maximum Breathing Capacity	*	*

* Patient was unable to tolerate testing.

findings of right heart failure were evident.

The clinical picture of the second patient was very similiar in regard to respiratory symptoms and impaired respiratory function. However, in this patient the pulmonary emphysema did not appear to be complicated by significant fibrosis or bronchiectasis. The heart shadow was smaller than normal on the x-ray (See Table II) and the electrocardiogram was reported as showing only mild cor pulmonale. No evidence of right heart

TABLE II. MEASUREMENTS OF CARDIAC SHADOWS

	Observed	Normal*
Case I:		
Transverse Diameter	152 mm.	107 mm.
Cardio-Thoracic Ratio	56 %	47 %
Frontal Area	158 cm^2	118 cm^2
$\frac{L \times B}{H \times W}$ **	24 %	20 %***
Case II:		
Transverse Diameter	105 mm.	104 mm.
Cardio-Thoracic Ratio	37 %	47 %
Frontal Area	94 cm^2	106 cm^2
$\frac{L \times B}{H \times W}$ **	13 %	20 %

* Normal values as defined by Stroud (39).
** $\frac{\text{Long and Broad diameters of the heart}}{\text{Height \& Width of thorax}}$
*** Normal for asthenic build.

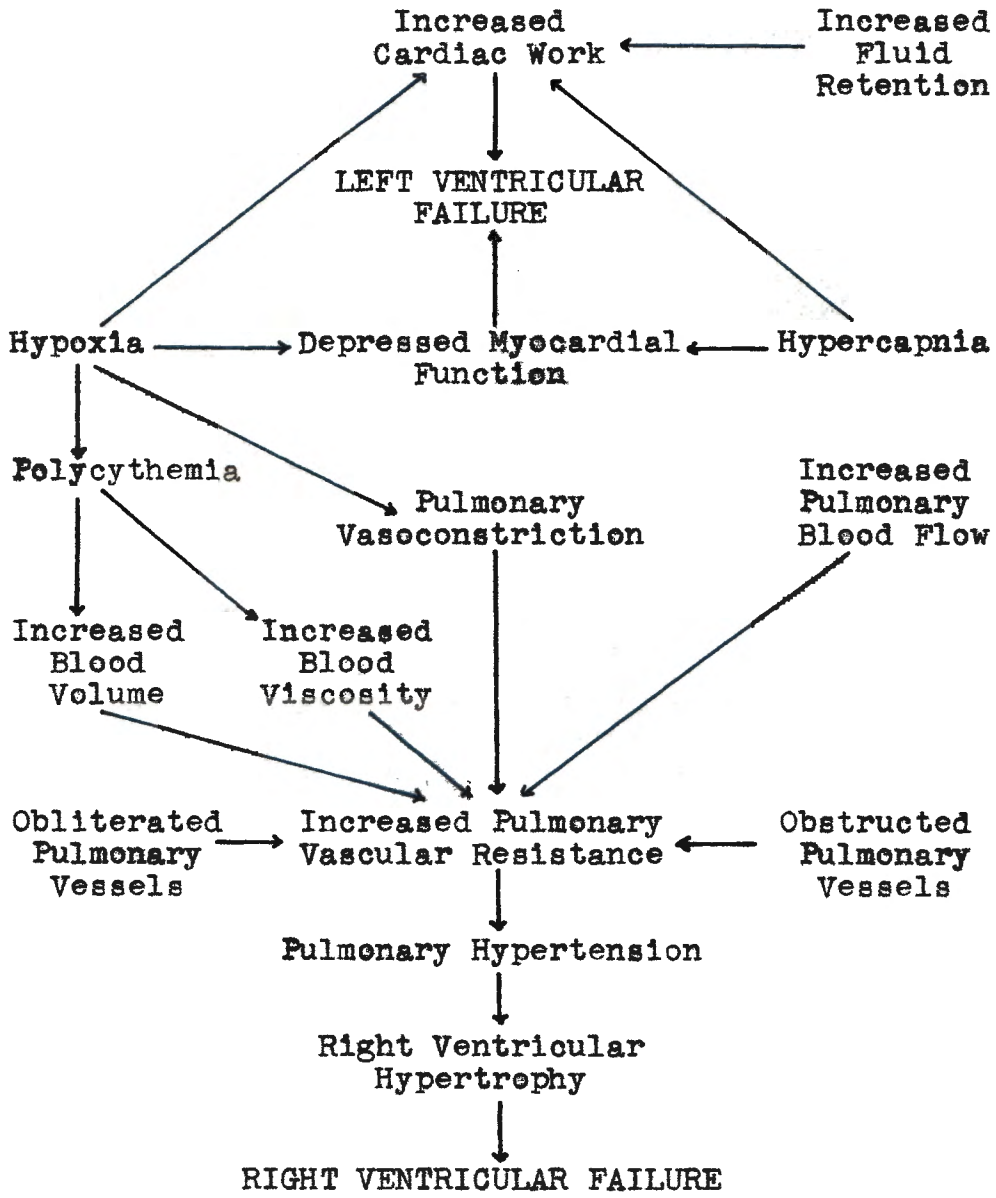
failure was observed.

It has been noted earlier in this paper that the extent of cardio-circulatory involvement in pulmonary emphysema is in large part dependent on the presence of co-existing pulmonary disease. This appears to be borne out by these cases. The first case exhibits a patient in which a long history of asthma is present, combined

with a long allergic history. Repeated respiratory infections, added to these findings, produced a pattern of complicated pulmonary emphysema resulting in cor pulmonale and subsequent right heart failure. The developmental pattern is perhaps best summarized in the scheme of Cherniack and Cherniack (6) shown in Table III.

The duration of clinical disease noted in the second patient was considerably shorter, and in this patient there appeared to be no complicating pulmonary disease prior to the development of tuberculosis. Although the impairment of respiratory function in this patient appeared to be as severe as in the first case, the heart was not enlarged and no evidence of failure was observed. In fact, the heart appeared to be smaller than normal on x-ray studies. It is quite probable that the heart was not actually smaller than normal, but appeared so because of flattened diaphragms and hyperexpansion of the thorax, leading to an abnormal cardio-thoracic ratio. The transverse diameter of the cardiac shadow on x-ray was shown to be normal, but the frontal area was less than the normal value established for a patient of similiar height and weight. Some authors have stated that the heart may appear smaller because of reduced filling of the heart with increased intra-thoracic pressure.

TABLE III. THE MECHANISM OF DEVELOPMENT OF HEART FAILURE IN RESPIRATORY DISEASE



SUMMARY

Pulmonary emphysema is a disease of unknown etiology associated with impaired pulmonary function. With progression of the disease, numerous changes occur in the cardio-circulatory system. Disruption of alveolar septa leads to the loss of capillary networks. In addition, arteriosclerotic and muscular changes occur in the larger vessels which supply them. As a result of this obstruction, pulmonary hypertension develops.

Hypoxia, an important factor in the genesis of pulmonary hypertension, occurs as a result of the perfusion of blood through poorly ventilated areas. Concomitant with this decrease in oxygen saturation of the arterial blood is an increase in carbon dioxide tension and a lowering of blood pH. Only when cor pulmonale and congestive failure occur is there any significant polycythemia.

In the pure form of emphysema, the bronchial arterial supply does not appear to be significantly involved. However, with associated fibrosis or bronchiectasis, bronchial arterial flow increases with the development of large precapillary anastamoses between the bronchial and pulmonary arteries. A peripheral vascular shunt tends to divert blood away from involved lung tissue.

The venous collateral flow is expanded much more in pulmonary emphysema than in other types of chronic pulmonary disease.

Pulmonary arterial hypertension is an almost constant finding in emphysema, and is found to be reversible. In uncomplicated cases of the disease, cardiac output is apparently normal at rest, but lower than normal with exercise. When congestive failure leads to polycythemia and increased blood volume, cardiac output may be increased. Altered renal hemodynamics may result from cardiac insufficiency.

Under the combined effects of these forces, right ventricular dilatation and failure may occur. The failure is reversible early in the disease, but as the disease progresses, deterioration of the myocardial fibers results.

Characteristic electrocardiographic changes are noted in pulmonary emphysema, felt to be due in part to disturbances in extracardiac conductivity. These changes progress to show right ventricular hypertrophy.

The cardiac shadow as noted on x-ray is not significantly abnormal in emphysema alone, except late in the disease. The most common change noted is enlargement of the pulmonary artery branches, with localized

enlargement of the right ventricle second in incidence.

Therapy of emphysema is directed at removal of respiratory irritation, elimination and prevention of infection, and relief of bronchospasm. When heart failure occurs, the use of digitalis glycosides, diuretics, salt restriction, and the discriminate use of oxygen is recommended. Steroid therapy has been beneficial in some cases.

Two cases of emphysema are presented to illustrate the clinical findings in a case with complicating pulmonary disease, and a case of uncomplicated emphysema.

CONCLUSIONS

Numerous cardio-circulatory changes occur in chronic pulmonary emphysema in addition to the disturbance of pulmonary function frequently reported. These changes may result in right ventricular hypertrophy and failure. The cardio-circulatory changes appear to occur more readily and are more severe in patients in whom the emphysema is complicated by concomitant pulmonary disease.

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