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Cor Pulmonale

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INDEX

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William A. O'Brien, M.D.

LAST WEEK

Date: November 7, 1941

Place: Recreation Room
Powell Hall

Time: 12:15 to 1:10 P.M.

Program: "Osteomyelitis of the Frontal Bone Complicating Frontal Sinusitis"
Emmet Milhaupt
Robert Movius
George M. Tangen

Discussion
H. O. Peterson
Lawrence Boies
William T. Peyton
Emmet Milhaupt

Present: 130

Gertrude Gunn,
Record Librarian

- - -

II. ANNOUNCEMENTS

1. WE WELCOME

We welcome the following to "mail" membership:

California
County of San Diego Hospital, San Diego
Florida
A. G. Leven, Miami
C. P. Trueg, Miami
Illinois
Milton M. Hurwitz, Chicago
Indiana
K. E. Selby, South Bend
Iowa
H. A. Tolliver, Charles City
Louisiana
R. H. Frost, Monroe
A. T. Ogaard, New Orleans
Theodore S. Wittels, Shreveport
Massachusetts
Harriet D. James, Brookline
Michigan
Battle Creek Sanitarium, Battle Creek
Minnesota
E. J. Tanquist, Alexandria
Walter P. Gardner, Anoka
A. H. Mork, Anoka
W. J. Bushard, Bird Island
E. G. Hubin, Deerwood
St. Luke's Hospital, Duluth
Stanley B. Lindley, Fergus Falls
Paul C. Benton, Gibbon
St. Gabriel's Hospital, Little Falls
Minneapolis
Arthur F. Bratrud
Walter E. Camp
J. C. Giere
Vernon L. Hart
Hennepin County Tuberculosis Ass'n.
Arthur L. Herman
George Hudson
Lawrence M. Larson
Johannes K. Moen
Russell W. Morse
Kenneth A. Phelps
J. A. Polzak
St. Barnabas Hospital
Justus Schifferes
Albert G. Bodaski, Montgomery
Theodore F. Hammermeister, New Ulm
Eugene F. McElmeel, Pipestone
St. Paul
George K. Hagaman
John E. Holt
C. E. Johnson
E. V. Kenefick
Henry N. Klein
A. A. Kugler
Charles T. Miller Hospital
E. W. Miller, St. Peter
Tracy Hospital, Inc., Tracy
P. W. Demo, Wells
E. A. Kilbride, Worthington
Montana
John A. Layne, Great Falls
New Jersey
Stanley Crosbie, Jersey City
New York
Thomas T. Mackie, New York
North Dakota
W. Lancaster, Fargo
South Carolina
Marion Sims Memorial Hospital, Lancaster
Virginia
Southside Community Hospital, Farmville
Angvald Vickeron, Petersburg
Canal Zone
G. M. Stenvaeon, Ancon

II. CHRONIC (PRIMARY) COR PULMONALE (PULMONARY HEART DISEASE)

Phillip Hallock
L. G. Rigler

Introduction and Definition

Two clinical types of cardiac strain and failure are recognized. By far the most common is the one representing strain and failure of the left side of the heart. This type does not require elucidation inasmuch as its etiological basis and clinical syndrome are well known. Suffice it to say that hypertension, coronary disease and lesions of the aortic and mitral valves are the underlying etiological factors. Dilatation and hypertrophy of the left ventricle characterize the pathological process and the clinical syndrome is manifested in general by dyspnea, cough, enlargement of the left ventricle and, in advanced cases, by the presence of moist rales over the lung bases.

Much less common are the diseases that primarily impose strain, with subsequent enlargement on the right side of the heart, independent of any abnormality of the left side of the heart. These are certain congenital defects, lesions of the tricuspid valve and an important group of diseases arising from the pulmonary parenchyma and the pulmonary circulation. It is to this latter group of diseases that the term "cor pulmonale" is generally applied although Brill has widened its application so that it covers practically all types of heart disease.

"Cor pulmonale" may thus be defined as enlargement with or without failure of the right side of the heart initiated by increased resistance to blood flow within the lesser (pulmonary) circulation as a result of pulmonary disease.

Pathologic Physiology

With the inception of increased resistance to blood flow a state of hypertension in the pulmonary circulation develops. The effect of this is to throw a strain on the right side of the heart with the result that the right ventricle is compelled to contract against an increasing blood comparable to the increased work imposed on the left ventricle as a result of arterial hypertension. Dilatation of the pulmonary artery and its branches and dilatation and hypertrophy of the conus pulmonalis and right ventricle characterize the pathological process and when failure of the right side of the heart eventually supervenes, the clinical picture is manifest by signs of increased venous pressure, namely, distention of the neck veins, hepatic enlargement, ascites, and edema.

The mechanism whereby increased resistance to blood flow is brought about in the pulmonary circuit is as follows: a disease process takes place which obliterates the capillary bed such as occurs in pulmonary emphysema or pneumoconiosis; or disease narrows the pulmonary circulation such as may occur in pulmonary arteriolarsclerosis. Because the vascular reserve of the pulmonary circulation is said to be great, it is apparent that considerable destruction of pulmonary parenchyma must occur before a sufficient amount of capillary bed is obliterated to increase pulmonary pressure. This concept is in agreement with clinical and pathological observations which show that considerable pulmonary involvement must be present before signs of right sided enlargement can be demonstrated.

Etiology

Chronic primary cor pulmonale is of manifold etiology. It should be stated that while mitral stenosis is the most important cause of enlargement of the right side of the heart, it is essentially a condition which originates in the left side of the heart and is therefore not included under the definition of cor pulmonale. The most important causes are pulmonary emphysema and extensive [illegible] fibrosis. When emphysema causes cor pulmonale, it is usually of a severe degree and in most instances is secondary to bronchial asthma, chronic bronchitis and bronchiectasis. Primary emphysema is not common. The manner by which [illegible] produces pulmonary

hypertension is through destruction of the interalveolar walls causing simultaneous obliteration of portions of the capillary bed. Extensive pulmonary fibrosis results from chronic pulmonary tuberculosis, pneumoconiosis, and other chronic pulmonary diseases. The mode by which pulmonary fibrosis causes pulmonary hypertension is by destruction of pulmonary tissue which similarly obliterates portions of the capillary system. Less frequent causes are pulmonary arteriosclerosis and kyphoscoliosis.

Pulmonary arteriosclerosis gives rise to pulmonary hypertension by narrowing of the pulmonary arterial tree. The narrowing may occur in the larger and medium sized pulmonary vessels and may present a pathological picture of advanced atherosclrosis, or the narrowing may be confined to the smaller vessels and arterioles and the morbid picture shows internal thickening and proliferation of subintimal connective tissue. The manner by which kyphoscoliosis operates to produce pulmonary hypertension is not definitely known. In this condition, large portions of the lung are atelectic and associated with other areas of compensatory emphysema. In addition, the possibility of kinking of the great vessels may play a role in obstructing blood flow in the pulmonary circulation. Congenital defects such as patent ductus arteriosus and septal defects and defects of the pulmonary valve are also causes of enlargement of the right side of the heart. Organic tricuspid valvular disease while extremely rare, is likewise a cause of right sided enlargement.

Review of Literature

In the past, chronic "cor pulmonale" was held to be a clinical rarity so much so that the possibility of its presence was entertained with a considerable hesitancy. In most instances it was observed as a pathological finding in the course of an autopsy examination where the cause of death was directed primarily to extensive pathological changes in the lungs. It is only within recent years that isolated right ventricular failure has been shown to be an important and direct cause of death in chronic pulmonary disease and that enlargement of the right side of the heart not infrequently developed as a complication. For the most part proof that the heart is frequently implicated in chronic lung disease is obtained from evidence afforded by post-mortem study.

Griggs, Coggin, and Evans recently reviewed the autopsy protocols of all chronic pulmonary diseases encountered in the Los Angeles County General Hospital. They employed the average thickness of the wall of the right ventricle as the criterion of hypertrophy. A right ventricular wall which averaged 5 mm. or more in thickness was considered definitely hypertrophied. They concluded that chronic pulmonary disease was an important cause of cor pulmonale. Pneumoconiosis showed the highest percentage incidence of cor pulmonale. Right ventricular hypertrophy occurred in 52% and congestive failure in 50% of 24 patients. Right ventricular hypertrophy occurred in 28.9% and congestive failure in 22% of 45 cases of emphysema. The hearts of patients with pulmonary tuberculosis showed right ventricular hypertrophy in but 3.7% and definite congestive failure in 1.8% of 1470 cases. These figures differ greatly from those reported by Nemet and Rosenblatt who found exclusive hypertrophy of the right ventricle in 34% of 71 cases of tuberculosis.

Kountz, Alexander, and Prinzmetal separated and weighed the right and left ventricles of the hearts of 17 patients with marked emphysema and found dilatation of the right ventricle in 10 out of 17 cases with hypertrophy of both right and left ventricles in most of the cases.

Scott and Garvin have recently reviewed a series of 6,548 autopsies to determine the incidence of primary right heart enlargement excluding congenital defects regardless of the presence or absence of right heart failure. They also used the thickness of the right ventricle (5 mm. and over) as a criterion for right heart enlargement. In this series there were 240 patients who had died of heart disease of which 50 or 5.3% approximately represent samples of cor pul-

...nale.

Clawson in a review of 30,265 autopsies in the records of the Department of Pathology of the University of Minnesota found 5,265 cases in which death resulted from heart failure. 102 or about 2% of the total could be definitely considered cases of pure right heart failure resulting from lesions of the lungs or of the lesser circulation. The smaller number in this series as compared to that of Scott and Garvins' is no doubt due to the fact that Clawson employed heart failure rather than enlargement as a criterion. The leading cause of right heart enlargement in this series was pulmonary tuberculosis, representing 29 cases. This high incidence is probably the result of Higgins' careful examination of the right heart in the autopsies of patients dying at the Glen Lake Sanatorium for tuberculosis. The second most common cause was bronchial asthma with emphysema representing 19 cases. Bronchiectasis was the cause of right heart enlargement in 15 cases, primary pulmonary arteriosclerosis in 11, silicosis in 5, and nonspecific empyema in 5. The remaining cases were of varying origin.

Higgins reports that approximately 30% of all the tuberculous patients coming to autopsy at the Glen Lake Sanitarium show evidences of right heart enlargement while approximately 10% show signs of right heart failure. In a series of 41 cases of bronchial asthma coming to autopsy Rigler and Truog found 25% to have died of right heart failure. Thus the incidence of right heart enlargement or failure is not rare as a complication or cause of death in individuals with chronic pulmonary disease.

Clinical studies relating the incidence of chronic cor pulmonale to chronic pulmonary disease are meager. This is because of the particular difficulty encountered in recognizing right sided cardiac strain by clinical examination. When signs of gross congestive failure are found, then the diagnosis becomes readily apparent. But in a large majority of cases clinical signs indicating failure of the right side of the heart have not yet developed to the point where edema, ascites, or hepatic enlargement are evident. In this group of cases clinical recognition of enlargement of the right side of the heart becomes a difficult and very important diagnostic problem. In the first place symptoms of cardiac disease are strikingly similar to symptoms which arise from chronic pulmonary disease; namely, cough, dyspnea, and orthopnea. Secondly, evidence indicating strain of the right side of the heart cannot be elicited with accuracy by physical examination. Percussion of the cardiac borders to determine the presence of right sided enlargement is too unreliable because in a great majority of instances pulmonary emphysema is present. Accentuation of the pulmonic second sound is often a very helpful sign but too much reliance must not be placed in it alone because pulmonary heart disease may be encountered in young individuals and furthermore, cor pulmonale in the elderly individual is occasionally associated with strain and enlargement of the left side of the heart, conditions which may give rise to accentuation of the pulmonic second sound. If it were possible to estimate the level of the blood pressure in the pulmonary circulation with the same ease (by syphomanometry) as in estimating the systemic blood pressure, then the recognition of right sided cardiac strain would become a simple matter.

From this it is apparent that the recognition of chronic cor pulmonale presents many diagnostic difficulties. Without further objective evidence the best the clinician can hope to do is to speculate on the possibility of the presence of right ventricular strain unless signs of right heart failure are present. To obtain definite objective evidence recourse must be had to more refined clinical methods such as are afforded by electrocardiography and roentgen examination of which the latter is far more valuable. By careful roentgen examination cardiac involvement in cases of chronic pulmonary disease can be recognized in a large percentage of cases by evidence of enlargement of the right side of the heart. Using this method, Parkinson and Hoyle have demonstrated the occurrence of right sided cardiac enlargement in approximately 50% of the [illegible] of 30 patients who [illegible]

ffering from a high degree of emphysema. a series of 52 cases of bronchial sthma with emphysema, Rigler and Truog ound some roentgen evidence of right ided enlargement in 46% of cases.

Choice of Cases

In the series of cases here reported, cardiac enlargement as demonstrated by roentgen examination constituted the most important and final criterion for evidence of right sided cardiac strain except in those cases where signs of cardiac failure were present.

Only those cases were selected for study which were found to have enlargement with or without failure of the right side of the heart. (The term "right side of the heart" implies anatomical divisions of the heart and great vessels which are commonly involved in pulmonary hypertension, namely, the pulmonary artery and its main branches, the conus pulmonalis, the body of the right ventricle and the right auricle. Any one or a combination of any one of these divisions may be involved in right sided cardiac strain.) In this series were 38 patients whose cardiac and pulmonary status was studied on the Medical Service and in the Out-Patient Department of the University Hospital. The etiological basis of chronic cor pulmonale in this series was as follows:

	Cases
1. Bronchial asthma and emphysema	19
2. Non-specific fibrosis and emphysema	5
3. Silicosis	2
4. Pulmonary arteriosclerosis	11
5. Thoracic deformities	1
Total Cases	38

In this series no cases of chronic pulmonary tuberculosis were encountered as a cause of cor pulmonale. The emphysema group constituted the leading cause of cor pulmonale. Primary pulmonary arteriosclerosis while considered a rare disease accounted for approximately one-third o. the cases.

Clinical Features

1. Emphysema group.

For the purpose of analysis the two emphysema groups (1 and 2) are combined. In this series of 24 cases were 14 males and 10 females, and their ages ranged from 23 to over 70 years. Sixteen cases showed no clinical signs of right heart failure and the remaining 8 cases had slight to far advanced failure. These were all evaluated and verified by venous pressure measurements. Four patients died of heart failure. As to the causes of emphysema all cases had bronchial asthma associated with or without chronic bronchitis and bronchiectasis with the exception of 4 cases all of which were found in association with non-specific fibrosis. In one patient (female, age 41 years) no cause of emphysema could be determined. This patient died of right heart failure. In approximately 75% of cases the asthma was present for more than 5 years. In the remaining 25% the asthma was present for at least three years and was of a very severe degree.

Post-mortem examination was carried out in three of the four patients who died of heart failure. It was interesting to note that the pulmonary arteries were normal in 2 patients and that the third patient showed marked pulmonary arteriosclerosis. The contention that pulmonary arteriosclerosis must be associated with emphysema to produce cor pulmonale is not borne out by this study.

Cardio-pulmonary Symptoms and Signs

Cough and dyspnea were the outstanding complaints and were present to some degree in all cases. In those cases where cardiac failure was present, dyspnea was present to a marked degree. Orthopnea was conspicuous in those patients with heart failure. Cyanosis was found in 10 patients, but eight of these had right heart failure. In these 8 patien s signs of increased venous pressure were noted. i.e., edema, ascites, hepatic enlargement, and distention of

neck veins. Slight clubbing of the fingers was found in only two cases.

Secondary polycythemia was found in only three cases. A history of hemoptysis was obtained in four cases.

Cardiac Findings

Normal rhythm was an outstanding feature. It was observed in all patients regardless of the presence or absence of heart failure. The blood pressure was normal in all patients except in one case where there was only a slight elevation. There was no evidence of enlargement of the left ventricle in this patient. Accentuation of the pulmonic sound indicating increased pulmonary pressure was observed in 8 patients. In no case was a diastolic murmur elicited. In three cases apical systolic murmurs were heard. Percussion of the cardiac borders to determine the presence of enlargement of the right side of the heart was of no value and in most of the cases misleading. Only in one case was enlargement in the region of the pulmonary artery elicited by percussion. Obviously in the presence of pulmonary emphysema percussion is a very unreliable method to determine the size of the heart.

The electrocardiograph was found to be of considerable assistance in determining the presence of predominant right ventricular strains. Right axis deviation was found in 9 cases (38%). Normal axis deviation was found in 5 cases and left axis deviation in 1 case. A striking feature of the electrocardiographic records was the presence of high and pointed "P" waves in the 2nd and 3rd leads. This was noted in 10 cases (40%). In the patient who had left axis deviation, these characteristic "P" waves were found. Low potential was noted in 2 records, and depression of ST interval in the 4th lead was observed in about 15% of the cases.

Roentgen Findings

The radiological appearance of the heart afforded the most reliable evidence for involvement of the right side of the heart except in those cases where congestive failure was present.

1. Emphysema

As might be expected, the major group in this series of cases of right heart enlargement have as their etiology emphysema of some type. Most of the cases were the result of bronchial asthma but 5 patients with emphysema or lung fibrosis of non-specific origin are also included. The roentgen appearance of the lungs is characteristic. The chest is long and narrow. The interspaces are wide, the sternum displaced anteriorly, the thoracic spine often kypliotic. The diaphragms are low and relatively immobile. The lung markings are very prominent, the root shadows enlarged, and there may be local areas of greatly increased radiability surrounded by a capsule, representing emphysematous blebs or bullae.

The heart in emphysema is, generally speaking, relatively small, and tends to be long and narrow. The left border shows a distinct convexity with particular prominence of the second curve, that is, the curve of the pulmonary trunk. There is also enlargement of the conus pulmonalis, the third curve on the left side, but this is usually overshadowed by the enlarged pulmonary trunk. When examination is made in the right oblique position, the unusual prominence of both the conus pulmonalis and pulmonary trunk can be readily made out. Often in this position the enlarged pulmonary trunk can be traced posteriorly across the aortic window into the right and left branches. In the right lateral view with the filled esophagus, no displacement of the esophagus is shown indicating no enlargement of the left atrium which tends to exclude mitral disease. The pulmonary trunk can be seen on end as a very dense, somewhat rounded shadow lying in the posterior portion of the heart just below the aortic window. The heart comes up to the anterior chest wall reflecting the enlarged right ventricle. In the left oblique position, the right ventricle can be made out projecting anteriorly and to the right with a distinct convexity. Generally speaking the root shadows of the lungs are enlarged because of the distinct enlargement

f the main branches of the pulmonary rtery. The whole appearance is that of n enlargement of the out-flow tract of he right ventricle and of the right entricle itself. When failure supervenes, the enlargement is much more arked and some dilatation of the right atrium extending over to the right side can occasionally also be made out. The conus pulmonalis and the trunkis pulmonalis both become much more prominent and the heart as a whole will show a distinct increase in size.

2. Pneumoconiosis

Two cases of silicosis were found to have chronic cor pulmonale. Both were males and gave a history of being sand blasters for many years. In both cases there was extensive bilateral pulmonary involvement. Both patients had an associated emphysema. In addition one patient had bronchial asthma and bronchiectasis. Right heart failure was present in both cases, existing in a mild form in one and far advanced in the other. The outstanding signs and symptoms were pronounced dyspnea and orthopnea. Cyanosis of the lips and finger tips were pronounced in both; clubbing of the fingers was observed in only one patient. The blood pressure was within normal limits in both instances. The electrocardiogram indicated predominant right ventricular strain in both with characteristic high and pointed "P" waves in only one case. One patient succumbed to right heart failure. The radiological picture showed characteristic right sided enlargement very similar to the pattern noted in emphysema heart.

3. Pulmonary Arteriosclerosis

This series comprised a very interesting group of 11 cases. Seven were females and 4 males. The ages ranged from 5 to 65 years. All patients except 2 were under 45 years of age. The age span of the majority of patients was between 20 and 45 years. Eight patients (73%) had right heart failure when first seen. As to the etiology of this disease very little is known. It was originally thought to be syphilitic in origin according to the teachings of Ayerza and Arrillaga, but subsequently it has been demonstrated that syphilis is not necessarily the cause. Only in 1 of our cases was a positive Wassermann reaction obtained, but we could not subscribe the origin of the disease to syphilis in this instance. A history of rheumatic fever was obtained in only one case. No history of bronchial asthma was obtained in any case. One patient gave a history of chronic bronchitis and bronchiectasis. A history of frequent upper respiratory infections was common.

Clinical Features

The outstanding symptoms were dyspnea, cough and fatigue. Of these, dyspnea was the most prominent symptom, and was described in many cases as being gradually progressive in character. It was elicited in all cases except one. This patient had no symptoms but on routine roentgen examination through the Students' Health Service, the chest revealed evidences of right sided cardiac enlargement. Subsequent examinations indicated that the patient had pulmonary arteriosclerosis. Various grades of dyspnea were mentioned, ranging from dyspnea on moderate exertion to dyspnea present at rest. A history of cough was elicited in all cases except two. In all instances, except one, the cough was described as being 'dry' and non-productive in character. A history of ankle edema was elicited in 7 patients.

Dyspnea and orthopnea were evident in 6 cases on physical examination. Tachypnea was a pronounced feature in two cases. Cyanosis from a slight to intense degree was noted in 9 cases (80%). Clubbing of the finger tips was noted in only one case. A very striking feature of the disease was the absence of rales over the lung fields in 9 patients. Eight patients had signs of venous congestion. Conus enlargement by percussion was elicited in 6 patients. The pulmonic second sound was accentuated in all cases. The cardiac rhythm was regular in all cases except one, which had auricular fibrillation. In no case was a diastolic murmur elicited. Secondary polycythemia

found in 50% of the cases.

All cases except one showed electrocardiographic evidence of predominant right ventricular strain. High and pointed P waves in the 2nd and 3rd leads were encountered in 40% of the cases. Inversion of T waves and depression of the ST interval in the 2nd, 3rd, and 4th leads were noted in 50% of the cases. Intraventricular heart block was present in 2 cases.

Radiological Examination

Inasmuch as the lesion in pulmonary arteriosclerosis is in the pulmonary vessels themselves rather than arising in the parenchyma of the lungs as is the case in emphysema, silicosis and other fibrotic conditions, the roentgen appearance of the lungs therefore is stikingly different. The enlargement of the right ventricle and pulmonary trunk is marked as in all cases of cor pulmonale, but in addition the major branches of the pulmonary artery are enormously enlarged and show an unusual degree of pulsation. The smaller branches are also greatly enlarged. The picture is often confused with pulmonary congestion but the clarity of the lung parenchyma about the sharply defined dense shadows of the arteries helps to differentiate. Disappearance may be simulated by certain congenital defects, notably patent ductus arteriosus and larger defects in the interatrial or interventricular septa. In such cases, in addition to the usual methods of roentgen examination, roentgen kymography is of some value. It will reveal a marked increase in the amplitude of pulsation of the major pulmonary branches, but the smaller vessels show only transmitted pulsations.

Five patients in this series died of right heart failure and the diagnosis of pulmonary arteriosclerosis was confirmed in all cases by autopsy examination. Examination of heart and great vessels revealed dilatation of the pulmonary artery with dilatation of the conus pulmonalis and dilatation and hypertrophy of the right ventricle. Extensive pathological changes were observed in the pulmonary vessels. Two cases showed striking intimal proliferation and medial fibrosis of smaller arteries and arterioles. One case revealed sclerotic thickening of larger branches of the pulmonary tree with no arteriolar changes, and 2 cases disclosed combined arteriosclerosis of large pulmonary artery branches and intimal arteriolarsclerosis.

4. Kyphoscoliosis (hunchback heart)

One case of cor pulmonale due to kyphoscoliosis was encountered. The patient was a male, age 17 years, in whom deformity of the chest was first noted at about 2 years of age. The deformity became more marked with age. When 12 years old, the patient displayed the typical hunchback appearance. He was admitted to the hospital on 5 previous occasions for pneumonia, chronic otitis media, and for mastoidectomy. On his last admission to the hospital, his complaints were those of dyspnea and cough. Physical examination revealed marked dyspnea, cough and cyanosis The blood pressure was 90/64, pulse rapid and respirations rapid. The liver was enlarged and tender. The patient died on the same day of admission and autopsy revealed marked dilatation and hypertrophy of the right ventricle and dilation of the right auricle. The left side of the heart appeared normal, with no valvular defects. The right lower lobe was atelectatic with partial atelectasis of the right upper lobe. The left lung appeared normal. An electrocardiogram was not made.

Course and Prognosis in Chronic Cor Pulmonale

Chronic cor pulmonale is characterized by its progressive but chronic course extending over a period of years. Most of the patients die of intercurrent infection especially pneumonia. Others, particularly in the older age group, die of associated cardiac disease such as hypertension or coronary disease. However, a certain number of patients as indicated above die of isolated right heart failure. In a small number of cases death may occur suddenly and unexpectedly from pulmonary embolism.

The treatment of chronic cor pulmonale should be directed primarily to

management of the underlying pulmonary disease. When failure of the right side of the heart occurs, then the usual measures for the treatment of congestive failure should be instituted such as fluid restriction, a salt-free diet, administration of digitalis, and the mercurial diuretics. For the relief of dyspnea and cyanosis oxygen therapy should always be employed as it is often found to be very beneficial. In many cases when congestive failure supervenes, the course is rapidly downhill and unfortunately therapy is usually ineffective.

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Differential Diagnosis

Any condition which may affect the pulmonary vessels and right side of the heart may simulate chronic cor pulmonale. Mitral stenosis is the most important as it is one of the most frequent causes of enlargement of the right side of the heart.

Chronic cor pulmonale	Mitral stenosis
1. No history of rheumatic fever.	1. History of rheumatic fever.
2. Often history of chronic pulmonary disease such as asthma, chronic bronchitis, and bronchiectasis.	2. Absence of history of chronic pulmonary disease.
3. Absence of murmurs.	3. Presence of presystolic or diastolic murmurs.
4. Usually regular rhythm.	4. Auricular fibrillation common.
5. Absence of left auricular enlargement on roentgen examination.	5. Presence of left auricular enlargement.
6. Marked enlargement of pulmonary trunk and pulmonary arteries.	6. Marked enlargement of conus pulmonalis.
7. High and pointed waves in second and third leads in the E.K.G.	7. Broad or bifid "P" waves in second and third leads in the E.K.G.

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Cor pulmonale must also be differentiated from certain congenital heart defects, particularly those associated with arteriovenous shunts such as patent ductus arteriosus, and septal defects. In chronic cor pulmonale the characteristic murmur of patent ductus arteriosus is absent, and likewise the loud systolic murmur of interventricular septal defect. Furthermore, the size of the heart is very little affected in interventricular septal defect. On the other hand, the differential diagnosis of interatrial septal defects from pulmonary arteriosclerosis is a most difficult one and oftentimes impossible to make. Other conditions which cause right sided enlargement such as hyperthyroidism and myxedema and Tetralogy of Fallot must be taken into account in differential diagnosis. The final diagnosis in many instances will depend on the radiological appearance of the heart.

Summary and Conclusions

1. Thirty eight cases of chronic cor pulmonale were clinically investigated, chiefly in regard to etiology and to the cardiovascular findings.

2. [illegible] of the right side of

the heart as judged primarily by radiological examination constituted the criterion for evidence of cardiac strain.

3. This data indicates that chronic pulmonary disease is an important cause of cor pulmonale and subsequent heart failure.

4. The data emphasizes the necessity for a close scrutiny of the cardiac status in all cases of chronic pulmonary disease.

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To be published.

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IV. GOSSIP

Under "Book Notices" in the J.A.M.A. 117:1657, (Nov. 8) 1941, we spied the following. Are we proud!

"Sulfanilamide and Related Compounds in General Practice. By Wesley W. Spink, M.D., Associate Professor of Medicine, University of Minnesota Medical School, Minneapolis. Cloth. Price $3. Pp. 256, with illustrations. Chicago: Year Book Publishers, Inc., 1941.

The importance of the sulfonamide drugs in modern therapeutics is reflected in the voluminous literature that has accumulated since the first report on sulfanilamide which appeared in 1936. The author of this volume states that he consulted over one thousand original articles, reviews and monographs in the preparation of the book. The first important textbook on the sulfanilamide drugs to appear was "Sulfanilamide Therapy of Bacterial Infections" by Mellon, Gross and Cooper. Shortly thereafter the authoritative textbook "Clinical Use of Sulfanilamide and Sulfapyridine and Allied Compounds" by Long and Bliss became available. Dr. Spink's volume comes in natural sequence to those previously published.

After disposing of historical considerations and general principles of therapy the author devotes a chapter each to neoprontosil, sulfanilamide, sulfapyridine and sulfathiazole. Ten chapters are given to specific problems in treatment. For example, chapters are devoted to treatment of pneumonia and its complications, treatment of staphylococcic infections, local use of the sulfonamide compounds and use of the sulfonamide compounds in dentistry.

The final two chapters are devoted to the consideration of sulfaguanidine and

...iradiazine. Developments have been so ...pid in the field that a book attempting ... cover the subject is in danger of being ...t of date by the time it makes its appear...nce. Fortunately for this volume the ...uthor was able to write the last two chapters just up to the date of publication.

This book provides a thoroughly authoritative discussion of the subject. All through the book the author reveals his critical judgment; he knows when something has been convincingly demonstrated and when reported findings should be taken with a grain of salt. Such critical judgment is particularly essential in this special field. So much has been written and so many unsound deductions have been recorded in the literature that a zealot can find material to prove or disprove almost anything. It is gratifying likewise to find that the author has been careful to weigh the expected benefits of sulfonamide therapy against the possible deleterious effects. For instance, a conservative attitude is revealed in the use of sulfanilamide in the treatment of scarlet fever and tonsilitis.

While the author submits recommendations for the use of the sodium salt of sulfapyridine orally, subcutaneously and intrathecally, he might have done a service by indicating his disagreement with such pointless and possibly injurious use of the drug. The same criticism applies to instances in which the author is merely content to quote an unsound opinion reported by some one else without definitely showing the reader that he disagrees with it.

Despite a few loose ends of this kind, the volume is an important contribution and may be recommended to any one who contemplates using the sulfonamide drugs discussed. The value of the book is enhanced by illustrative case reports and clinical charts that accompany the discussion of specific clinical problems. It is hoped that in subsequent editions the use of sulfadiazine and perhaps sulfaguanidine will be absorbed into the marrow of the volume; but one may also express the hope that the author will have the courage to disregard, as he commendably already has, the numerous unessential proprietary modifications of sulfanilamide and its derivatives that are now beginning to flood the market."

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"I have now been in Great Falls for a period of three months and like it very much. The remainder of the family are arriving tomorrow. We had some difficulty in obtaining the proper sized house. We have been very busy this summer. The work, of course, is somewhat different from that at the University but I enjoy it a great deal. There are many opportunities, and we are laying out a program of clinical and laboratory investigations.

Yours sincerely,
John A. Layne, M.D.
Great Falls Clinic.

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MINNESOTA PATHOLOGICAL SOCIETY

Speakers for the November and December meetings of the Minnesota Pathological Society are announced.

On November 19, Dr. Melvin E. Knisely, assistant professor of anatomy, University of Chicago and University of Tennessee, will speak on "The Effects of Ilasmodium Malaria on the Blood Vascular System," and will illustrate his talk with kodachromes. Dr. Knisely has been loaned by the University of Chicago to the University of Tennessee and the Tennessee Valley Authority for the year 1941-42 to continue studies of anti-malaria drugs on the parasites and on the hosts.

Dr. Karl F. Meyer of George Williams Hooper Foundation, University of California Medical Center, San Francisco will address the Minnesota Pathological Society, December 10, at its meeting in the Amphitheater of the University of Minnesota Anatomy Building. His subject will be "The Animal Kingdom, a Reservoir of Infection."

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