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## THE HEART IN THE PNEUMONIAS \*

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According to the vitality and endurance of the heart and the circulatory system (including its vasomotor supply), may be reckoned the outcome of the given instance of pneumonia. Seldom, if ever, will the mechanical obstruction of the lungs prove a serious factor except in its influence on an already crippled cardiac mechanism. The less perfect the aeration of the blood in its passage through the pulmonary tissue, the more positive is the deprivation of the cardiac structure (muscle, ganglion and nerve) of its proper supply of nutriment.

The importance of this influence is emphasized by the reflection that the blood, under conditions of health, demands over 90 per cent. saturation with natural oxygen as the result of its passage through the lungs.<sup>1</sup> The more complete the stoppage of the blood- and lymph-streams in their course through the walls of the air-vesicles, the more real is the mechanical overload thrown on the pulmonic valve, and soon on a toxemic, tired, right ventricular wall. The more intense and virulent the toxemia, the more certain and extensive will be the damage to the entire cardiac mechanism, vascular, nervous and muscular. Yet there is often to be noted at the necropsy table a surprising discrepancy between the severity of the pneumonic process and of the toxemia, and the apparent damage sustained by the heart.

Many observers have commented on the fact that the heart in certain virulent, fatal pneumonias may appear to be affected in surprisingly mild degree. There may be little or no dilatation, no apparent incompetence of the valves, in short, seemingly no abnormality of a heart in a patient who has from the onset of the toxemia appeared to be doomed in spite of every effort to combat the disease. Such instances are practically always examples of intense, fulminant poisoning, in the so-called croupous or fibrinous type of the disease. The course is brief, the attack is largely on the nervous centers, and the heart, though always participating in the storm, seems to maintain a remarkable degree of power and equilibrium to the very end.

Not infrequently the microscope will reveal appreciable damage that escapes the naked eye. Clinically, as well as at the necropsy, however, the heart-muscle appears to be the last vital force to give way. Not so, in the many instances of bronchocatarrhal pneumonia,

in which we practically never observe a sthenic patient or a heart which gives either clinical or post-mortem evidence of strength, stability or endurance.

One learns to associate the bronchocatarrhal type of pneumonia with a weak, dilated right heart, and on this basis very early to discriminate it from the croupous or fibrinous form of the disease. The temperature is irregular and low, the pulse running, rapid and often irregular, the patient asthenic far beyond the degree commensurate with the amount of pulmonary involvement—such is the clinical picture of the form of the disease in which the cardiac musculature and the vasomotor nervous system seem to sustain to such a preponderant degree the brunt of the attack. After such a pneumonia the post-mortem examination reveals almost invariably a flabby, characterless heart-muscle, of which incompetence is the main feature and dilatation the most meaningful expression. Routier<sup>2</sup> has recently reported a pneumonia in which the influence of the toxin on the fibers of the auriculoventricular conducting path resulted in a transient heart-block which, disappeared spontaneously. By no means infrequently, however, the walls of both the left and right ventricles are thinned, and all of the valves, pre-eminently the pulmonic and tricuspid, are insufficient. I have one interesting specimen in my collection (Fig. 2) in which the conus arteriosus and the pulmonary artery both show marked permanent stretching and distention, the pulmonic valve appearing wide open between.

### THE MICROSCOPIC CHANGES

I think it permissible to say that the heart shows post-mortem evidences of the toxemia in every fatal case of fibrinous, as well as of bronchocatarrhal pneumonia, that reaches the microscope. In the latter type of the disease the changes are more likely also to be macroscopic. I think it likely that every heart that weathers the pneumonic storm sustains similar lesions, of milder degree perhaps, yet from which it probably never fully recovers.

During the past year I have taken great interest in studying microscopic sections of the cardiac musculature from fatal cases of pneumonia in the wards of the Philadelphia General Hospital. In not a single instance was there a lack of evidence that a toxic influence had been actively at work. Usually the process appeared in the form of an acute diffuse myocarditis, with cloudy swelling of the cells, often with fragmentation of the muscle-fibers. In the great majority of cases the striations had nearly disappeared from the fibers, over wide areas, if not generally throughout the heart. Not infrequently could be found here and there areas showing extensive round-cell infiltration,

\* Read before the Section on Practice of Medicine at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

1. Cooke and Varcroft: Jour. Physiol., 1914, xlvii, 35.

2. Routier: Arch. d. mal. du coeur, May, 1914.

mainly of the interstitial substance between the fibers. In quite a number of instances, as exemplified in Figure 3, there was wide-spread hemorrhagic infiltration. The nuclei of the muscle-cells were proliferated, were often swollen, and here and there vacuoles appeared in their place. Many of the muscle-fibers gave the appearance of being atrophied. In no case except in old persons, or in those afflicted with organic cardiac

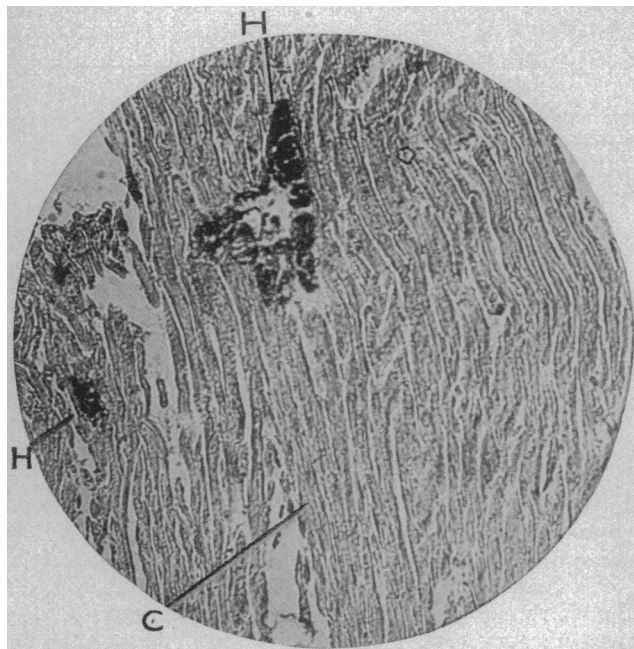


Fig. 1.—The heart-muscle in a case of croupous pneumonia, showing the rich capillary blood-supply to each fiber, the capillaries lying within (or in a groove in) the latter (C); inter-fibrillar hemorrhage (H), and complete loss of striae.

disease, was fatty degeneration of the heart-muscle observed.

Macroscopically as well as microscopically, there was evidence in every heart examined of some degree of pericarditis. I observed not one instance in over a year's time of fresh endocarditis, except such as was superimposed on an old lesion.

#### THE BLOOD-PRESSURE

Before using the blood-pressure as an evidence of either cardiac competence or insufficiency in the pneumonias, it is incumbent on us to show that in a sthenic pneumonia the customary or normal pressure is due to an overacting or at least a capable heart; while in the low-grade or asthenic pneumonias the low pressures are due to an incompetent cardiac mechanism rather than to vasomotor failure. The personal equation will of necessity enter largely into the verdict rendered by various investigators of this question. During the past three years I have studied more or less thoroughly over seven hundred cases of pneumonia on the wards of the Philadelphia General Hospital. Both with the finger on the radial, carotid and apical pulses, and with the sphygmomanometer, I have measured the blood-pressure in very many of these cases. I have a few records of the systolic pressure made in certain sthenic fibrinous pneumonia cases reading as high as 170 and 180 mm. of mercury. Far more often the readings followed approximately the normal line (from 120 to 140 mm.). I refer, of course, only to the active and sthenic stages of the toxemia. I have yet to see in a patient with pneumonia

of the bronchocatharrhal type (except in childhood) an instance of high or even of normal systolic blood-pressure. Even in instances associated with symptoms of nephritis, or with arteriosclerosis of high grade, there has persisted a low systolic pressure, indicating insufficiency of cardiac vigor and driving power.

The diastolic pressure, elicited by the auscultatory method, furnishes a fair index of the vasomotor tone. While this also is usually lowered in pneumonias of the asthenic type, it falls by no means in a measure commensurate or parallel with the cardiac power. Thus the systolic pressure in cases of bronchocatharrhal pneumonia during the stage of active toxemia has averaged not far above 100 mm., while the diastolic pressure has seldom registered below 80 mm., yielding an average pulse-pressure of 20.

This fact, together with such manifest indications of cardiac insufficiency as systolic jugular pulsation, arrhythmia, small rapid pulse, enlargement of the area of cardiac dullness to the right, and often a series of valvular murmurs that can only be interpreted as indicative of muscular (relative) insufficiencies, apparently allow us but one possible conclusion, namely, that in the great number of low-grade pneumonias the heart-muscle must be ever an object of care and concern. Gibson<sup>3</sup> has called attention to a possible prognostic relation of the systolic cardiac pressure to the pulse-rate. Lauder Brunton has made a similar study of the diastolic pressure. In my own experience the systolic pressure has furnished a very uncertain prognostic index, for the reasons already stated. The majority of the low-grade pneumonias have presented a systolic pressure almost constantly lower than the pulse-rate, and yet the vast majority of these patients, when properly treated, have recovered.

#### THE INFLUENCE OF TREATMENT

Considerable evidence may be gained as to the actual condition of the cardiac mechanism through studying the results of treatment. It is a current belief and

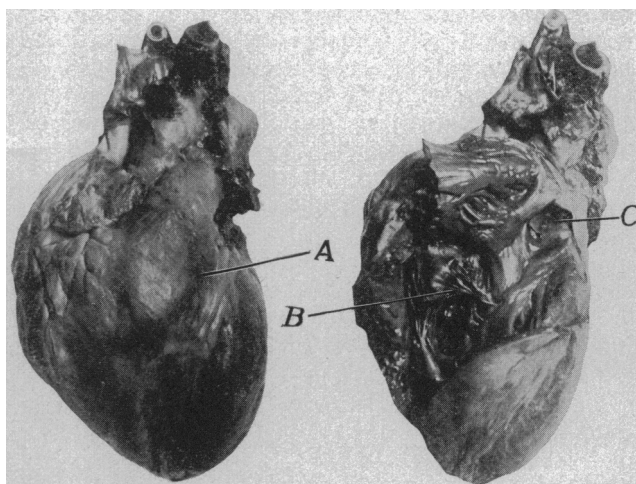


Fig. 2.—Pneumonia heart, showing (A) dilated conus arteriosus and right ventricle from without, and insufficient tricuspid (B) and pulmonic (C) valves.

teaching, employed perhaps more freely than is justified by circumstances, that the pneumonic patient dies not of his pulmonary involvement, and not of his cardiac embarrassment, but owing to paralysis of the vasomotor system. In acute fibrinous sthenic pneumonia there would seem in certain instances to be at

3. Gibson: *Edinburgh Med. Jour.*, 1908, lxx, 22.

least some reason to support this view. In the bronchocatarrhal type of the disease there is exceedingly little. No better illustration could be given of the truth of this assertion than the fact that the high or normal blood-pressure in the fibrinous type appears to be due to overaction of a sthenic heart; while conversely, in the bronchocatarrhal form the asthenia and the almost invariably low blood-pressure appear to be

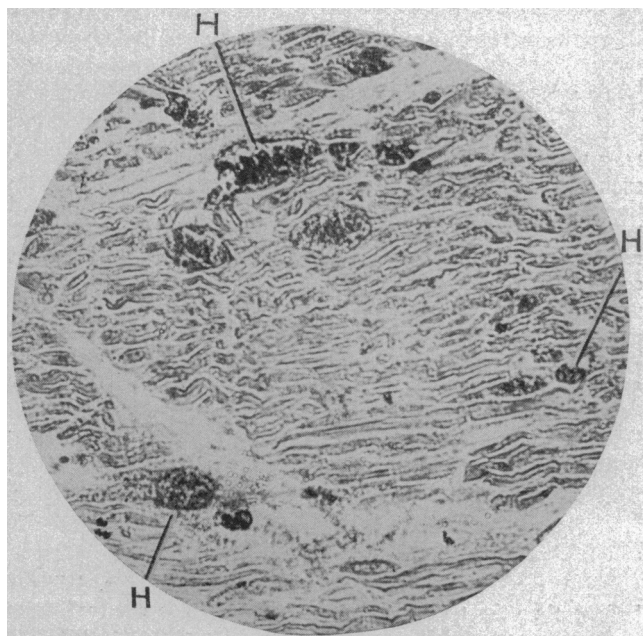


Fig. 3.—Heart-muscle from the left ventricle in a case of bronchocatarrhal pneumonia. Extensive degeneration of the fibers, with widespread hemorrhages (H). Striae completely lost.

due not nearly so much to loss of vasomotor tone as to actual cardiac dilatation and disability. Instances that admit of reestablishment of the cardiac tone do well. The most telling advantage seems to come from the systematic and thorough emptying of the intestinal tract, in many cases the manufactory of the toxic products. When the bowel has been deliberately rendered and kept clean the heart has, in my experience, seemed to have its best opportunity for rehabilitation. The moment the bowel has been neglected the heart has apparently suffered in a measure that is not to be explained on the basis of mere mechanical embarrassment. Such remedies as *nux vomica*, quinin, camphor, digitalis and oxygen in Nature's own form of warm, clean air, may serve as cardiac tonics and, on occasion, as indispensable temporary crutches. Under their influence the blood-pressure rises and may be maintained at a more favorable level.

On the other hand, such drugs as epinephrin and pituitary extract I consider dangerous in the presence of a weak heart. While they are certain to afford a momentary rise in the blood-pressure as the result of their influence on the vasomotor system, I have more than once seen evidence of their mischievous, and possibly lethal, action on a weak heart, through the imposition on its crippled musculature of an additional load in the form of a tense peripheral circulation. Nature knows better than we when to elevate and when to lower the intravascular tension.

In a sthenic fibrinous pneumonia practically any treatment may be employed. We start and finish with the knowledge that in the vast majority of instances,

otherwise healthy, non-alcoholic men and women should recover. Not so with the bronchocatarrhal, asthenic pneumonia patient, who must be supported actively through the disease because his vital forces, especially his cardiac mechanism, are all below par. Many a case of mistreated pneumonia takes on a totally different appearance when placed under conditions that favor and nourish instead of depleting the tone and strength of the heart. Perhaps the best illustration may be had in the misuse of cold air on an asthenic patient suffering from pneumonic toxemia. I have placed many such shivering, cyanotic subjects in warm, clean air and witnessed the immediate reestablishment of forces that were fast beginning to wane. Uppermost and most striking of all appeared to be the influence on the heart, which craved oxygen as its natural stimulant and tonic, but needed it in the form of warm, as opposed to cold, clean air.

#### PREEXISTING CARDIAC DISEASE

A final bit of evidence as to the importance of a thoroughly healthy cardiac mechanism—including musculature, valves, endocardium, ganglia and nerves—is observed in the high mortality of cases that enter on a pneumonic toxemia with preexisting cardiac disease. We have all seen instance after instance of a patient making a bold fight to weather the storm, and experiencing sudden cardiac collapse, either in mid-stream or on the brink of convalescence. Only recently I witnessed an acute pneumonia in a young man of 22 years, an alcoholic, with myocardial degeneration and high-grade mitral stenosis and insufficiency, all sclerotic and probably syphilitic in origin. He went to bed in the beginning of an intense pneumococcus infec-

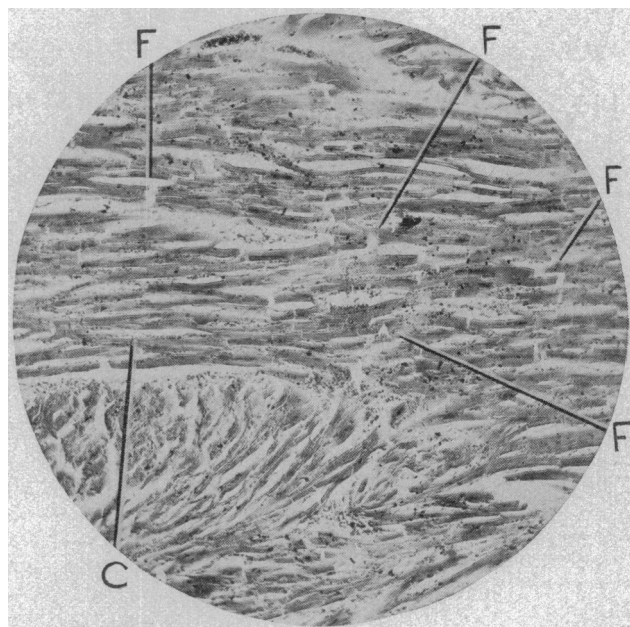


Fig. 4.—Heart-muscle in a case of bronchocatarrhal pneumonia. Striae show here and there, but have mostly disappeared. Considerable fragmentation of the fibers (F). The capillary blood-supply is beautifully distinct (C).

tion with a heart widely dilated, an irregular pulse and edematous extremities. Within two days he showed a steady, remarkable cardiac gain. The jugular pulses became almost undiscernible, the apical pulse regained its regularity, the edema entirely disappeared from the extremities. With these signs of cardiac rehabilita-

tion, the whole aspect of the pneumonia (croupous or fibrinous, as shown at the necropsy) altered. The patient was no longer cyanotic, the breathing became more free, and it appeared almost as though even this desperate case might win out against the acute disease. Suddenly and coincidentally with an apparent crisis, all the signs of cardiac distress reappeared, and in a brief period evident cardiac, not pulmonary, death supervened. The heart had reached its recuperative limit, and could draw no further on a reserve force, because it had no further stock in store. In the same manner a tobacco- or alcohol-weakened cardiac muscle may decide the issue between life and death in an otherwise favorable pneumonia.

#### CONCLUSION

Without attempting to minimize the influence of the vasomotor system in the causation of a high mortality in pneumonia, it would seem as though we ought not to be satisfied, on the basis of either clinical or laboratory experience, to regard the heart in any other

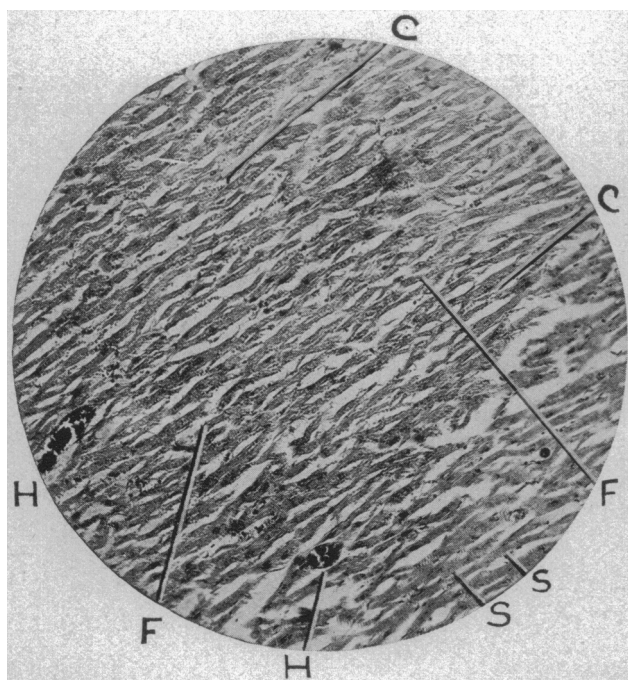


Fig. 5.—Heart-muscle in a case of bronchocatarrhal pneumonia, showing considerable atrophy of the fibers, scattered hemorrhages (H) and extensive fragmentation (F). Striae are seen here and there (S). Capillary blood-supply is exquisite (C).

way than as the factor of prime importance in the safety or danger of the great majority of asthenic pneumonia patients. More and more does the prevalent pneumonia seem to be tending away from the well-recognized, frank sthenic type, to the low-grade, asthenic, bronchocatarrhal forms. The more certainly this is true, the more inevitable will be the necessity of safeguarding the heart from start to finish, not so much by the use of drugs as by their avoidance (especially cardiodepressants like nitroglycerin and alcohol), not so largely by treatment as by the studious prevention of an overdose of solicitous care. Hygiene of the intestinal tract, sane feeding, the intelligent use of an abundance of clean air of the right temperature, and the insuring of ample periodic freedom from the doctor, nurse and family will go farther than any others toward supporting the organ whose staunch cooperation is essential to the happy outcome of the case.

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#### ABSTRACT OF DISCUSSION

DR. JAMES M. ANDERS, Philadelphia: I feel convinced that Dr. Willson is right in stating that pneumonia has been assuming an irregular bronchopneumonic type in recent years to a greater degree than formerly. This fact may be attributable to the more constant and general prevalence of various forms of influenza. The view that death in uncomplicated cases of pneumonia is due to the specific effect of the toxemia on the heart is widely accepted. Perhaps it would be more nearly correct, in the present state of our knowledge, to say that death is caused by the general rather than the local effect of the toxemia. As pointed out by Dr. Willson, the whole vasomotor system is profoundly involved in many cases. The heart is frequently dilated, especially the right side, and filled with firm coagula. It has long been held that if the more usual cardiac complications, such as acute endocarditis, pericarditis and fatty degeneration in protracted cases, be excluded, the changes presented by the myocardium are conspicuous by their absence. The results of the microscopic studies by Dr. Willson, therefore, are not a little surprising, but will constitute a distinct advance in our knowledge of pathologic changes found in the heart in pneumococcal infections, especially if confirmed by the findings of other competent observers. The frequency and seriousness of the disease in question make it desirable that further histologic investigations of the subject be undertaken. I quite agree that cold or fresh air in the treatment of pneumonia is one of the best, if not the best measure at our command, but it should not be indiscriminately employed. It has not a specific influence, but does increase resistance to the toxemia. Again, it should not be employed in cases in which active kidney lesions preexist.

DR. WILLIAM S. THAYER, Baltimore: Does Dr. Willson believe that the type of pneumonia has really changed from a sthenic to an irregular asthenic type? On what does he base his opinion? In over twenty-five years' observation of pneumonia in various regions I have not been able to detect any essential differences in the clinical character of the disease beyond, of course, the common variations in different epidemics. I am quite familiar with this assertion with regard not only to pneumonia but to other acute febrile diseases, especially typhoid fever. I rather think that the old-fashioned "typical case" is essentially a picture in the mind of the observer based on text-book descriptions—an impression so strong that in the course of years it becomes interpreted as actual experience; and that the common impression that such a disease as pneumonia is changing its type is largely based on the circumstance that in practice pneumonia shows frequent variations from this picture. I am not aware that such change in the clinical manifestations of the disease has occurred.

DR. ROBERT N. WILLSON, Philadelphia: In answer to Dr. Thayer's question, I have two grounds for the assertion that the prevalent type of pneumonia is gradually changing from that of the old-fashioned book type of fibrinous or croupous pneumonia to the low-grade or bronchocatarrhal form. First, my own personal experience in a series of several hundred cases, both in hospital and private work, leads directly to such a conclusion. This experience covers and includes considerable necropsy and microscopic work in confirmation of the diagnoses. My second basis of argument is the result of not a few conferences held with men in various localities over the country, who agree with me in my conviction as the result of their experience and study. Dr. Anders' statement, made a few moments ago, in discussing my paper, furnishes a case directly in point. I had not previously heard his position in the matter. I shall welcome similar studies of the heart in the pneumonias as confirmatory of my own. In every instance of pneumonia widespread damage to the cardiac musculature will be found. When this fact is recognized I think we will begin again to regard the heart as the organ and force mainly to be safeguarded and conserved, and to lay a little less stress on the vasomotor factors in grave cases of pneumonia.