

## THE HEART MUSCLE IN PNEUMONIA.<sup>1</sup>

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PLATE 22.

(Received for publication, April 24, 1915.)

It is very generally believed that the heart muscle is seriously injured in pneumonia and that heart failure from this source is a frequent cause of death in this infection.

The experiments presented in this communication show that the cardiac ventricle from dogs that have died from pneumonia contracts as well as the ventricle from healthy dogs, provided the pneumonic muscle is fed with normal blood. When a normal ventricle is fed with pneumonic blood, the contractions are much impaired. If, however, the ventricle from a dog with pneumonia is fed with pneumonic blood, the contractions are almost normal in extent and may be normal in duration.

Thus in pneumonia the heart muscle is essentially normal, whereas the pneumonic blood is distinctly poisonous to heart muscle suddenly fed with it. In the body, during the gradual course of the disease, the blood is progressively affected and the heart muscle gradually adjusts itself to the poison, with striking success.

### *Method.*

The experiments consist of four series of ten dogs each. In the first, the normal ventricle was fed with normal blood; in the second, the pneumonic ventricle was fed with normal blood; in the third, the normal ventricle was fed with pneumonic blood; in the fourth, the pneumonic ventricle was fed with pneumonic blood.

<sup>1</sup> Aided by a grant from the Dalton Scholarship of the Massachusetts General Hospital. A preliminary abstract of the conclusions reached in this investigation was published in the *Boston Med. and Surg. Jour.*, 1915, clxxii, 718.

The contractions of the heart muscle were recorded by a method devised by Porter in 1897.<sup>2</sup> The dog is anesthetized with ether (no morphin), bled from the carotid artery, the blood defibrinated and filtered through glass-wool. Meanwhile, warm normal saline solution<sup>3</sup> is allowed to flow into the crural vein. After a short interval, the dog is bled again from the carotid artery and the blood defibrinated as before. The heart is now rapidly removed and placed still beating in a beaker filled with warm saline solution. Often the beats are so vigorous that the heart with each ventricular systole springs more than an inch from the bottom of the beaker. Thus the organ is self-cleansed from blood. A cannula is now tied into the branch of the left coronary artery supplying the area the contractions of which are to be studied, and the part of the left ventricular wall supplied by the artery is cut out. The cannula bearing the attached ventricular segment is filled with defibrinated blood and joined to a glass tube containing defibrinated blood at a temperature of 23° to 25° C. and at a pressure of 56 mm. of mercury. This tube is surrounded with a larger glass tube through which warm water is circulated to keep the blood at the desired temperature. An adjustable clamp supports the coronary cannula and thus the attached heart muscle in a suitable position. A bent hook is passed through the lower end of the muscle and attached to a light lever magnifying five times. The contractions are recorded on a kymograph moving 40 mm. per hour (Fig. 1). The defibrinated blood in all our experiments was diluted with twice its bulk of normal saline solution.

The work done by the ventricular muscle was judged (1) by the length of the period during which the heart contracted, and (2) by the total area of the contractions. The curves, illustrated by Fig. 1, were laid upon a glass plate illuminated from below with electricity. Over the curve was placed a card and the total area of the curve was carefully traced upon this card. The area was then cut out with scissors, placed with the other contraction areas

<sup>2</sup> Porter, W. T., *Jour. Boston Soc. Med. Sc.*, 1897, i, 15; *Jour. Exper. Med.*, 1897, ii, 391; *Am. Jour. Physiol.*, 1898, i, 514-515.

<sup>3</sup> Sodium chloride, 9.00 gm.; calcium chloride, 0.26 gm.; potassium chloride, 0.10 gm. in 1,000 cc. of water.

in its own group and the entire group weighed in a good balance. The cards used were of uniform thickness. The small errors made in tracing the areas and cutting them out were "accidental errors"; those falling above the true value were compensated by those falling below it. Ventricular strips from large hearts sometimes give higher contractions than the strips from small hearts, thus influencing the contraction area. As there were ten hearts in each group, compensation took place here also, and there is no reason to doubt that for the purpose of comparing contraction areas in groups of ten the method is substantially accurate in a problem in which only marked similarities and differences are of value.

When a kymograph moves so slowly that the contractions of the slowest heart in a series are fused, the contractions of a more rapid heart will overlap. In such a case, the fused curve from the more frequently contracting heart will contain more contractions and should thus express more work than the fused curve of equal height from the less frequently contracting heart. But under these circumstances, the more frequently contracting heart will conceal its extra work and the two curves, although equal in area, will not be equal in work done. In our experiments, this error does not affect the main conclusion, because the great majority of the ventricles beat at nearly the same rate, about 55 per minute. The relatively infrequent variations were chiefly in Series III, in which normal ventricles were fed with pneumonic blood, which seemed to make some of them more irritable. Such ventricles beat more rapidly than 55; their contractions, however, were increased in force as well as in frequency, giving a higher contraction area and thus compensating in part for overlapping. At the most, the error from occasional overlapping disappears in comparison with the wide difference in performance between the normal ventricle fed with normal blood and the normal ventricle fed with pneumonic blood.

The organism employed was the *Bacillus pneumoniae* (Friedländer) obtained from the stock culture in the Bacteriological Laboratory of the Harvard Medical School through the kindness of Dr. Sisson. It was passed through three guinea pigs to increase

its virulence to such a degree that 1 cc. of a broth culture injected into the peritoneum killed a guinea pig in twelve hours. Its virulence was kept at this point by occasional passage through additional guinea pigs. Broth cultures incubated from eighteen to twenty-four hours were used for injection into the trachea. The dose was 5 to 7 cc. per kilo.

The culture was administered as follows:<sup>4</sup> The dog was given subcutaneously from 0.5 to 1.0 cc. of a 3 per cent solution of morphin sulphate so that it might not cough up the culture. From thirty to sixty minutes later, the dog was placed on the operating table, etherized, the jaws held open by an assistant, the tongue drawn forward and upward, the epiglottis drawn forward, and a tube passed through the glottis into a bronchus, usually the right bronchus. The culture was then forced through the tube with a syringe, and the dog was taken from the table and placed in a sick room to await the disease. Many of the dogs were at the point of death as early as eighteen hours after the inoculation.

In six of the ten experiments in which the pneumonic heart was fed with normal blood, the dogs were allowed to die of the disease, to forestall critics who might say that the dogs could not have been very ill. In one of the remaining four, the temperature had fallen to 38° C., and the respiration was very rapid and labored; the second of the four was entirely insensible to pain; the temperature of the third had fallen from 40° to 36° C.; and the fourth was in coma with a temperature of 32° C.

In the ten experiments in which the normal ventricle was fed with pneumonic blood, the normal ventricle was taken from ten healthy dogs and the pneumonic blood from ten dogs in that stage of the disease that just precedes death. The temperature of these animals had begun the fatal descent which in dogs with pneumonia is the precursor of the end. It had been 40° to 41° C., and the operation was performed when the temperature had fallen to 38° or 37°, sometimes as low as 34°. Five of the dogs were in complete coma; these dogs were entirely insensitive to pain.

In the ten experiments in which the pneumonic heart was fed

<sup>4</sup>Lamar, R. V., and Meltzer, S. J., *Jour. Exper. Med.*, 1912, xv, 133.

with pneumonic blood, the blood of each animal was used for its own heart preparation. In nine of these animals, the fatal descent in temperature had begun. Six of them were entirely comatose. The tenth dog in this series was not used until the respiration had ceased and the pulse was absent from the exposed carotid artery. The dog was thereupon revived with normal saline injections and artificial respiration until the blood had been withdrawn and the heart extracted. In every experiment upon a pneumonia dog, an autopsy revealed an extensive consolidation of the lung.

### Observations.

TABLE I.

*The Average Duration of Contraction and the Total Weights of the Contraction Areas in Four Series of Ten Dogs Each.*

Preparation.	Average duration of contractions. <i>min.</i>	Total weights* of contraction areas. <i>gm.</i>
Series I. Normal ventricle fed with normal blood...	181	8.84
Series II. Pneumonic ventricle fed with normal blood	187†	8.46
Series III. Normal ventricle fed with pneumonic blood	70	3.40
Series IV. Pneumonic ventricle fed with pneumonic blood .....	176	6.46

\* Each contraction area was cut out of a thin card. These cards were of uniform thickness, and 3.5 sq. cm. of card weighed 0.1 gm.

† By a clerical error, this figure was given as 200 minutes in the abstract published in the *Boston Med. and Surg. Jour.* (Newburgh and Porter, *loc. cit.*).

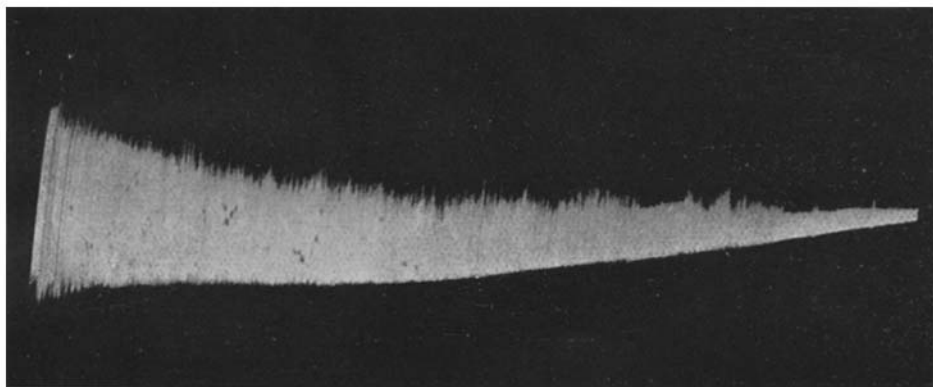
The forty experiments on which our conclusions are based are presented in Table I. It is at once evident that the results in Series I and II are identical. The pneumonic ventricle fed with normal blood contracts as long and as vigorously as the normal ventricle. In Series III the normal ventricle is fed with pneumonic blood, under which condition the duration of contraction and the contraction area are little more than one-third the normal value. In Series IV, in which the pneumonic heart is fed with pneumonic blood to which it had been exposed during the course of the disease, the duration of contraction is but 4 per cent, and the area of contraction 27 per cent less than normal.

## CONCLUSIONS.

1. The heart muscle is not functionally impaired in pneumonia, since the pneumonic ventricle beats normally as soon as its food is normal.
2. Pneumonic blood, suddenly fed to normal heart muscle, lowers its efficiency, lessening the duration and the area of contraction.
3. The heart muscle in pneumonia, exposed gradually to the action of the poison, largely adjusts itself to its poisoned food.

## EXPLANATION OF PLATE 22.

FIG. 1. Original size. Experiment of May 17, 1915. Contractions of the part of the left ventricle supplied by the first portion of the circumflex branch of the left coronary artery. The preparation was perfused with defibrinated blood mixed with two parts of normal saline solution. The blood was from a healthy dog. The lever magnified five times. May 16, 3 p. m., 45 cc. of a broth culture of Friedländer's bacillus were injected into a bronchus. May 17, 9 a. m., the temperature was 41° C. At 6 p. m. the temperature was 38°, the dog was completely insensible, and no ether was required during the operation.



6.55 p. m.

9.45 p. m.

FIG. 1.  
(Newburgh and Porter: The Heart Muscle in Pneumonia.)