at this time we are taking cultures for diphtheria bacilli of all sick and wounded returning from overseas (from 4,000 to 5,000 a week and up to 2,000 at a time) and getting results showing high efficiency of work on a massive basis. There is no evidence that the results on our large numbers of cultures were less accurate than were the results of other workers who took cultures of only a few at a time. In fact, we got better results when we were hard pushed then when we had less work, for when there was much to do the officers were more keen.

2. The taking of cultures of whole organizations or even entire camps need not interfere with military training; taking out the carriers and putting them in a carrier camp interferes less than quarantining even only the contacts.

3. Detecting and taking the carriers out of organizations is of value, and the routine taking of cultures of the whole command seems indicated if the troops come from areas where epidemic meningitis is endemic.

4. Detected carriers have the disease, but our data seem to show that those that developed the disease were already in the incubation period when the cultures were taken. We have no evidence that chronic carriers developed the disease.

5. The cotton swab on a straight wooden applicator, the cultures being taken from the nasopharynx through the nose, gave us good results, and its use is much more feasible than the West tube and bent wire applicators when cultures of large numbers of troops are to be taken.

Sixth Avenue and Eighteenth Street.

THE PATHOLOGIC ANATOMY OF INFLUENZAL BRONCHOPNEUMONIA *

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In this report, based on approximately 200 necropsies, no attempt will be made to distinguish between changes due to the unknown virus and those from mixed or secondary infection; only the most outstanding features are discussed.

Perhaps the most lasting impression from long association with lobar pneumonia in postmortem examination is that when it alone is responsible for death, with very few exceptions,[1] a considerable part of the total pulmonary parenchyma is consolidated, undistensible and heavier than normal; even when limited to one upper lobe, that lobe is as a rule huge, and the lung weight as a consequence frequently doubled.

Therefore, the first feature of the lungs in influenza to attract attention was the relatively small amount of lung tissue solid with grossly demonstrable pneumonia. Even when measured, the total of such regions is so small that it is difficult to ascribe death to the pneumonia with an assurance at all comparable to that with which death is accounted for by lobar pneumonia; in fact, the amount of lung tissue in influenza actually pneumonic seems too little in many cases to explain death by pneumonia. This is best shown by distending the lungs with air, for many of the dark red or purple places, apparently airless and semisolid, balloon out and become quite pink. When surfaces are made by cutting tissues actually pneumonic from influenza, it is not common to find gray or gray-red and finely granular plugs of fibrin and cellular exudate which come away on the knife, as is the case in lobar pneumonia and most bronchopneumonias other than those of the influenzal type. Such surfaces in the nondistensible places in influenzal pneumonia are wet, "velvety" and not granular, but smooth; they resemble raw meat or wet skeletal muscle. The redness of the button-like, firm, superficial regions of pneumonia, likened by some observers[2] to hemorrhagic infarcts, is due perhaps as much to the well-preserved cells of the exudate, many of which are mononuclear, as to the blood content.

However, ranking in importance with the relatively small amount of actually pneumonic lung, or perhaps entitled to first place as a conspicuous feature, is the huge, often thin and watery bloody exudate in the lung tissue and bronchioles. This bloody fluid, on the development of rigor mortis, often pours out of the nostrils so as to stain a large part of the white sheets in which bodies are wrapped. It mainly is responsible for the low level to which the lungs sink when put into water and for the total submersion of some; many are like the lungs of the drowned. The fluid mixed with air and forming a blood-tinged froth is abundant in the trachea, larynx and large air passages after death. Accumulating under the visceral pleura, it makes one of the most interesting and tell-tale signs of influenza; for normally the pleura, fitting the underlying lung tissue snugly as a transparent membrane, allows every detail of the coal-dust mosaic-like markings, as well as the coarser details of the pink, air-containing vesicles, to be clearly seen; but, with the presence of this bloody fluid in the subpleural lymph vessels in abundance so as to distend them, there are produced opaque, reddish brown places, usually a few centimeters in diameter, frequently seen at once when the sternum and costal cartilages are removed. They are so distinctive that influenza is at once suggested as the cause for death; and when no clinical details are known, are highly important clues as to the cause of death. This dissection of the outermost layers of the pleura is very common in the angles formed by fissural surfaces—in the bottom of the cleft. Small hemorrhages in the pleura are also common.

Another feature also due to this bloody exudate is that usually there is some fluid of the kind just described in one or both pleural cavities—more, as a rule, on the side of greater lung involvement. Now, with most pneumonias the pleural exudates are not so blood tinged. With lobar pneumonia they are not only commonly yellowish but also heavy with fibrin; large masses of yellow fibrin wet with plasma often make the lung in lobar pneumonia actually "shaggy"; this I have not seen in influenza. There is commonly some fibrin on the lung, but search is often necessary to find it. It is seldom more than 1 mm. thick, and is often less. The amount of the blood-tinged fluid in the pleural cavities varies widely. When measured, from 25 to 100 c.c. are frequent; larger amounts have been weighed, and the following weights will serve as examples of these exceptional instances: fluid in the

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1. Compare report by Clark and Batman (J. Infect. Dis. 1: 229, 1904) on capillary bronchiolitis, with the symptoms of lobar pneumonia including a crisis.

2. Abstracts of Foreign Literature Compiled by the British Medical Research Committee, J. A. M. A. 71: 1575 (Nov. 9) 1918.

right pleural cavity, 840, 345, 860, 600, 320, 930 and 600 gm., each one case; fluid in the left pleural cavity, 900, 1,000 and 320 gm., each one case; 660 gm. in the right with 800 gm. in the left; 1,120 gm. in the right with 1,140 gm. in the left, each one case. These large collections of fluid are in no sense "empyemas." The fluid is only slightly turbid. When time has elapsed for sedimentation after death, the fluid taken for bacteriologic examination, if the pipets are not introduced too deeply, is quite transparent and clear, often with a little of the lilac tint of hemolysis; that collected from deeper layers is turbid.

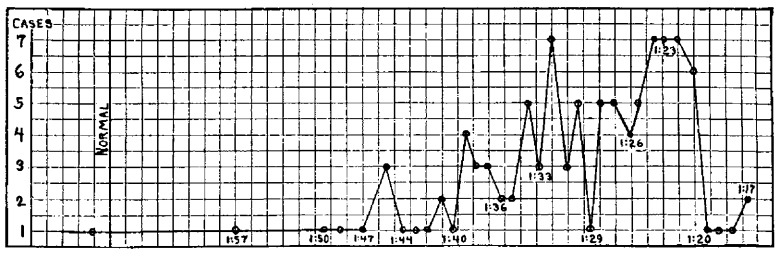

Chart 1.—Ratios of lung weight to body weight in 103 cases of lobar pneumonia from necropsies in 1915 and 1916.

This bloody exudate is not pus in the sense that pus is the product of liquefaction necrosis; there is no suppuration. Postinfluenzal empyema, however, does occur, as well as empyema with repeated thoracentesis. This thin, characteristic, bloody exudate of influenza contains only a little fibrin; or none at all may be visible grossly. It is unlike the pleural exudate of any other form of acute pneumonia with which I am familiar. It reminds one most of the inflammatory exudates in such places as the arm from acute sepsis following wound infection, or the thin, bloody fluid of woody phlegmon of the neck; it is more like exudates due to streptococcus than those we are familiar with in pneumococcus inflammations. The fat of the thymic body and other mediastinal fat, as well as all of the tracheobronchial lymph glands, are also often thoroughly wet with this fluid.

Since so much fluid was found in the lungs and as part of an exudate which in many instances escaped into the pleural cavity, less because of pleuritis there than because the lungs, so to speak, "overflowed," the lung weights were compared with the weights of lungs in lobar pneumonia. This comparison is shown by the accompanying charts of the ratio of lung to body weight.[3] From the charts it is apparent that the ratio of lung weight to body weight in lobar pneumonia and influenza are roughly similar; whereas with lobar pneumonia, the precipitate of fibrin is considerable, other parts of the pulmonary parenchyma being compensatorily emphysematous, in influenza, the pneumonia is less and the increased weight due in large part to fluid.

The pneumonia of influenza is commonly referred to as bronchopneumonia. It may be so designated, but it differs from other bronchopneumonias in its predilection for the periphery of the lungs and in the extent to which the inflammation is hemorrhagic.[4] These features, as well as the relatively little consolidation, are well shown by the following account, which may be taken as fairly typical of conditions found with death early in the disease:

The upper lobe of the right lung is easily ballooned out with air, likewise the middle lobe, so that the black places due to coal dust are very black, the intervening lung tissue pale gray or pink. Except for a place 4 by 2 cm. on the mesial surface of the upper lobe where fibrous adhesions were torn, the pleura is smooth. There are two solid places in the back of the upper lobe, and the pleura covering them is a little swollen. These are 3 by 4 by 5 cm., not at all sharply outlined, with moist gray plugs (hand lens) in the air sacs which cut so as to leave smooth wet surfaces; a thin, watery red fluid comes out of the minute bronchioles in them. The lung tissue around them is very wet but contains air; their bronchi contain a thin fluid with opaque flocculent masses in it. Another similar solid place near the front margin of this upper lobe, 5 to 6 cm. in diameter and 1 cm. from the pleura, has a good deal of blood in it. A solid place is in the back of the lower lobe, like the others but with a layer of fibrin on the pleura over it, as thin as transparent paper. This is also very superficial, and the bronchus to it is full of a frothy, thin, red fluid. A fifth place, from 5 to 6 cm. in diameter, less sharply outlined, with thin fibrin on its pleura and 2 cm. deep (the third dimension), is in the lower lobe near the diaphragmatic surface, which is about one-third covered by fibrin. All the bronchi to this lobe contain these flocculent masses, some adherent to the lining. In the depths of this lobe about the large bronchi there is some consolidation 1 to 2 cm. wide, sharply outlined and deep red. Except in these solid places this lobe distends easily with air, as does the middle lobe, which has no solid places.

The pleura of the left lung is smooth, and both lobes distend completely with air except in two solid places 2 to 4 cm. in diameter, one in the front of the upper lobe and 1 cm. deep, dark red and apparently full of blood, the other in the lower lobe and in all essentials similar. The first of these has sharp margins at lobule septums, and the pleura covering it is raised and translucent. In the smaller bronchioles of the left lung there is a little creamy mucopus.

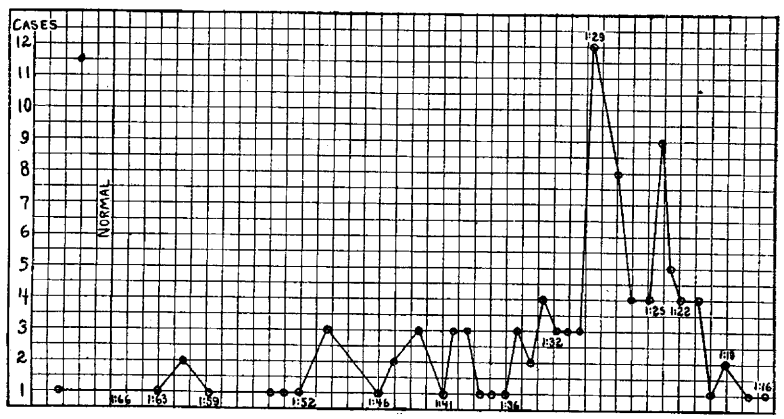

Chart 2.—Ratios of lung weight to body weight in 100 cases of influenzal pneumonia.

There was a little slightly turbid bloody fluid in each pleural cavity.

Next in importance as a characteristic of this disseminated pneumonia, hemorrhagic in its early stages

3. The normal line is the 1.5 per cent. of the body weight, or 1:66, as given by Vierordt (Anatomische, physiologische und physikalische Daten und Tabellen, Ed. 3, 1906, p. 44).

4. Symmers, in consequence of the hemorrhages, has compared influenzal pneumonia to that of pneumonic plague (J. A. M. A. **71**: 1482 [Nov. 2] 1918).

and phlegmonous later, is the change in the medium-sized and smaller bronchioles to the involved lung tissue. Early their linings are simply red, their channels filled with a thin, blood-stained froth or fluid; later the linings are necrotic and their content either a thin mucopus resembling that of early gonorrhea or such a mucopus with small flocculent gray or gray-brown masses in it; slight dilatation of the smaller bronchi is common.

The brain substance is quite regularly swollen, the convolutions of the cerebrum flat; and although this may be evident at once by examining the head first, it is also very well shown by comparing segments made by coronal sections of influenzal brains after hardening in formaldehyd with other brains similarly treated; they are not so swollen as are the brains of septic abortion or other forms of generalized streptococcus infection, nor so pale; but the even external contour and narrowness of the lateral ventricles is a conspicuous feature of influenza; this edema of the brain substance is less than that of uremia or heat stroke.[5]

Still another feature of influenza is that the spleen is not so large as in lobar pneumonia. This statement is a general one and is made without reference to the stage of either disease. The kidneys are regularly the seat of cloudy swelling, and have tense capsules. If any gross alteration of the liver deserves mention as fairly constant, it is the presence of patches of yellow on the front surface due to superficial fatty change.

Less constant are generalized icterus; the early appearance of staining of the lining of large blood vessels by laked blood; patches of necrosis of the larynx or trachea lining; hyperplasia of the lymphoid tissues of the thorax and neck; slight acute serofibrinous pericarditis; acute interstitial emphysema of the lungs and mediastinal tissues, or of these with subcutaneous tissues as well; otitis or accessory (nasal) sinus inflammation, or actual suppuration as small abscesses in the lungs.

It is difficult to believe that a disease with so many distinctive features and affording, as it has, so much of novelty in pathologic anatomy can fail to possess a correspondingly definite etiology.

A more complete report with results of microscopic studies will be made later.

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5. McKenzie, Pierce, and LeCount, E. R.: Heat Stroke, with a Second Study of Cerebral Edema, J. A. M. A. **71**: 260 (July 27) 1918.

Children in the Connecticut Courts.—Prior to 1917, Connecticut had no special laws governing the trial of children. They were subject to the same legal processes as adults. A law passed in 1917, provides that no child under 14 shall be committed to a jail or common lockup while awaiting trial. In a report on the subject named, prepared by Prof. William B. Bailey of Yale University for the National Children's Bureau, the need for better care of dependent and delinquent children in Connecticut is brought out. Mental examinations to determine the mental condition of delinquent children are rarely had, and repeaters are numerous, until it is determined by a mental examination that such children are feebleminded. In New Haven 692 children were brought before the courts in 1914 and 1915, of whom 672 were boys; most of them for trivial offenses, the most serious being that of a boy of 6 for burglary. The report points out that one fourth of the children brought before the courts in New Haven were from families which had at one time received aid from the associated charities, though the connection between poverty and delinquency is difficult to establish.

THE DIAGNOSIS OF MITRAL STENOSIS

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Mitral stenosis has not been generally regarded as a common valvular lesion. In the examination of 25,813 soldiers, mitral stenosis was diagnosed in 0.56 per cent., while the diagnosis of mitral regurgitation was made in only 0.43 per cent. In a previous communication, the strict criteria used in diagnosing mitral regurgitation were recorded.[1]

The definite recognition of mitral stenosis is based on the presence of a presystolic or diastolic, apical murmur[2] with or without other classical signs or symptoms. The characteristic type of heart's action is a physical sign of paramount importance. In well-marked cases, when there is hypertrophy, the almost ringing first sound, the accentuated second pulmonic, the small pulse and symptoms, the diagnosis is simple. However, in examining young adults from 21 to 31 sent to the various camps by the local boards, these obvious cases are weeded out. There remains a certain number of cases with no symptoms and no cardiac hypertrophy. The latter group requires very careful study. As a class these men are remarkably free from symptoms, and resent not being allowed the privilege of serving. The pulse rate is slow, the response to exercise usually excellent. The diagnosis rests purely on the presence of a presystolic murmur.

The relationship of a history of acute articular rheumatism in these cases was investigated. In 168 cases we obtained a definite history of rheumatic fever in sixty, or 39.1 per cent. From the remainder, histories of sore throats, scarlet fever, pneumonia, smallpox and other infections were obtained.

In order to obtain some comparative figures, 100 normal recruits were questioned for a history of sore throats. Ninety-five gave a definite history and forty-six of such severity as to confine them to bed. By a comparison of statistics, it was impossible to determine a relationship between previous infection and endocarditis. It is obviously unsound, after determining that a patient has mitral stenosis and has had tonsillitis, to say that the tonsillitis is the cause of the endocarditis.

The recognition of the murmur of mitral stenosis in these early cases requires careful study and examination. It has not been my experience that I have been able to diagnose mitral stenosis after exercise that I have not suspected before exercise. It is true, however, that it has been suspected in far more cases than it has been proved, especially in the recruits with tachycardia. In a recruit with a slow-acting heart, with a snappy or split first sound and a ringing second, unless the soldier is exercised, cases will be overlooked. It is necessary to auscultate in the upright as well as in the reclining posture. It is often of service to have the soldier reclining and well over on his left side in order clearly to bring out the murmur. The variability of the murmur is surprising. It may be heard in any posture, usually best in the reclining. In one instance,

1. Rothschild, Marcus A.: The Diagnosis of Mitral Regurgitation (Valvulitis), J. A. M. A. **72**: 327 (Feb. 1) 1919.

2. The so-called Flint murmur occasionally heard in aortic insufficiency must be differentiated. This differentiation is a matter of extreme difficulty and is based purely on the personal equation. It is my opinion that a presystolic murmur heard in a definite case of aortic insufficiency would better be regarded as evidence of mitral stenosis when there is a definite history of acute articular rheumatism.