

entered very ill, November 1, having been ill one day. He appeared slightly cyanotic and dyspneic. Pneumonic complications were anticipated, though the chest signs were negative. The common symptoms, however, were present. Despite a favorable blood count, the pneumonic signs increased, and the clinical course became progressive; also the urinary chlorids were diminished, and general acidosis was marked. A transfusion was performed on the seventh day. The temperature dropped to that of a slightly delayed resolution type. The laboratory and clinical disturbances became restored to normal types on the fifteenth day.

CASE 24.—P. McK., aged 35, a well developed man of good physical status, entered on the second day of illness with typical influenza. He was given sodium salicylate with no apparent effect. Pneumonia developed on the fifth day. Transfusion was performed on the seventh day. Uneventful convalescence followed, and the patient was discharged on the thirteenth day.

CASE 25.—R. B., aged 20, a man of slender build, entered the hospital on the second day of illness with typical influenza. He was given sodium salicylate. There was a typical influenza curve of temperature. A secondary rise in temperature with diffuse bronchopneumonia was noted on the sixth day. Transfusion was performed on the seventh day with the resultant typical temperature drop and clearing of symptoms. The patient made an uneventful recovery except for slight otitis media. He was discharged on the sixteenth day.

CASE 26.—W. H. H., aged 32, well developed physically, presenting a case of toxic type and a white cell count of 5,000, entered the hospital on the second day of illness with typical influenza symptoms. He was given sodium salicylate with apparently good results. On the fourth and fifth days he was given 10 grains of quinin intravenously with no increase in the leukocyte count. Pneumonia developed on the fifth day, and transfusion was performed on the sixth day. The general status and white blood count were much improved, the white cell count reaching 12,900; but the high temperature continued. The temperature became normal on the tenth day, and an uneventful convalescence followed. The patient was discharged on the sixteenth day.

CASE 27.—A. J., aged 23, of slender, tuberculous type, entered on the first day of illness with typical influenza. Sodium salicylate was given without effect. The patient developed typical bronchopneumonia of the central type on the fifth day. The white blood count was 2,300. Transfusion was performed on the sixth day. The white blood count was from 4,950 to 9,000. There was a slight secondary rise with decline by lysis. Convalescence was interrupted by pleurisy of the plastic type on the seventeenth day. For four days there was high tem-

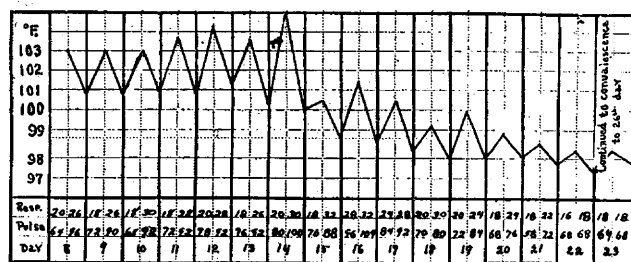


Fig. 8.—Clinical chart, Case 19.

perature. The patient was discharged convalescent on the twenty-fifth day.

CASE 28.—L. D., aged 21, a well built man, entered on the second day of illness with typical influenza symptoms. A typical diffuse bronchopneumonia became evident on the fifth day, and transfusion was performed on the sixth day. An initial drop and secondary rise followed with a resulting clearing up of disturbances. This patient was very toxic prior to transfusion, which condition cleared up early in convalescence. The patient was discharged on the fourteenth day of illness in perfect convalescence.

## CONCLUSIONS

1. The cause of influenza is an intoxicant of unknown origin.

2. The clinical course and pathologic physiology indicate that this intoxicant acts primarily on the central nervous system, with especial reference to the medullary centers. Vasomotor paresis is a prominent and constant feature.

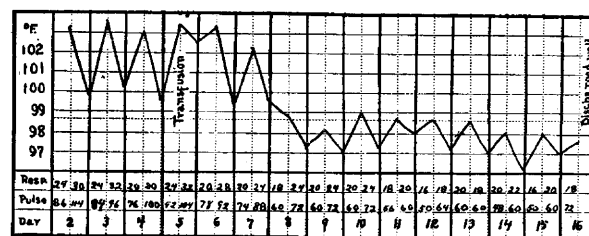


Fig. 9.—Clinical chart, Case 20.

3. Theoretical deductions and practical application affirm that a natural or an acquired immunity is corrective to influenza and its complications. Immune total blood transfusion is theoretically correct, and practical application has affirmed this.

4. The transfusion of total citrated immune blood with proper technic is a very simple and safe procedure.

5. The fulminant nature of the pneumonic complications of influenza demands the very early use of transfusion. Its use is of no avail if the reserve powers of nature have been allowed to progress to an incorrectable degree.

## EPIDEMIC MENINGITIS AND DETECTION OF MENINGOCOCCUS CARRIERS \*

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Early after the mobilization of troops, epidemic cerebrospinal meningitis appeared. It was my opportunity to be stationed at one of the cantonments in an endemic area and later to be at the port of embarkation from which most of our troops were sent overseas.

The English had developed means for detecting carriers, but in this country the monograph of Flexner was used largely as the guide. The steps are:

1. Selection of those from whom cultures are to be taken.
2. Taking of cultures with the West tube.
3. Inoculation of veal glucose serum agar.
4. Selection of colonies.
5. Identification of organisms by:
  - (a) Morphology in stained specimens.
  - (b) Agglutination tests (polyvalent serum and normal horse serum).
  - (c) If found to be meningococci, agglutination tests with the various monovalent type serums.

This system we tried to follow, and from it developed our methods. Oct. 16, 1917, we had our first case of meningitis. The laboratory was not equipped, and only Lieut. (now Major) O. F. Broman and I were ready to go to work. We made West tubes, poured plates, and went to the barracks from which the patient

\* Because of lack of space, this article is abbreviated in THE JOURNAL by the omission of certain tables. The complete article appears in the author's reprints.

in the hospital had come. Here we learned that this man had been only one day with his present company, and having no special friends in the company, visited with most of the men. Furthermore, he had been in Company 62-164 D. B. until the day before. When we came to select contacts, we found that we had the company of more than 200 men of Company A, 354th Infantry, and the company of more than 200 men of Company 62-164 D. B., or more than 400 cultures in all to take.

We started at our task and completed it under the greatest of difficulties. The next day cases occurred in other organizations, and cultures of more companies had to be taken. In a very short time we decided that it is hard to determine the contacts in the Army, that friends do not sleep or sit near each other, and that carriers are found to be widely distributed in the company or even in the camp. On these matters we have the concrete data which bear out these assertions, and it is our opinion that to be of most value, companies must be examined for meningococcus carriers when a case occurs. It also developed that taking cultures with the West tube is not feasible or practicable. In place of West tubes we substituted at first only a few nasal swabs, gradually more, and finally we used altogether the nasal swab.

#### METHOD OF TAKING CULTURES

Thin cotton swabs on small diameter wooden applicators are prepared, and usually ten are sterilized in one test tube. To get the culture the nose is slightly pressed back by the tip, the mouth held open, and then the swab is carried back and downward, usually without touching the sides, clear to the posterior wall of the nasopharynx. Then the swab is rotated and again carefully withdrawn. The plate is inoculated directly with the swab unless there is much mucus, when a spreader is used. Before beginning to take cultures, the men in the company are lined up in two lines, the doors are locked, the windows are closed to get the air as quiet as possible, and a clerk on each side of the table takes the name of the soldier and gives him a number, which number also is used to indicate his culture on the agar plate. Usually the one clerk gives odd numbers and the other gives even numbers. The check list is marked to indicate the organization from which cultures are taken, and it is turned in to the clerk at the laboratory. The medium used at Fort Riley consisted

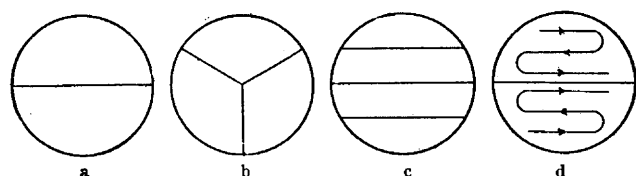


Fig. 1.—Division of plates into sections (a, b and c); method of making inoculation streak (d).

of the regular glucose veal serum agar as advocated by Lieutenant-Colonel Flexner. Later Huntton's agar to which laked human blood is added to produce a slightly pink color was used and found to produce more and larger meningococcus colonies.

When possible, the plates are incubated over night in the inverted position before they are inoculated. As we did not have enough Petri dishes, at first we made cultures from two individuals, later from three, and at times from four on the same plate (of course, we preferred not to use less than one-half the area of a plate for each person from whom a culture was taken). The plate was divided into from two to four sections by marking on the bottom of the Petri dish with a wax pencil as is shown in Figure 1 a, b and c. The segment of the plate is inoculated directly with the swab,

unless there is much mucus, when a spreader is used. The inoculation streak is made as is shown in Figure 1 d without rotating the swab. The segment is numbered to correspond to the soldier's number on the check list. The plates are kept warm and put in the incubator. The colonies are selected after from eighteen to twenty-four hours' incubation, the small translucent dew drop colonies being ringed with the wax pencil. When several colonies from one segment were selected, the colonies were lettered a, b, and so on. It was soon decided that unless none of the colonies could possibly be meningococci, the most meningococcus-like colonies should be ringed. Slides are then divided into ten spaces and numbered 0, 1, 2, 3 and so on. A drop of distilled

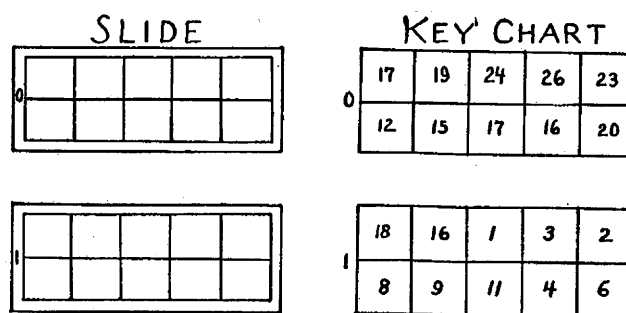


Fig. 2.—System of checking.

water is placed in each segment of the slide, and a key similarly ruled is made on paper; then as a portion of a colony is transferred to the center of a segment on the slide, the soldier's number is put in the corresponding square on the key chart. The plates from which colonies are placed on one slide are put up in one pile. The system may be explained by an illustration (Fig. 2).

After drying, the slides are fixed, and stained by Gram's method, with a counter stain of safranin or Bismarck brown, and the gram-negative diplococci noted.

After going through the plates of a whole organization, the worker has only the numbers of the probable meningococcus colonies to deal with. His list might read like this: 6, 21, 13, 190; and these he may have found on Slides 0, 2 and 18. Then the worker takes from Pile 0 the plate containing Colony 6, from Pile 2 the plate containing Colony 21, and so on. Slants of veal glucose blood agar or veal glucose hormone agar enriched with laked blood are inoculated from what remains of the colonies that are possibly meningococci. The tubes are incubated over night; but if the results must be given at once (that is, within twenty-four hours after taking the cultures), Krumwiede's slide agglutination method is used, that is, finely divided growth is added to polyvalent antimeningococcus serum and to normal horse serum as a control. The growth on the slant medium the next day is washed off in sterile salt solution and added to polyvalent antimeningococcus serum with a final dilution of 1:200, and to normal horse serum with a final dilution of 1:50. We really preferred diluting the serum properly and then mixing in carefully a portion of the agar growth. The tubes are then put in the 55 C. incubator for from four to six hours. If there is agglutination only in the polyvalent antimeningococcus serum, the gram-negative diplococcus is a meningococcus; but if there also is agglutination in the normal horse serum, it is not so regarded.

The technic is satisfactory but tiring. It is possible to train officers readily in the method. At Camp Funston and in the camps of the port of embarkation we had frequently to give results early; but it was regarded better to take out those suspected of harboring true meningococci than to wait until the second day for the absolute, final diagnosis. We may miss a portion of the carriers, but we certainly get those harboring any number of meningococci, and it is essential to remove all the infected men as soon as possible.

As time went on, more cases of meningitis developed at Camp Funston, the disease was occurring in new organizations, and it became evident that we must go still farther to check the disease. As we had been able to accomplish results in the control of meningitis in organizations from which cultures had been taken, it was decided to take cultures of the entire command, about 40,000 troops. This seemed impossible, but it was accomplished.

Six officers from the medical officers' training camp were selected as nasopharyngeal swabbers, nine officers were placed in the laboratory, and the enlisted men worked in day and night shifts. Whether the efforts were worth while and whether it is worth while to get carriers is to be judged from the results obtained and herewith tabulated.

#### EPIDEMIC MENINGITIS AT CAMP FUNSTON

The first case of meningitis occurred Oct. 16, 1917, in Company A, 354th Infantry. October 17, there were two cases in Company H, 353d Infantry, and one in Company B, 353d Infantry. The cases that occurred and the organizations involved are shown in Tables 1 and 2. In all there occurred, from

TABLE 1.—CLINICAL CASES OF EPIDEMIC MENINGITIS  
ARRANGED ACCORDING TO MONTHS AND  
ORGANIZATIONS

Month	89th Division	92d Division	M. O. T. C.	13th Cavalry	Civilians
October, 1917.....	15	..	..	1	2
November.....	75	..	..	0	0
December.....	30	4	1	0	2
January, 1918.....	12	5	4	..	0
February (3d inc.).....	0	0	1	..	0
	132	9	6	1	4

TABLE 2.—CULTURES TAKEN FOR THE DETECTION OF  
CARRIERS, AND CARRIERS FOUND

Month	Cultures Taken	Carriers— Number Per Cent.
October (16 to 31 inc.).....	4,712	136 2.8
November.....	11,337	532 4.7
December.....	37,435	1,152 3.1
January, 1918.....	48,695	1,470 3.0
	102,179	3,290 3.22

October 16 to February 3, 148 cases in eighty-three organizations, and two cases in civilians. On the reservation there were at the outbreak of the disease about 50,000 troops in regular organizations of the 89th Division, the 164th Depot Brigade, 13th Cavalry and medical officers' training camp. October 15, there was a considerable interchange of soldiers, men being sent from the 89th Division to the 164th Depot Brigade and from the 164th Depot Brigade to the 89th Division, so that on the day before the first case occurred, men were sent to and from practically every organization in the camp. All of the men except those of the 13th Cavalry were from an area in which meningitis is endemic. In December, the 92d Division (colored) was filled up with men from various parts of the United States, and large numbers of men were sent to the medical officers' training camp. The cases of epidemic meningitis arranged according to months and organizations are shown in Table 1.

In the various organizations, 102,179 cultures were taken. To these must be added the cultures taken for the release of cases and carriers as well as whole wards and nurses and attendants in the hospital.

The cultures for the detection of meningococcus carriers were taken with the results given in Table 2.

Our culture work must be further divided, for we took cultures of organizations: (a) when a case

occurred; (b) in the routine, and (c) when a large percentage of carriers was found in an organization. (The carriers were always removed from the organization and taken to a carrier camp. The workings of this will be shown farther on.)

The analysis of results is shown in Tables 3 and 4.

TABLE 3.—NUMBER OF CULTURES AND NUMBER AND PER-  
CENTAGE OF CARRIERS FOUND WHEN MENINGITIS HAD  
OCCURRED IN AN ORGANIZATION AND ON ROUTINE  
CULTURE OF THE WHOLE CAMP

Month	After a Case—			Routine—		
	Cultures Taken	Carriers— No. Per Cent.		Cultures Taken	Carriers— No. Per Cent.	
October, 1917.....	4,712	136 2.8		..	108	..
November.....	6,817	258 3.77		2,358	108	4.58
December.....	2,591	114 4.76		25,711	798	3.10
January, 1918.....	2,140	91 4.25		38,700	1,268	3.27

TABLE 4.—NUMBER OF CULTURES AND NUMBER AND PER-  
CENTAGE OF CARRIERS FOUND ON ORIGINAL AND  
RE CULTURE IN ORGANIZATIONS IN WHICH A  
LARGE NUMBER OF CARRIERS  
WERE FOUND

Month	Cultures Taken		Carriers—		Per Cent.
	Orig- inal	Recul- ture	Orig- inal	Recul- ture	
October, 1917.....	4,712	..	136	..	2.8
November.....	2,192	2,138	123	52	5.61
December.....	1,974	1,928	196	76	9.93
January, 1918.....	137	129	10	5	7.29

These tables, based on a large number of cultures, show that generally the percentage of carriers was higher after a case than in the routine cultures. The first complete routine cultures disclosed 3.43 per cent. of carriers, while the second routine cultures on the same troops disclosed 3.02 per cent. A most interesting observation was obtained on reculture when a large percentage of carriers was found in the first culture. While in general less than one-half as many carriers were found, it many times happened that no carriers were found on the second culture. It must, of course, be remembered that the carriers were always removed from the organization. The effects on the epidemic of taking out carriers may be interpreted differently by different observers. For this reason the clinical

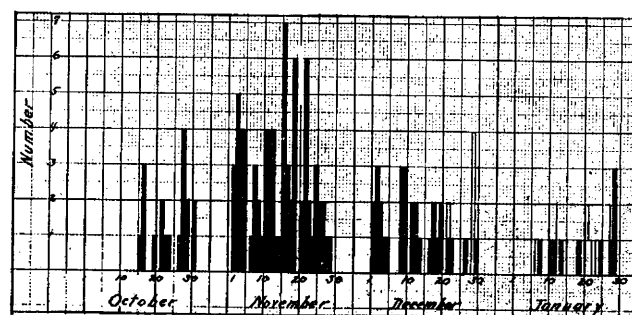


Fig. 3.—Number of cases and dates of occurrence of positive spinal fluid meningitis at Camp Funston, from Oct. 16, 1917, to Feb. 1, 1918: unshaded portion, cases occurring in newly recruited organizations (92d Division); shaded portion, cases occurring in original organizations (89th Division).

cases as they occurred are shown according to date of occurrence and according to the organization in which they occurred. Clinical cases arranged according to date of occurrence are shown in Figure 3 and Table 5, and according to organization in Table 6.

Of the 152 cases, four, or 2.6 per cent., occurred in officers, and no cases occurred among medical officers, nurses, or enlisted men in the wards. Four cases occurred in civilians, two being admitted to the hos-

pital in the first day of October, or about two weeks before any soldiers had the disease. There was no traceable connection between these first two civilian cases and the first cases occurring in soldiers.

From October 16 to February 3 is 171 days, and there occurred 150 cases of clinical epidemic meningitis. If this interval is divided into three parts of thirty-seven days each, we find that there occurred seventy-five cases in the first, forty-seven cases in the second, and thirty cases in the third thirty-seven days. But in the third thirty-seven day period only fourteen cases occurred in organizations that had been in camp the first and second thirty-seven day periods. The thirty-seven day periods correspond in the epidemic approximately as follows:

First thirty-seven days, before routine cultures were taken.

Second thirty-seven days, time of first complete routine cultures in the camp.

Third thirty-seven days, time of second complete routine cultures in the camp.

It may be objected that epidemics die out naturally; but in the third period, when new troops came in they came in gradually, so that cultures could be made, and there occurred only fifteen cases in approximately 15,000 troops, as compared to ninety cases in the 89th Division of about 40,000 troops from October 16 to December 1. But even if the spread and severity of the epidemic would usually have decreased, still by

TABLE 7.—PROTECTION AFFORDED BY CULTURES

	Cases of Spinal Fluid Meningitis—			Per Cent. of Protection
	Total	In Carriers	Organizations Exposed	
1st 37 days	75	11	64 times	18.7
2d 37 days	47	2	45 times	4.44
3d 37 days	14	3	11 times	27.27

taking cultures of whole companies and the routine culture taking of the camp, another very important result was obtained. Sixteen of our carriers developed clinical meningitis, and five more developed sufficient clinical signs and symptoms to have lumbar puncture performed. Nine cases of meningitis with purulent spinal fluid developed in soldiers from whom cultures were taken and who were not detected as carriers; but in at least sixteen cases, by means of our cultures we decreased the probable spread of the disease. According to the thirty-seven day periods this may be presented as in Table 7.

This percentage of protection is a considerable one and indicates that taking cultures after cases and the routine taking of cultures of troops from endemic areas are of value. The development of meningitis in carriers will be discussed later.

Apparent benefits of taking cultures after cases and the routine taking of cultures of troops from endemic areas are demonstrated by these facts: (a) Sixteen of the thirty cases occurring from December 24 to February 3 were in newly recruited organizations. (b) Repeated cases of meningitis in an organization occurred almost entirely before December 1 (directly after the beginning of routine culture taking).

#### EPIDEMIC MENINGITIS AT THE PORT OF EMBARKATION, HOBOKEN, N. J.

Most of the troops sent to France, England and Italy were sent through this port. There are two embarkation camps, of which the one at Camp Mills has been under canvas, while at Camp Merritt bar-

racks have been used. In addition to the camps, the Medical Department under Col. James M. Kennedy controls embarkation and debarkation hospitals (nine in number), two post hospitals, General Hospital No. 1 and such part of the Rockefeller Institute Hospital as is used for soldiers. The first case of meningitis occurred in September at Camp Mills. The occurrence in embarkation cases is given in Table 8.

TABLE 8.—CLINICAL CASES OF EPIDEMIC MENINGITIS AT THE PORT OF EMBARKATION, ARRANGED ACCORDING TO MONTHS

	Total	Camp Mills	Camp Merritt	Embarkation Hospitals
October, 1917.....	1	0	0	1
November.....	2	0	0	2
December.....	3	1	1	1
January, 1918.....	3	0	1	1
February.....	2	0	1	1
March.....	7	0	1	6
April.....	4	1	3	0
May.....	9	5	2	2
June.....	15	7	4	4
July.....	18	13	2	3
August.....	6	2	3	1
September.....	3	2	1	0
October.....	5	1	2	2
November.....	5	0	4	1
	83	32	26	25

The entire value of taking out the detected carriers cannot be determined, for until about May, 1918, the records of communicable diseases on the voyage overseas were not kept at this office, and the Surgeon-General's Office has no records available on this point. Since June, the ship's surgeon has provided the surgeon at the port of embarkation with a copy of the report delivered to the debarkation officers overseas, and from these reports we learn that two cases of meningitis occurred in organizations from which patients were taken out; but the men were gone from the camps before cultures could be taken. A number of cases occurred on the voyage in organizations from which no patients were taken during the stay in embarkation camps, but none occurred in organizations from which the carriers were removed. Three cases of meningitis occurred in carriers taken out on positive culture.

TABLE 9.—NUMBER OF CULTURES AND NUMBER AND PERCENTAGE OF CARRIERS FOUND WHEN MENINGITIS HAD OCCURRED IN THE PORT OF EMBARKATION

	Cultures Taken	Carriers Found—	
		Number	Per Cent.
October, 1917.....	30	4	
November.....	0	0	
December.....	0	0	
January, 1918.....	333	16	4.8
February.....	230	13	5.6
March.....	156	20	12.8
April.....	634	17	2.6
May.....	701	11	1.5
June.....	1,623	38	2.3
July.....	1,191	27	2.2
August.....	463	7	1.5
September.....	5	0	0.0
October.....	208	1	0.4
November.....	459	4	0.9
Total.....	6,033	158	2.6

#### CARRIERS

Detection of carriers is of scientific and practical interest only so far as it effects the spread of the disease, as carriers develop the disease, and the manner of clearing up carriers. It is necessary to consider these points, especially as so many officers, without knowing a thing about it, have tried to discredit the value of detecting and removing carriers. That carriers spread the disease can hardly be doubted.

This is true from analogy to other diseases, such as diphtheria, and because taking out carriers certainly seems to have prevented the spread of the disease.

Detectable carriers must be divided into three groups: (a) those who are in the incubation period of the disease; (b) casual carriers, and (c) chronic carriers.

As was stated earlier, by our methods we probably detected the more heavily infected carriers. On those we detected we have considerable epidemiologic data to substantiate the assertion that our carriers were able to spread the disease; and that taking the carriers from the organization stopped the disease in that organization is evidenced by the following concrete cases, of which more could readily be added:

(a) In Company C, 356th Infantry, two cases occurred, Nov. 19, 1917, and two, Nov. 22, 1917. Cultures were taken, November 22, 25 and 29. The number of men from whom cultures were taken was 152, 157 and 129, and the number of carriers detected was 7, 9 and none, respectively.

(b) In Company A, 340th Field Artillery, 200 routine cultures were taken, November 22, and eleven carriers were detected. November 25, 150 recultures were taken and five carriers detected. November 30, 176 recultures were taken and no carriers detected. November 28 and December 3, two carriers taken out developed spinal fluid meningitis.

(c) In Company E, 353d Infantry, November 21 there occurred a case. November 22, 163 cultures were taken and no carriers found, and no further cases occurred in the company.

The development of the disease in carriers has been much discussed. Gates,<sup>1</sup> in his article on antimeningitis vaccinations, says, in his conclusions: "Moreover, agglutinins have been demonstrated in the blood serum of chronic carriers of the meningococcus. Evidence is thus brought forward that the relative immunity of chronic carriers to epidemic meningitis may be due to the presence of specific antibodies in the blood stream." This evidence we do not refute; but some of our detected carriers did have clinical meningitis with purulent spinal fluid. We made many examinations and many kinds of examinations, and from these believe that we have noted different varieties or degrees of epidemic meningitis:

1. Infection in the nasopharynx only or principally. These are our ordinary carriers.

2. Those having the clinical symptoms and signs of meningitis, but clear spinal fluid free from bacteria and polymorphonuclear cells. Five of our detected carriers had sufficient symptoms and signs to have lumbar puncture performed on them, and a very large percentage of our carriers early in the epidemic had severe headache, nausea and fever but cleared up shortly and lumbar puncture was not performed on them.

3. Those having clinical symptoms, clear spinal fluid and no increase in cells, but meningococci in the fluid. Of such cases we observed two: S., Company F, 354th Infantry, and R., Company C, 356th Infantry. That injection of the killed vaccine should, in at least a number of instances, produce severe headache, vomiting (projectile at times) and opisthotonos shows that the endotoxin has a predilection for the meninges, and it is possible that the toxin selects the meninges, injures them, and makes the field fertile for the meningococci already in the blood stream and gives rise to the frank epidemic meningitis with purulent spinal fluid.

4. Those having frank epidemic meningitis. Nineteen of our detected carriers developed the frank type of epidemic meningitis, one developed meningeal symptoms with meningococci in the fluid but no pus cells, and four others developed sufficient symptoms to have lumbar puncture performed. Nine cases of purulent spinal fluid meningitis developed in soldiers missed as carriers on cultures taken less than thirty days and more than seven days before the onset of the disease, and two soldiers missed as carriers in this way developed sufficient symptoms to have lumbar puncture performed. These data when further analyzed may be presented as in Table 10.

TABLE 10.—DEVELOPMENT OF EPIDEMIC MENINGITIS IN SOLDIERS WHOSE CULTURES HAD BEEN TAKEN

	Detected as Carriers		Missed as Carriers	
	Developed Meningitis with Purulent Spinal Fluid	Developed Clinical Meningitis, but No Purulent Spinal Fluid	Developed Meningitis with Purulent Spinal Fluid	Developed Clinical Meningitis, but No Purulent Spinal Fluid
Within 7 days or less after cultures were taken.....	19	5	4	2
More than 7 days after cultures were taken..	0	0	5	0
Total.....	19	5	9	2

The incubation period lies between six and twelve days after exposure, so that it is evident that our detected carriers who developed meningitis (purulent spinal fluid and with sufficient clinical symptoms to have lumbar puncture done) were probably not carriers but early cases; and we have no evidence to prove that what are generally known as carriers (especially chronic) develop meningitis. Probably the five carriers who had sufficient clinical symptoms of meningitis had enough toxin to affect the meninges. At Camp Funston about 0.2 per cent. of the troops developed meningitis, while 0.48 per cent. of the detected carriers had the frank form of the disease, or the disease was more than twice as frequent in detected carriers as in all the troops coming from an area in which the disease is endemic. The disposition of carriers is an important factor. At first we sent detected carriers to the hospital; but later they were taken to detention camps where military drill could be continued, that is, the soldiers remained in training. At these detention camps at least once a day silver nitrate solution or argyrol was thoroughly applied to the tonsils and the nasopharynx. However, even without special treatment, about 90 per cent. cleared up very soon after they were taken from the active focus of infection; that is, at least 90 per cent. are only casual carriers. By February 1, out of 3,290 carriers at Camp Funston, only seventy-two, or 2.19 per cent., were found to be chronic carriers. These were all later cleared up by the laryngologists. At the port of embarkation about the same percentage of chronic carriers was hard to clear up as at Camp Funston.

#### SUMMARY AND CONCLUSIONS

1. Detection of carriers is possible and practical and has marked advantages over quarantining of contacts as has been generally practiced. Cultures of large numbers of troops can be taken with a high percentage of efficiency, and to get the best results it is necessary to take cultures of whole organizations rather than only contacts. The taking of cultures of large numbers of persons at one time has been necessary, and

1. Gates, F. L.: *J. Exper. Med.* 28: 449 (Oct.) 1918.

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 than 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.

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## 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,<sup>1</sup> 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 non-distensible 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<sup>2</sup> 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 bronchitis, 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.