A Study of a Type I Pneumococcus Epidemic at the State Hospital at Worcester, Mass.*

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AN epidemic of 110 cases of lobar pneumonia, which occurred at the State Hospital in Worcester, Mass., during the early months of 1937, offered an unusual opportunity to study the epidemiology of Type I pneumococcus infections.

We shall first present, in brief, the organization of the hospital. We shall then describe the epidemic as it swept through the institution, and indicate the various steps that were taken to check its spread.

DESCRIPTION OF THE HOSPITAL

The State Hospital for mental diseases at Worcester, Mass., has a fairly constant adult population of about 2,400 patients and 600 employees. It is well organized and expertly administered. It consists of two parts: the Main Hospital with some 1,800 patients and 500 employees, and the Summer Street Division with 600 patients and 100 employees. Summer Street Division is essentially an overflow hospital, to which patients are transferred when approaching their period for discharge. It is 3 miles from the Main Hospital, and has its own separate staff, which has little contact with the Main Hospital staff. It receives no new patents, but admits only those patients that are transferred from the Main Hospital.

The institution has approximately equal numbers of male and female patients. The male and female patients are segregated in separate wards, and are classified in accordance with their mental condition. Each side of the Main Hospital has an infirmary, one for males, one for females, for the treatment of acutely ill patients. All patients who develop pneumonia or other acute illness on the general wards, and also in Summer Street Division, are transferred immediately to the infirmary for special medical or surgical treatment. All responsibility for the medical and surgical care of the acutely ill patients is centered in one staff physician.†

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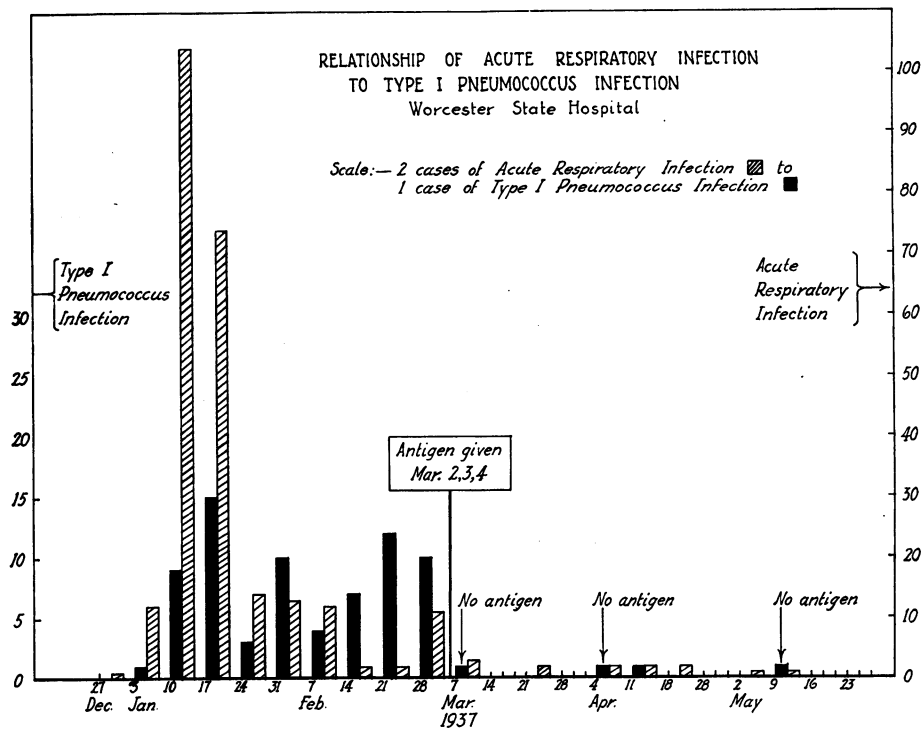
† We wish to express our appreciation of the fine coöperation and aid that was given to us by all members of the hospital staff, and particularly by Dr. F. H. Sleeper, Assistant Superintendent of the Hospital, and by Dr. W. E. Glass, staff physician in charge of acutely ill patients.

CHART I

Chart I shows the relationship of the epidemic of Type I lobar pneumonia at the Worcester State Hospital to the epidemic of influenza. Type I pneumococcus infection prevailed from the very first of January, and increased as influenza increased. The influenza outbreak was over by the middle of February, but Type I pneumonia did not decline until Type I pneumococcus antigen was given on March 2.



Bronchopneumonia has been quite prevalent among the patients of the hospital, as is always the case in institutions for mental diseases. Lobar pneumonia, however, has shown only a slightly higher incidence among the patients and staff of the hospital than in the population of the state at large.

The average annual incidence of pneumonia in the hospital during the past 4 years, including terminal bronchopneumonia, has been as follows: bronchopneumonia, 70.5 cases, 48 deaths; lobar pneumonia, 18.0 cases, 8 deaths.

Pneumonia of both types has been more prevalent in the male than in the female patients of the hospital. The sex ratio of pneumonia incidence in these patients has been about the same as that of the general population.

	Males	*Females*
Proportion of Cases of Bronchopneumonia in the Institution	55%	45%
Proportion of Cases of Lobar Pneumonia in the Institution	64%	36%

As a general rule, bronchopneumonia has prevailed throughout the year, but lobar pneumonia has been much more prevalent during the late winter and early spring months than in the summer and fall.

THE INFLUENZA EPIDEMIC

Beginning early in January, 1937, an epidemic of moderately severe upper respiratory infection invaded the Main Hospital. This outbreak was similar in all respects to an epidemic of acute respiratory infection that prevailed at the same time along the whole North Atlantic Coast. Though many of the cases of this illness, both in and outside the hospital, were relatively mild, nevertheless it seems quite probable that the outbreak was true epidemic influenza.* The course of this influenza outbreak in the Main Hospital is indicated on Chart I.

Summer Street Division, although only 3 miles from the Main Hospital, escaped the influenza outbreak.

The hospital routine was completely upset by the epidemic. It was necessary to set aside certain special isolation wards for care of the acutely sick. The disease spread with rapidity throughout the institution. The peak was reached in the week of January 10, during which 96 cases occurred.

The hospital physicians noted that an unusual number of cases of pneumonia accompanied the influenza outbreak, but at first this fact caused no special concern.

The epidemic of influenza in the Main Hospital followed a characteristic incidence curve, and at the end of 6 weeks, conditions in the hospital had returned almost to normal. However, it soon became apparent that pneumonia was continuing long after the influenza epidemic had terminated. Instead of a concomitant decline in pneumonia with the decline in the incidence of influenza, as had been anticipated, lobar pneumonia continued unabated (see Chart I).

The hospital authorities noted that most of these cases of pneumonia were due to the Type I pneumococcus. They noted also that the onset of the attacks of pneumonia in those cases that occurred in the latter part of February was sudden, without a history of recent acute upper respiratory infection.

THE PNEUMONIA EPIDEMIC

On February 25, we were invited, in our capacity as State Consulting Epidemiologist, to aid in an investigation of the pneumonia situation at the Main Hospital.

A rapid survey revealed the following facts:

1. The epidemic of acute upper respiratory infection (influenza) at the Main Hospital, which began the week of January 3, had terminated, with no invasion of Summer Street Division.
2. Cases of lobar pneumonia were developing at a rapid rate in the Main Hospital. The infirmary was full of them, and new cases were coming in every day.
3. The cases of pneumonia were coming from many different parts of the hospital. The outbreak was not limited to one ward, to one occupational section such as farm hands and outdoor workers, or any single type of patient. Physicians, nurses, and attendants were affected as well as patients.
4. Practically every case of lobar pneumonia was due to the Type I pneumococcus.
5. The pneumonia outbreak was limited to the Main Hospital, and had not invaded the Summer Street Division.
6. There was no unusual prevalence of Type I pneumococcus pneumonia in the city of Worcester, nor in the State of Massachusetts as a whole.

In view of our previous experience with the Type II pneumonia epidemic at Bedford Hospital,[1] we felt quite certain that these findings indicated very strongly that the Type I pneumococcus had become widely distributed and had established itself in all parts of the institution. The source of the infection of the patients was not a single heavily infected individual

* The virus of epidemic influenza was obtained many times in our own and other laboratories by ferret inoculation of material obtained from victims of this epidemic.

(case or carrier) who transmitted the disease through a common vehicle such as food. Carriers must have become highly prevalent throughout the Main Hospital, and these disseminated Type I pneumococci among their fellow patients and the attendants.

A random sample of Type I carrier prevalence in the Main Hospital was made at once. We employed the same technic in detection of carriers that we have used in previous studies.[2] Our surmise was verified almost immediately. The survey indicated that at least 10 per cent of the patients of the Main Hospital harbored the Type I pneumococcus in the nasopharynx. This is a great contrast with a normal population, where Type I pneumococcus is found in the nasopharynx only once[3] or twice in 500 cultures.

The hospital authorities were faced with a serious emergency. They were responsible for a large population of presumably highly susceptible persons who were continuously exposed to a virulent pneumococcus; for it was certain that Type I pneumococcus was widely dispersed throughout the hospital. The month of highest pneumonia prevalence in New England was at hand. New cases of Type I lobar pneumonia were developing every day. The rapid detection and isolation of all the Type I pneumococcus carriers in the Main Hospital was an impossible task, for there were 200 of them at least.

In the face of this situation we determined to make an attempt to check further spread of the epidemic of pneumonia by a rapid immunization of the hospital population.

The antigen * used was a preparation of an antigenically active polysaccharide of the pneumococcus, a compound similar to that described originally by Schiemann and Casper[4] in 1927 and more recently by Avery and Goebel[5] under the name of acetyl polysaccharide. However, the sample had properties more nearly like the polysaccharide studied by Felton[6] in that it was practically devoid of acetyl groups and yet was fully antigenic. The analysis of this antigen, made by Felton, was as follows:

Nitrogen—per cent	3.16%
Glucose number	25.1 %
Acetyl	1.0 %
Precipitin titer	1:2,500,000
Type I immunity in mice	1:50,000,000

In addition, Felton[7] had tested the antigen on human beings and found that a single injection of 2 mg. stimulated antibody production effectively. The obvious advantages of an antigen of this kind are: it is soluble, stable, readily rendered sterile, and easily standardized to insure a satisfactory antigenic dose. We selected the Felton antigen for the above reasons, and also because of the fact that the antigen was immediately available.

The antigen was given subcutaneously in a single dose of 0.5 c.c. (2 mg.) to each adult. Over 1900 patients and attendants in the Main Hospital were injected on March 2, 3, and 4. No ill effects were produced.

The epidemic of pneumonia in the Main Hospital stopped almost immediately following the administration of the antigen. We cannot affirm, of course, that the antigen stopped the outbreak. It is possible that the epidemic might have stopped spontaneously on March 4. The results would have been much more clear-cut if it had been feasible for us to immunize every alternate person, but this was not possible. The hospital authorities felt that if we had any reason to believe that the antigen would give some protection, then we were unjustified in withholding this possible protection from a single patient. A small num-

* This antigen was kindly furnished to us by Dr. Malcolm of the Lederle Laboratories.

ber—about 200—of the patients and a few attendants refused the antigen, and this group served, in some degree, as a control series.

The course of the epidemic is shown graphically in Chart 2. The striking feature is that the pneumonia ceased abruptly following the administration of the antigen. Eighty-four cases of lobar pneumonia had occurred in the Main Hospital between January 3 and March 3. Nine cases of lobar pneumonia occurred after the antigen was given (from March 4 to June 1), only 4 of which were due to the Type I pneumococcus. Three of these had not received the antigen. One patient, I. L., female, 78 years old, who had been given the antigen on March 31 developed lobar pneumonia on April 18 and died in 4 days. This case represents a definite failure of the antigen to protect against Type I pneumococcus invasion. Four of the other patients who developed lobar pneumonia (March 4 to June 1) had been given the antigen, but their pneumonias were due to some other organisms than the Type I pneumococcus.

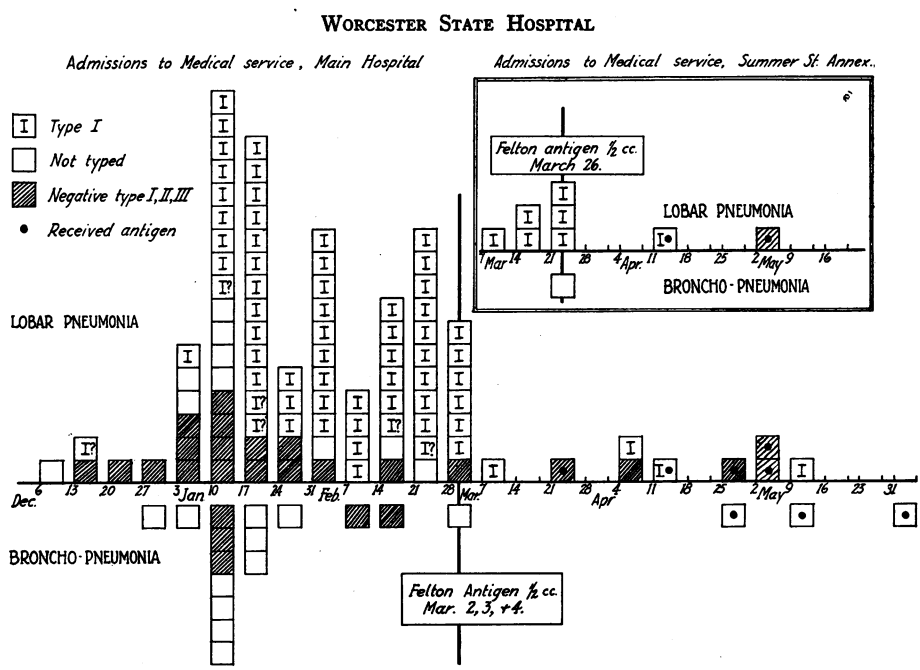
Nine individuals were discovered who had a respiratory tract infection in which Type I pneumococcus was the prevailing organism, yet they never developed typical lobar pneumonia.

CHART 2

Graph of the Course of the Epidemic of Type I Lobar Pneumonia at Worcester State Hospital.

Each square represents a case of pneumonia. Bronchopneumonia cases are shown below the base line—lobar pneumonia above. Each case of proven Type I pneumonia is indicated by a [I] in the square. If the individual had received antigen, it is indicated by a dot within the square.

The small diagram inserted in the upper right hand corner represents the Summer Street Division outbreak which occurred after the Main Hospital epidemic had stopped.



Three of this group were diagnosed clinically as cases of influenza, and 5 as common colds. One had an otitis media, followed by a fatal attack of Type I pneumococcus meningitis.

Case P. D.—Only one in this latter group (P. D.) had been given the antigen on March 3. He came to the infirmary on the evening of March 6 with a temperature of 104° F., pulse 120, and respirations 20 per minute. His leucocyte count was 16,000 per c.mm. and he gave every evidence of a beginning lobar pneumonia. The X-ray report next morning read "questionable early lobar pneumonia." His temperature fell by crisis on March 8. He was quite well on March 9. His sputum on March 7 was positive for Type I pneumococcus. He may be said to have run a complete course of pneumonia in 4 days.

It seems possible that the antigen which was administered 3 days previous to the onset of his illness may have given this patient some degree of protection against Type I pneumococcus invasion.

THE SUMMER STREET DIVISION PNEUMONIA OUTBREAK

The Summer Street Division, 3 miles from the Main Hospital, escaped the epidemic of influenza in January. At the time we began our investigation on February 28, no cases of Type I lobar pneumonia had occurred there.

The first case of Type I lobar pneumonia developed at Summer Street on March 9, nearly a week after the Main Hospital epidemic had ceased. This first patient was a newly appointed attendant, who had had no contacts with the patients of the Main Hospital. In view of these facts we should have been on the alert, and should have made an immediate investigation of Summer Street to determine the prevalence of Type I pneumococcus carriers there, and also to find the source of infection of this first case, but we did not do so.

Beginning on March 19, 5 cases of Type I lobar pneumonia developed in quick succession among the male patients in the Summer Street Division. A fresh supply of antigen was obtained and all the patients and attendants were given a single subcutaneous injection of this antigen on March 26.

The outbreak of pneumonia at Summer Street Division ceased, following the administration of the antigen, just as it had done at the Main Hospital. The course of this outbreak is shown in the insert of Chart 2.

Cases—It will be noted that 2 cases of lobar pneumonia occurred in Summer Street, after the antigen was given. One of these represents a failure of the antigen to protect against Type I pneumococcus invasion. This individual, M. C., 63 years old, was given the antigen on March 26, developed Type I lobar pneumonia on April 17, and died in 4 days. The second pneumonia victim also had the antigen on March 26, but his pneumonia, which developed on May 2, was due to Type IV pneumococcus.

How can one explain the invasion of Summer Street Division by Type I pneumococcus after the epidemic in the Main Hospital had ceased? It seems most probable that the infection was introduced by carriers that were transferred to Summer Street from the infected zone.

Thirty persons had been transferred to Summer Street Division from the Main Hospital between January 1 and April 1. In Table I we have given the dates of transfer of these individuals.

Cultural studies of the nasopharynx of each of the entire group of transfer patients were made on March 29. Two, a woman transferred on March 1 and a man transferred on March 29, were found positive for Type I pneumococcus. The woman could not have been the direct source of the infection of the Summer Street cases of lobar pneumonia, because all the pneumonia cases had come from the male wards. The man was not responsible, because the infection had been introduced be-

TABLE I

Table of Transfer of Patients from the Main Hospital to the Summer Street Division from January 1 to April 1, 1937

Date	*Number of Men*	*Number of Women*	*Remarks*
January—1st week	4	1	No further transfers in January because of influenza epidemic at Main Hospital.
February 1	3	1	
" 17	3	0	
" 19	4	0	One man, J. F., was convalescent from lobar pneumonia, which he had developed Jan. 14. Type I pneumococcus was never found in his sputum.
" 24	0	1	
March 1	0	2	One woman, L. W., was found to be Type I carrier on March 29.
" 3	1	0	
" 5	0	1	
" 8	2	0	
1st case of Type I pneumonia developed.			
March 25	4	0	
" 29	3	0	One man, M. D., was found to be a Type I carrier on March 29.

fore he arrived, probably between February 15 and March 1. (See Chart 2). It seems quite possible that patient J. F. (see Table I), who developed lobar pneumonia in the Main Hospital on January 14, and who was transferred to Summer Street on February 19, was the source of infection. We know that the new cases originated in the ward to which he was sent, and the outbreak began shortly after his arrival. The chain of evidence is not complete, however, since the throat culture of J. F. was negative on March 29, the date that the Summer Street carrier studies were first made.

We can be fairly certain that Type I pneumococcus was transferred from the Main Hospital to the male wards of Summer Street Division late in February. The infection spread through the male wards, but not the female wards. Subsequent events showed that both carriers and cases of Type I pneumococcus infection were produced following the introduction of the specific organism.

STUDIES OF TYPE I PNEUMOCOCCUS CARRIERS

When we began our study on February 27, the epidemiological evidence suggested that Type I pneumococcus carriers were widely distributed throughout the Main Hospital. Our first step was to take a random sample of cultures from the nasopharynges of patients and attendants from the various wards. We found almost at once that our surmise was correct.

The search for carriers was continued after the antigen had been given, and after the epidemic of pneumonia had ceased. A total of 280 different persons were tested in order to secure an estimate of the prevalence of Type I pneumococcus carriers in the hospital population. Twenty-two normal individuals were discovered to be harboring virulent Type I pneumococci—a carrier rate of 7.8 per cent—or 35 times the normal carrier rate of the general population. Sixteen of these normal carriers were found in the Main Hospital and 6 in Summer Street, most of them during the first month of the

study. The duration of the carrier state in each person is shown in Chart 3. It will be noted that most of the carriers cleared up spontaneously within a short time. By June 7 all but 5 had become negative.

Case—Only one discovered carrier later developed lobar pneumonia. L. R.—41 years old, male, Summer Street Division—was found to be a nasopharyngeal carrier on March 24. He was given 0.5 c.c. antigen on March 26. Some 650 other patients and attendants at Summer Street were inoculated on the same day. Twenty-four hours later he suddenly developed Type I lobar pneumonia.

It is possible that the antigen may have precipitated the attack of lobar pneumonia in this individual. However, 4 other patients at Summer Street were also known to be carriers, and were injected with antigen at the same time as L. R., but without ill effect.

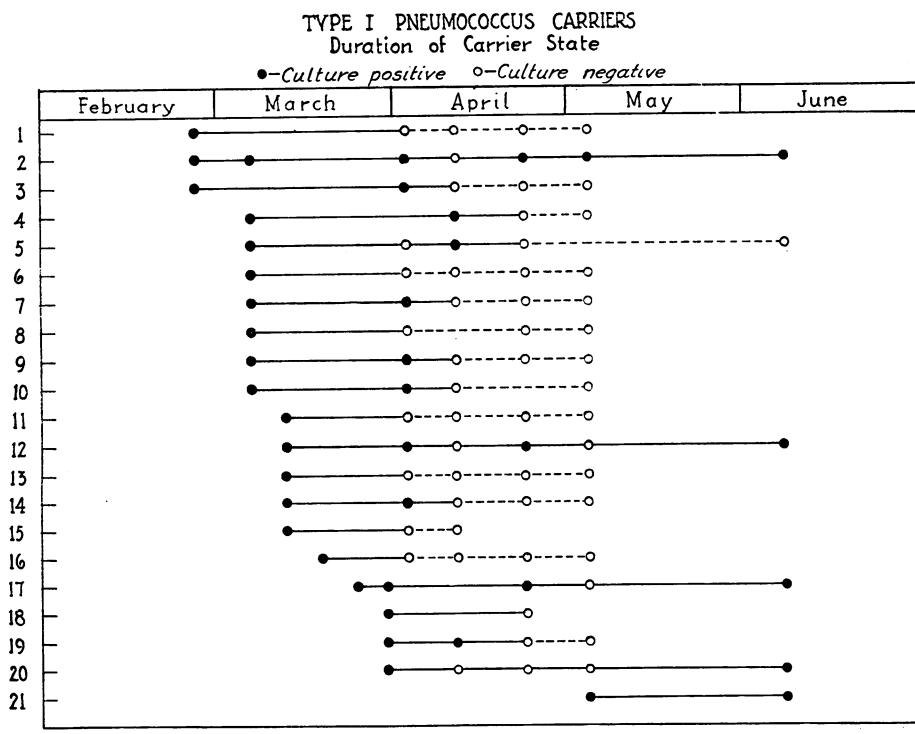
DISCUSSION

The epidemic of lobar pneumonia at the Worcester State Hospital was very extensive and serious in its effects. Between December and May, 110 cases of lobar pneumonia occurred, most of them in January and February.

The fatality rate of the epidemic was high, despite extensive use of Type I anti-pneumococcus serum. Many of the patients were debilitated and noncoöperative. The group in which no pneumococcus type was determined had a particularly high death rate (see Table II). These persons were extremely noncoöperative, and no sputum could be obtained from them. Often the type of infection was not determined

CHART III

The graph illustrates the duration of the carrier state in the 21 Type I pneumonia carriers that were discovered at Worcester State Hospital.



because of the rapid progress and fatal outcome of the disease. It is probable that most of them were infected with Type I pneumococcus.

Sixty-seven pneumonia patients had a positive Type I pneumococcus sputum. It seems probable that most of the remaining 43 cases of the series of lobar pneumonia were also caused by Type I pneumococcus, but this point is difficult of proof. We made an attempt to do so.

TABLE II

Mortality Rates from Lobar Pneumonia in the Epidemic at the State Hospital in Worcester, Mass., 1937

Lobar Pneumonia	*Cases*	*Deaths*	*Mortality Rate*
Type I	73	16	22%
Not Type I, II or III	25	7	28%
Not typed	12	6	50%
Total	110	29	26%

When we began our study, many of the untyped patients were dead. We secured a list of all the convalescent pneumonia patients in whom Type I pneumococcus had not been found, and obtained blood from each of them. They were tested for the presence of Type I specific immune bodies, using standard methods.[8] The bloods of 17 out of a group of 21 that were tested showed a protective power of 100 m.l.d. in standard mouse protection tests. The blood of 6 of these persons agglutinated the Type I pneumococcus in dilution of at least 1–5, and showed a mouse protection test of at least 1,000 m.l.d. We have felt justified in including these 6 persons in the list of those who undoubtedly had Type I pneumococcus pneumonia. They are indicated in Chart 2 as [I ?]. We feel quite sure that all 17 convalescents that showed a blood protection power of 100 m.l.d., and many more of the group of lobar pneumonia patients as well, had also been infected with the Type I pneumococcus, but we have no conclusive proof.

In addition to the actual cases of Type I lobar pneumonia, there were discovered 9 persons who were infected with Type I pneumococcus who never developed typical lobar pneumonia. We can make a fair estimate, therefore, that the total toll of the epidemic was at least 80 to 100 cases of Type I lobar pneumonia, together with at least 9 Type I pneumococcus infections that were not lobar pneumonia.

A cross-section of carrier prevalence indicated that there were at least 200 carriers of Type I pneumococcus in the institution during the epidemic; probably there were many more. As the pneumonia season ended, these carriers cleared up so that it is quite probable that by June 1, not more than 50 carriers remained. Most of these carriers would become negative before the summer was over.

The antigen was given in each hospital division at a time of high pneumonia prevalence. Practically all the carriers were given antigen, but there is no evidence that this procedure affected their carrier state. There is strongly suggestive evidence, however, that the administration of antigen at the Main Hospital checked the further spread of Type I lobar pneumonia in that institution. The disease then spread to Summer Street Division, after it had receded in the Main Hospital. The sudden abatement of lobar pneumonia at the Summer Street Division, following administration of the antigen was striking. The picture here was quite different from that at the Main Hospital. It might be claimed that the pneumonia epidemic at the Main Hospital had run its course when we began our study, and would have stopped on March 4 without any interference on our part.

The same argument cannot be em-

ployed at Summer Street. Conditions were ideal there for the spread of the infection. There were 600 patients and 100 employees. Presumably these persons were as susceptible to pneumonia as had been the group at the Main Hospital. The virulent infection had been introduced recently from the Main Hospital, and carriers became widely established. The cases began to develop rapidly in March, and stopped abruptly following universal administration of antigen.

If a single injection of 0.5 c.c. Type I pneumococcus antigen will protect susceptible individuals from an attack of Type I lobar pneumonia in the face of a heavy exposure and under conditions that are highly favorable for the spread of this infection, then this is a matter of far-reaching significance and worthy of continued study.

CONCLUSIONS

1. An extensive epidemic of lobar pneumonia in 1937 at the State Hospital in Worcester, Mass., which began during an influenza outbreak, was due to the Type I pneumococcus.

2. Type I pneumococcus carriers became widely distributed throughout the Main Hospital. These carriers, and not the actual cases of pneumonia, were responsible for the spread of the infection.

3. The epidemic at the Main Hospital ceased abruptly following administration of Type I soluble antigenic substance (Felton antigen) to the hospital population.

4. A fresh outbreak of Type I lobar pneumonia occurred at Summer Street Division (a branch hospital) 1 week after the epidemic at the Main Hospital abated. This infection entered Summer Street Division through normal carriers of Type I pneumococcus, who were transferred from the Main Hospital to the subsidiary hospital. The administration of antigen at Summer Street was also followed by immediate cessation of this outbreak.

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* Also see previous and subsequent papers in the *Journal of Immunology* and elsewhere upon this subject by Dr. Felton.

Contact

MY last word is this. Every person who contacts the public in any health organization, no matter what it may be, has some effect on public relations. If you have somebody at the telephone desk, or somebody in the reception bureau, whose attitude is gruff or sharp, he can do more harm than the head of the organization can undo. When we turn the public away with an uncivil answer, we create bad public relations. There really ought to be a training course, however brief, in every organization employing a large personnel in the public health field, to teach their staff how to meet the public, what to say to them, and how to say it. Those who can't learn the knack should be placed where they do not deal with their clientele.—*The Health Officer*, January, 1938, page 435.