STUDIES ON PNEUMOCOCCAL CROSS INFECTION AND THE RELATIONSHIP OF PNEUMOCOCCI TO ACUTE RESPIRATORY DISEASE IN EARLY LIFE.

By

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PREFACE

This investigation was commenced during my second year as house physician at Knightswood Infectious Diseases Hospital. During the previous year my interest had been aroused in the subject by the investigations on pneumococcal carriage then in progress at the hospital under the direction of Dr. Thomas Anderson, Reader in Infectious Diseases at the University of Glasgow.

Prior to undertaking this investigation a period of several months was spent on training in the bacteriological methods required. Subsequently, several investigations of a preliminary nature were carried out, on cross infection by streptomycin resistant strains of Bacillus coli amongst patients treated by streptomycin and on cross infection by pneumococci amongst children in open wards.

I am deeply indebted to Dr. Thomas Anderson, through whom I was first introduced to the subject and with whose encouragement this particular investigation was undertaken.

Finally I would like to express my thanks to the nursing and domestic staffs of Knightswood Infectious Diseases Hospital whose willing co-operation made the investigations possible; to the laboratory technicians who ensured a constant supply of media; and especially to the senior technician, Mr. A. McLean, who executed the drawings in the text and did the photography.

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INTRODUCTION

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The Purpose of the Investigation.

The occurrence of cross infection amongst children in an open ward has been the subject of investigation by many writers. Such investigations have been carried out in medical and surgical wards, and in wards used for the treatment of infectious diseases. The occurrence of such cross infection, no matter what anatomical system be involved, has been shown to cause considerable lengthening of the period in hospital and to have a deleterious effect on the health of the child, already debilitated by the original illness. Complications occurring in patients suffering from scarlet fever, measles or diphtheria have been proved to be due to cross infection. (Allison and Brown, 1937; Allison, 1938; Cruickshank and Godber, 1939).

Excluding gastro-intestinal pathogens, the organism most frequently studied has been Streptococcus pyogenes, and this has proved to be an ideal organism for such studies since distinct serological types can be identified. It has been shown that the acquisition of a type of Streptococcus pyogenes distinct from that causing the original illness, may cause the development of a complication in a patient suffering from scarlet fever. (Allison and Brown, 1937).

Staphylococcus aureus has more recently been investigated as the cross infecting agent and the development of bacteriophage typing has facilitated the tracing of the

infection to the source. The epidemiology of this organism has been studied in nursery outbreaks of pemphigus neonatorum, (Elliot, Gillespie and Holland, 1940; Allison and Hobbs, 1947), in the investigation of infected wounds in a surgical ward (Rountree, 1947) and in outbreaks of diarrhoea due to the staphylococcus.(Williams, Swift, Vollum and Wilson, 1946; Oddy and Clegg, 1947).

It has also been shown that dust is an important vehicle for the cross infecting organism and that dust-borne, as distinct from droplet or direct contact infection, occurs. Although it is difficult to implicate directly the floor dust of the ward as the vehicle for the organism, the implication may be inferred since methods of dust suppression prevent cross infection in wards. (Wright, Cruickshank and Gunn, 1944; Medical Research Council, 1951). The importance of such dust suppression methods in controlling respiratory infection, at least in a military unit, has been shown by Anderson, Buchanan and MacPortland (1944).

There have been no such studies on cross infection in a ward used for the treatment of infants or young children suffering from an acute respiratory illness, nor has the pneumococcus previously been used as a tracer organism, except in actual outbreaks of lobar pneumonia. The distinct serological types render it a suitable organism for study. It would seem to be of value to study the occurrence of cross infection in this kind of ward with special reference

to the pneumococcus for the following reasons:-

1. To determine if the pneumococcus may be transferred from individual to individual by floor dust. The epidemiology of pneumococcal respiratory infections has been studied by many writers and their findings have been reviewed by Finland (1942a). Although the pneumococcus is probably transferred by droplet infection there is little direct evidence of this and so, the possibility of some other means of infection exists.
2. To determine if there is any difference in the method of transference of the pneumococcus, Streptococcus pyogenes and Staphylococcus aureus and if one could account for any such difference found.
3. To determine if any relapse occurring in a child's respiratory illness was due to the acquisition of an organism not previously present in the respiratory tract.
4. To determine if the acquisition of a new type of pneumococcus did ever cause any clinical upset on the part of the patient.
5. To determine how great was the possibility of an adult, in contact with a patient, infecting that patient with organisms present in the adult's upper respiratory tract and to what extent this constituted a danger to the patient.
6. To determine if the higher types of pneumococci play any part in the etiology of respiratory illness in the infant and young child. The pneumonia which

occurs in infants differs from that which occurs in adults. The latter usually affects an entire lobe and is frequently due to the lower fixed types of pneumococci (types 1, 2 and 3). In contrast, the infant usually suffers from broncho-pneumonia and the types of pneumococci isolated from the nose, throat, empyema pus or autopsy specimen are those higher types found in the general healthy population. (Finland, 1942b; Guthrie and Montgomery, 1948). It may be that the adults and older children infect these infants with the higher types of pneumococci and cause a respiratory infection. On the other hand, the respiratory infection may be the result of some other factor, for example, a virus infection, which damages the mucous membrane and allows secondary invasion by the pneumococci and other organisms already present in the upper respiratory tract. (Dochez, Mills and Kneeland, 1933). If it could be shown that there was no clinical upset when patients acquired pneumococci from other patients or from members of the nursing, medical and domestic staff in contact with them, it would be evidence against the higher types of pneumococci being the primary causal organisms of pneumonia or bronchitis in the infant or young child.

Ideal opportunities were offered at Knightswood Hospital to carry out such investigations. The laboratory facilities were excellent and the writer was able to carry out unaided all the bacteriological work entailed. The

nursing, medical and domestic staff readily volunteered as subjects for the investigation. No specific measures were taken in the wards to prevent cross infection but the general nursing standards were high. No dust suppressive methods were used in the wards and the nursing staff did not wear masks.

It was hoped that by carrying out (a) a frequent swabbing of the noses and throats of all patients admitted to a ward, used solely for the treatment of acute respiratory illness in infants and children under six years of age, (b) a similar swabbing of all nursing, medical and domestic staff in contact with these patients and (c) a frequent examination of the bacterial content of the floor dust, important information might be obtained about the incidence and effects of cross infection by the pneumococcus and the part played by the pneumococcus in the etiology of pneumonia and bronchitis in infants and young children. Since the incidence of cross infection by Streptococcus pyogenes and by Staphylococcus aureus was studied for comparative purposes, the part, if any, which these two organisms play in the etiology of pneumonia or bronchitis in the infant and young child might likewise be determined.

Finally, it seemed to the writer that it would be of considerable value to conduct similar investigations in other wards and compare the results obtained with those from the main investigation. Wards used for the treatment

of measles were chosen since, in this disease, the occurrence of bronchitis or pneumonia as a complication is frequent. In one of these wards, the patients were purposely overcrowded to observe the effects of overcrowding on cross infection.

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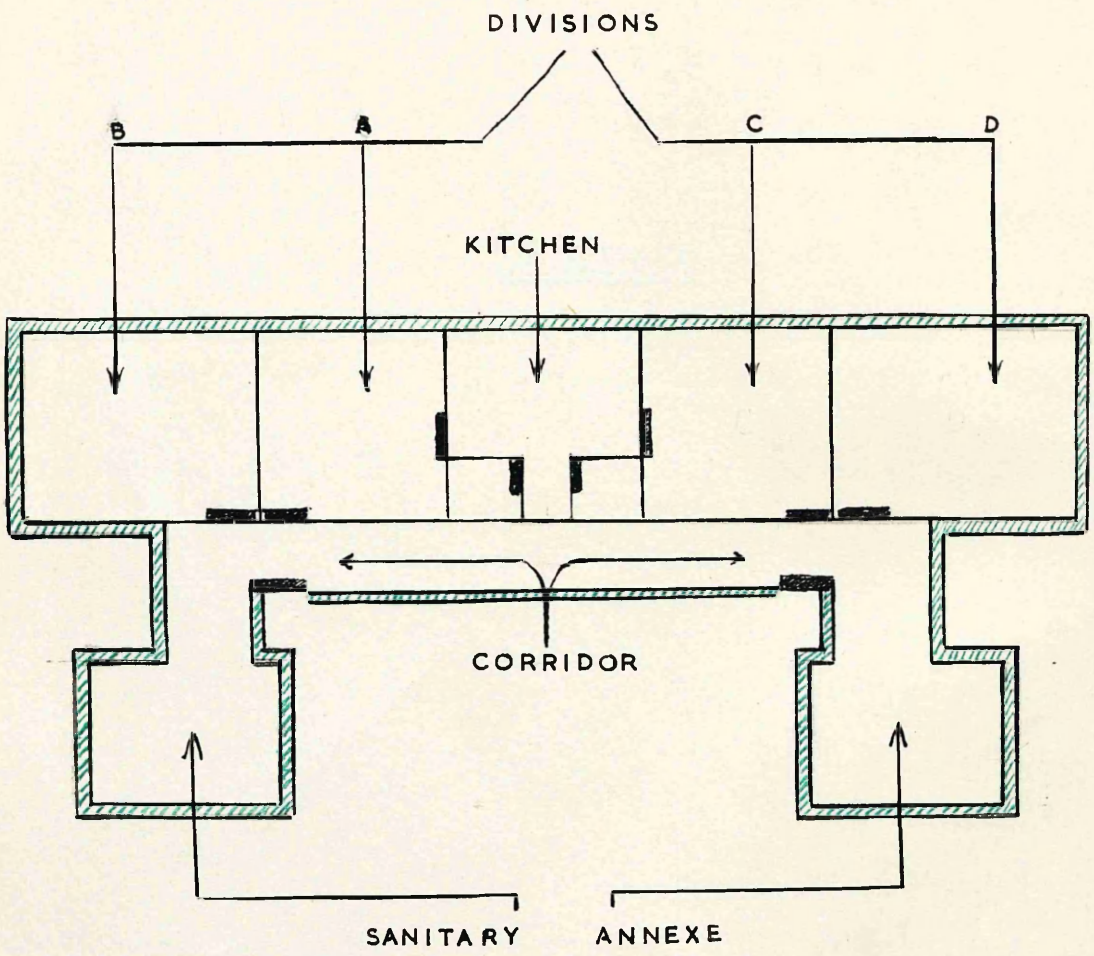
SECTION I.

Figure Ia

DIAGRAM TO SHOW LAYOUT OF WARD IN WHICH THE MAIN INVESTIGATION TOOK PLACE.



SECTION I.

Detailed description of the Investigation.

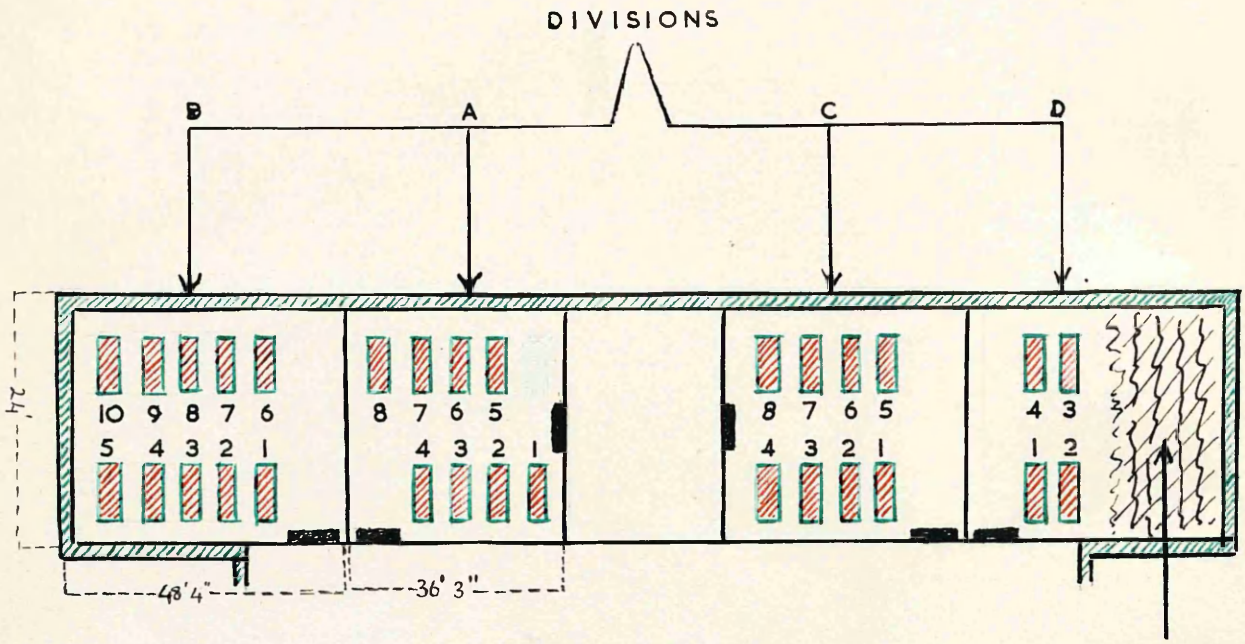
The investigations which form the basis of this thesis were carried out in Knightswood Infectious Diseases Hospital, in a ward used for the treatment of children suffering from pneumonia. The bacteriology was carried out by the writer in the hospital laboratory where all necessary facilities were available. The subjects of the investigations were the patients admitted to the ward between October 1949 and April 1950 and the medical, nursing and domestic staff who were in contact with them. The patients were all aged under 6 years and of these, more than half were aged under 1 year. Of the 131 patients admitted, 125 were notified to the hospital as cases of pneumonia. The remaining 6 included 5 non-infective transfers from other wards and 1 patient who was known to have tuberculous meningitis when he was admitted. Not all of the 125 patients had pneumonia although notified to the admission bureau as such. Some were subsequently found to have degrees of respiratory infection milder than pneumonia, or even to have no respiratory illness at all and to be suffering from some other condition. Whatever the ultimate diagnosis, all patients were investigated in a similar fashion.

Description of the Ward.

The ward was of the pavilion type and Figure Ia shows the plan of its four divisions. A thorough disinfection and

Figure Ib

DIAGRAM TO SHOW THE NUMBERING OF THE COTS IN THE WARD.



DISTANCE BETWEEN COTS = 6'6"

USED FOR STORAGE OF BEDS.

cleaning had been carried out prior to the admission of any patients.

Divisions A and C each contained 8 cots, Division B 10 cots and Division D 4 cots - a total of 30 cots. The remaining space in Division D was used for storage and not for patients.

The cots were numbered A1 - A8, B1 - B10, C1 - C8, D1 - D4, as shown in Figure Ib.

Position of Patient in the Ward.

For each patient a record was kept of the number of the cot to which he or she was admitted and of any subsequent changes in the position of this cot in the ward. Such changes were sometimes found necessary since Divisions A and C were more suitable for nursing the acutely ill and the very young patients, whereas Divisions B and D were more suitable for the older and the convalescent patients. Occasionally, acutely ill patients had to be admitted to these outer divisions.

Routine Procedures with Patients. (1) Before and immediately following admission to the ward.

Before being admitted to the ward, each patient was seen in the ambulance by the doctor on duty with three main objects in view. (a) To ascertain if there were any infectious condition present and if so, to have that patient isolated elsewhere. (b) To order any emergency treatment and (c) to obtain from the ambulance nurse or

relatives, if present, the history of the patient's illness and the details of any treatment given prior to admission. Details of this previous treatment were usually inadequate and inaccurate since, more often than not, there was no letter from the patient's general practitioner.

The patient was ready to be seen in the ward by the doctor within half an hour of admission. A thorough clinical examination was made and the necessary steps taken to determine the correct diagnosis, e.g. lumbar puncture, blood examination, urine examination, stool examination. An x-ray of the chest was taken the following day. If the patient had a definite respiratory infection treatment by chemotherapy was commenced as soon after admission as possible. The first patient was given sulphadiazine tablets, the second oral penicillin tablets, the third intramuscular penicillin, the fourth sulphadiazine, the fifth oral penicillin - and so on. Should treatment, as decided in this fashion be insufficient or unsuitable for the patient, other more suitable treatment was prescribed. One however wished to have approximately equal numbers of patients receiving each of the three drugs used. Dosage of the drugs varied according to the age of the patient and the details of the treatment routines are given in Section 2, (Chapter 4).

For the purposes of the writer's investigations, it was necessary to determine what organisms were present in

the nose and throat of the patient at the time of admission to the ward. Therefore, when the patient was examined in the ward and prior to the commencement of any treatment, the doctor on duty took 3 swabs, one from the anterior nose, one from the posterior nose and one from the throat. These were plated out on blood agar and gentian violet blood agar plates, incubated overnight and the organisms present identified subsequently. (See Appendix).

Note: The words 'a swabbing' are used to denote the taking of swabs from the three areas, the anterior nose, the posterior nose and the throat.

(2) After admission to hospital.

Chemotherapy was continued until midnight of the patient's seventh day in hospital when it was usually discontinued, unless there was some reason why it should be prolonged. Routine nursing treatment measures were continued throughout the patient's period in hospital.

A swabbing was made on the morning of the eighth day in hospital or, if treatment was continued for an extra day or so, on the morning following cessation of treatment. If later, a further course of treatment was prescribed, the patient was swabbed before and after treatment. The patient was finally swabbed on the day of dismissal or the day preceding it. Patients in hospital for long enough were swabbed at weekly intervals until dismissal. If there was any clinical evidence of possible cross

infection, the patients were swabbed. Any rise in temperature or any worsening of the clinical condition was regarded as being suspicious of infection and as such, warranted a swabbing. Patients with conditions other than pneumonia or bronchitis were also swabbed on the eighth day in hospital and then at weekly intervals and/or on dismissal.

This regime for subsequent swabbings was adhered to as closely as possible but on some occasions, pressure of work prevented all swabs being dealt with and a few swabbings were inadvertently missed. Moreover, an outbreak of smallpox in the hospital brought to a standstill the writer's bacteriological work for one week. No swabs were taken this week. However, 514 swabbings were taken out of a possible 547, which is an average of 3.9 per patient.

Records.

A case sheet and temperature chart were kept for each patient. Into the case sheet were entered details of the admission findings, the medical history and subsequent clinical progress and details of treatment, x-rays and any laboratory reports. A separate record card was kept for each patient with notes of the dates of admission and dismissal, the dates of all swabbings made and a note of the occasions which merited the taking of the swabs. On this card was also kept a record of the position of the cot to which the patient was admitted and any subsequent

changes in its position in the ward. On this card a note was also kept of the age group, the diagnosis and the nature and total dosage of the chemotherapeutic drugs given.

Routine Procedures with Members of Staff.

Ideally, for the purposes of this investigation, every member of the nursing, domestic and medical staff who came into contact with the patients would require to be swabbed. This was found to be impracticable and those actually swabbed included the ward-sister, the nurses, the part-time nurses, two maids, the hospital superintendent, one other doctor and the writer. Forty-two members of staff were involved. Other members of the staff, e.g. x-ray staff, the matron and administrative nursing staff, the doctors on duty for admission purposes, relieving nurses, etc. came into occasional contact with the patients but were not swabbed. As was the case with the patients, swabs were taken from the anterior nose, the posterior nose and the throat at each swabbing. Members of staff were swabbed on first coming to the ward and thereafter at weekly intervals and the total number of staff swabbings was 284.

A record card was kept for each member of staff and a note taken of the dates between which contact with the patients was possible, and also a note of the dates of all swabbings made. On this card any illness on the part of a member of staff was recorded.

Note: All swabs, both from patients and members of staff, excepting some of the admission swabs, were taken by the writer.

Dust Sampling.

Using a portable dust sampler, samples of the ward dust were collected and examined for their bacteriological content. Samples were taken at weekly intervals and a total of 275 samples was taken and examined. The dust was sampled from under each cot occupied, or, when the ward was full, from under a random selection of cots. To make this selection, cards carrying the numbers of the cots were put into a box and 10 pulled out. Samples of dust were taken from under these 10 cots.

Prior to opening this investigation 50 dust samples were taken from a ward, accommodating patients suffering from scarlet fever. The purpose of this was to test the efficiency of the dust sampler in the isolation of Streptococcus pyogenes which would be present in considerable quantity in the dust of such a ward. The dust sampler was proved efficient.

Other investigations.

(1) Investigation in a smaller ward used for the accommodation of patients suffering from measles.

After the investigations, described in previous pages, had been completed the writer decided to carry out a similar

investigation in a smaller ward which was then about to open for the treatment of children suffering from measles. In later pages the results are used for comparative purposes, and this in fact was the sole reason for the additional work being undertaken.

This smaller ward provided accommodation for 18 patients, there being 3 divisions each of which contained 6 cots. The same routine procedures were carried out for the 50 patients admitted to this ward as had been carried out in the main investigation. Swabbings were made with similar frequency and the records were kept in identical manner. Chemotherapy was given in similar fashion and each patient received either sulphadiazine, oral penicillin or intramuscular penicillin.

Members of the nursing staff in this ward were swabbed at weekly intervals and the dust was sampled each week using the portable dust sampler.

(2) Investigation in an overcrowded ward.

In order that the writer might determine whether overcrowding of the patients would significantly modify the incidence of cross infection, a cubicle in the isolation ward, normally accommodating 4 patients, was used for the treatment of 6 patients suffering from measles. In all, 18 patients were involved and each was investigated in similar fashion to the patients in the main investigation.

The dust was sampled at weekly intervals.

(3) Investigation of 4 patients suffering from measles.

These patients were nursed in a cubicle of the isolation ward and were swabbed by the writer every second day. The dust was sampled daily. The results are mentioned in the chapter on dust sampling and used for comparative purposes.

(4) The "Baby Families" Investigation.

Reference is made in later pages to this investigation. It took place concurrently with the writer's investigation and is reported by Landsman (1953). The results obtained give an indication of the bacteria carried in the nose and throat by healthy children and adults and are therefore used for comparison with the writer's findings for the children with a respiratory infection and for the hospital staff. The families studied by Landsman were considered to be a true sample of the healthy general population. They were selected from the general population because of the recent occurrence of a birth in the household. The posterior nose and throat of each member of the family were swabbed and the swabs thus obtained were dealt with in similar fashion to those obtained in the writer's investigation. The bacteriological work was done in the same laboratory as was used by the writer, using the same methods.

(5) The "Random Families" Investigation.

This investigation took place two years prior to the present investigation. It was carried out by Landsman in like fashion to the above but the families swabbed were selected by random selection from the general population. The bacteriology was carried out at Knightswood Hospital again using the same techniques. The results obtained are used in comparison with the writer's findings for children with a respiratory infection and for hospital staff.

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SECTION 2.

SECTION 2.

The Results of the Investigation.

During the six month period covered by this investigation, a considerable volume of information was amassed concerning the patients and staff involved in the investigation and concerning the floor dust of the ward. The data obtained will be dealt with under the following headings:

Chapter I. Details about (a) the patients involved in the investigation and (b) the organisms present in the noses and throats of the patients on admission to the ward and, during their subsequent period in hospital.

Chapter 2. The results of the investigation of the flora of the noses and throats of the members of the medical, nursing and domestic staff who were in contact with these patients.

Chapter 3. The organisms isolated from the floor dust of the ward during the period of the investigation.

In the pages that follow, the information gained will be set down, in a factual manner, with no more than passing comment on the significance of the findings which are more fully discussed elsewhere.

Chapter 4 completes the section. In this chapter there are given the results of the investigation of the changes which the use of antibiotics brings about in the flora of the noses and throats of the patients. Their

role in the prevention of cross infection is also considered.

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Note:

All the bacteriological findings from the examinations of the patients and the staff are shown in tabular form in the appendix.

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Chapter I.

Investigation of the Patients.

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(a) The diagnosis and age of the patients involved.

A total of 131 patients was admitted to the ward and investigated. Not all patients were suffering from pneumonia although they might be admitted as so doing. It was advisable that attention be paid to the exact diagnosis of these patients in order to determine if the diagnosis had any bearing on the organisms present in the nose and throat or on the incidence or the effects of cross infection. Similarly, the age of the patients might be an influencing factor and as such, must be considered.

The proper classification of the form of a respiratory infection may present considerable difficulty especially in the young child. On clinical grounds, it is not easy to decide whether a child has an acute bronchitis, a broncho-pneumonia or an actual lobar or segmental consolidation. Classification by the etiology is equally difficult as Skadding pointed out.(1953). It is only recently that the importance of inhalation of upper respiratory secretions, as an etiological factor in the causation of pneumonia, has been realised. Only recently has the term viral pneumonia been correctly used for infections of true viral origin.

It was thus necessary to adopt some simple form of classification which merely defined the anatomical site affected and the main characteristics of the child's illness. Three diagnostic groups were formed.

(1) Those patients with a mild bronchitis. Such patients were only moderately ill and had only a minimal systemic upset. Cough was the main clinical feature and pyrexia was not marked.

(2) Those patients with a more severe attack of bronchitis or a bronchiolitis. The patients in this group were more sharply ill than the foregoing, symptoms of cough and breathlessness being more severe and the systemic upset more marked. The average temperature was higher than was found in the patients in Group I. In both groups, the x-ray of chest was clear.

(3) Those patients with pneumonia. In only about half of the patients who were considered to have pneumonia, did the x-ray of chest show evidence of the pneumonic consolidation. For convenience, these were called, "Group 3a" patients. In all but one, the x-ray showed an area of lobar or a small area or small areas of segmental consolidation. In the remaining patient, the consolidation was scattered throughout both lungs.

In the other patients, who accounted for slightly more than half of the patients with pneumonia, the x-ray of chest did not show any evidence of consolidation and a diagnosis of pneumonia had to be made on clinical grounds only. They were "Group 3b" patients and were often extremely ill with a high temperature, rapid distressed respirations and cyanosis.

Cough was very troublesome and frequently the respiratory embarrassment was considerable. On examination of the chest, there were often areas of harsh breathing with widespread, fine, crackling, inspiratory rales. There was usually an associated bronchitis with rhonchi and coarser rales. These patients were suffering from broncho-pneumonia on clinical grounds, yet the x-ray appeared to be clear. One does however feel justified in blaming the x-ray technique to some extent for this. It is well known, that even with the most efficient equipment, it is difficult to x-ray the chest of a young child satisfactorily and moreover, if one does presume that the distribution of the consolidation was broncho-pneumonic, the areas might well have been too small and of insufficient density to be radio-opaque. In view of this, the writer is of the opinion that the patients in "Group 3b" had in actual fact broncho-pneumonia whereas, those in "Group 3a", had lobar or segmental pneumonia.

Of the 131 patients admitted to the ward, 104 had either bronchitis or pneumonia. There were 19 patients with mild bronchitis, 18 with severe bronchitis and 67 with pneumonia. In 32 of the patients suffering from pneumonia there was radiological evidence of pneumonia. In the other 35 patients the diagnosis of pneumonia was made on clinical grounds alone. As was explained above, the former were considered to have lobar or segmental pneumonia, the latter to have broncho-pneumonia.

TABLE I.

Conditions from which the patients suffered in the "Miscellaneous group".

Diagnosis.	No.	Diagnosis.	No.
Primary tuberculosis.	5	Dysentery.	1
Miliary tuberculosis.	1	Generalised herpetic infection.	1
Acute disseminated tuberculosis.	1	Pyogenic meningitis.	2
Tuberculous meningitis.	1	Tonsillitis.	1
Pulmonary atelectasis.	1	Mumps.	1
Coryza.	3	Non infective transfers from	
No disease found.	2	other wards.	5

TABLE 2.

Age distribution of the patients.

Ages.	All patients.		Patients with bronchitis or pneumonia.	
	No.	%.	No.	%.
One year and under.	69	52.7	61	58.7
0 - 1 month	4		4	
1 - 6 months	36		32	
6 - 12 months	29		25	
Over 1 year	62	47.3	43	41.3
TOTAL:	131	100.0	104	100.0

TABLE 3.

Showing the difference in ages of patients with pneumonia confirmed radiologically and those in whom the pneumonia was not thus confirmed.

	Total	Pneumonia confirmed radiologically.		Pneumonia not confirmed radiologically.	
		No.	%	No.	%
Aged 1 year and under.	61	10	16.4	28	45.9
Aged over 1 year.	43	22	51.2	7	16.3
All ages.	104	32	30.8	35	33.6

A further 2 patients had pnuemonia and empyema due to Staphylococcus aureus.

The remaining patients, 25 in number, formed the "miscellaneous group". The conditions from which they were suffering are shown in Table I. Included in this group were 5 non-infective convalescent transfers from other wards. All these patients were however included in the investigation since they also could be sources or victims of cross infection. Moreover, they constituted, as it were, a control group for the patients suffering from bronchitis or pneumonia.

The age distribution.

All patients were aged under 6 years, but 52.7 per cent of them were aged under 1 year. The age distribution is summarised in Table 2. Neo-natal patients were virtually excluded from the investigation.

The age of 1 year was taken as a suitable dividing line between the infant and the young child because of the known severity of respiratory infection in the child of 1 year and under and because these children are especially prone to cross infection. (Medical Research Council, 1951).

It was noted that most of the children suffering from pneumonia which was confirmed radiologically, were aged over 1 year, whereas, when the pneumonia was not so confirmed, and the diagnosis made on clinical grounds alone, the child was usually aged under 1 year. (Table 3).

TABLE 4.

Correlating the organisms carried in the nose or throat with the diagnosis.

	Total No. of patients.	Patients carrying Pneumo.		Patients carrying S. pyog.		Patients carrying Staph.aur.	
		No.	%.	No.	%.	No.	%.
Patients suffering from bronchitis or pneumonia.	104	69	66.3	13	12.5	16	15.4
Patients with a staphylococcal pneumonia and empyema.	2	0		0		1	
"Miscellaneous" patients.	25	12	48.0	11	44.0	6	24.0
All patients	131	81	61.8	24	18.3	23	17.6

NOTE:
Pneumo. = Pneumococcus.
S. pyog. = Streptococcus pyogenes.
S. aur. = Staphylococcus aureus.

(b) The Results of the Initial Swabbing of the Patients.

There was isolated from the nose or throat of 61.8 per cent of the patients, a pneumococcus; from 18.3 per cent Streptococcus pyogenes and from 17.6 per cent Staphylococcus aureus.

On comparing the findings for the 104 patients suffering from either bronchitis or pneumonia with those for the 25 patients in the "miscellaneous group" one finds that the figures to be compared are 66.3 per cent and 48 per cent for the pneumococcus, 12.5 per cent and 44 per cent for the streptococcus and 15.4 per cent and 24 per cent for the staphylococcus. (Table 4).

The higher pneumococcal and the lower streptococcal isolation rate for the former group of patients, is of little significance, considering the dissimilar totals involved in the two groups and the mixed population of the "miscellaneous group."

It was noted that frequently the heaviness of the growth of pneumococci on the culture plate from the nose and throat swabs was considerable, and especially was this so when the patients were suffering from bronchitis or pneumonia. One was given the impression that this growth was heavier than that usually obtained when pneumococci are isolated from swabs taken from healthy children. Unfortunately the writer has no comparable results from healthy children to confirm

TABLE 5.

Showing the types of pneumococci isolated from the patients.

All Patients.			104 "bronchitic and pneumonic" patients.			25 miscellaneous patients.	
Type of Pneumo-coccus Isolated.	No. of Isol-ations.	% of all Isolat-ions.	Type of Pneumo-coccus Isolated.	No. of Isol-ations.	% of all Isolat-ions.	Type of Pneumo-coccus Isolated.	No. of Isol-ations.
23	17	18.3	23	16	19.8	19	3
6	17	18.3	6	15	18.5	6:11	2
19	11	11.8	19	8	9.9	12:14:18	1
11:14	6	6.5	1:14:21	5	6.2	23:33.	
1:21	5	5.4	11	4	4.9		
7:18:22	3	3.2	7:22	3	3.7		
8:12:20	2	2.2	8:18:20	2	2.5		
33			2:5:10	1	1.2		
2: 5:10	1	1.1	12:17:29				
17:29:31			31:33:34				
34:Gr.A.			Gr.A.				
U.T.T.			U.T.T.				

NOTE: The type of one pneumococcus could not be determined, and is referred to as "U.T.T." Another pneumococcus gave a reaction to Group A antiserum but could not be further identified.

this point.

Streptococcus pyogenes on the other hand, was never isolated freely from the patients and of the 16 patients who carried Staphylococcus aureus, only 7 carried the organism in any great quantity.

The Type of Pneumococcus Isolated.

Only one type of pneumococcus was isolated from the majority (86.4 per cent) of the patients carrying a pneumococcus. In a further 12.3 per cent, two types of pneumococci were isolated and from the remaining 1.2 per cent (one patient) three types were isolated. Of all the pneumococcal types, types 23 and 6 were most frequently isolated and each accounted for 18.3 per cent of the isolations. The next most frequently found was type 19, then types 1 and 14, then types 11 and 21. Considering only the patients with either bronchitis or pneumonia the same type prevalence was observed.

Table 5 further shows that, with the exception of type 1, the pneumococcal types associated with adult pneumonia were seldom isolated from those young patients.

Correlation of the Bacteriological findings with:

a) the diagnostic category of the patient.

b) the age of the patient.

a) Patients suffering from pneumonia were carriers of pneumococci more frequently than those suffering from bronchitis. The two groups however are not strictly comparable

TABLE 6.

Correlating the organisms carried in the nose and throat with the nature of the respiratory infection.

Diagnosis	Total No. of Patients	Patients carrying Pneumo.		Patients carrying S. pyog.		Patients carrying S. aur.	
		No.	%.	No.	%.	No.	%.
(1) Mild Bronchitis.	19	11	57.9	2	10.5	3	15.8
(2) Severe Bronchitis or Bronchiolitis.	18	9	50.0	2	11.1	5	27.8
(3) Pneumonia.	67	49	73.1	9	13.4	8	11.9
a) Pneumonia confirmed by radiography.	32	27	84.4	4	12.5	3	9.4
b) Pneumonia not confirmed by radiography.	35	22	62.9	5	14.3	5	14.3

TABLE 7.

Correlating the organisms carried in the nose and throat with the age of the patient.

Age	Total No. of Patients.	Patients carrying Pneumo.		Patients carrying S.pyog.		Patients carrying S.aur.	
		No.	%.	No.	%.	No.	%.
Under 1 year	61	33	54.1	9	14.8	12	19.7
Over 1 year	43	36	83.7	4	9.3	4	9.3
TOTAL:	104	69	66.3	13	12.5	16	15.4

because of the different numbers of patients involved. The percentage of pneumococcal carriers was however significantly higher when the pneumonia was confirmed radiologically, i.e. pneumonia of a lobar or segmental distribution than when it was not so confirmed, i.e. pneumonia of a bronchopneumonic distribution. No type of pneumococcus was especially found in any particular diagnostic category.

Finally, the number of carriers of Streptococcus pyogenes and Staphylococcus aureus in any particular diagnostic category was too small for the purposes of comparison. (Table 6).

b) Pneumococci were isolated more frequently from patients aged over 1 year than from patients aged under 1 year. For the Staphylococcus aureus the converse was true. (Table 7.)

In interpreting the difference between the two age groups, as regards pneumococcal isolation, one must remember that most of the patients with a radiologically proved pneumonia were aged over 1 year and those patients have already been shown to have a high isolation rate for pneumococci.

It would therefore seem that, when pneumonia occurs in a child over 1 year there is frequently radiological confirmation of a lobar or segmental consolidation, and moreover, pneumococci are more frequently isolated from those patients, either as a result of their older age or as a result of the character of the respiratory infection.

TABLE 8.

Demonstrating the frequency with which the patients were swabbed.

No. of times each patient was swabbed.	All Patients. (131).	Patients suffering from bronchitis or pneumonia. (104).	Other Patients. (27).
1	3	0	3
2	19	14	5
3	58	52	6
4	20	20	0
5	9	6	3
6	7	6	1
7	7	3	4
8	2	0	2
9	2	2	0
10	0	0	0
11	2	1	1
12	1	0	1
13	0	0	0
14	0	0	0
15	1	0	1

The type prevalence in the two groups was investigated and it was found that all 5 isolations of type I were from patients over 1 year in age. Furthermore, type 6 accounted for 29.5 per cent of isolations from the older group whereas it accounted for only 5.4 per cent of isolations from the younger group of patients aged under 1 year. Other types occurred with equal prevalence in the two groups.

(c) The Results of Subsequent Swabbings of the Patients.

All patients were swabbed on their eighth day in hospital. In those patients receiving chemotherapy, this corresponded to the day following cessation of treatment. Swabs were also taken on the day of dismissal, if this were more than two days after the eighth day. In those patients who were in hospital for more than two weeks, swabs were taken at weekly intervals until dismissal, when further swabs were taken. Should there be any relapse in the clinical condition, or symptoms suggestive of an acquired infection, swabs were also taken.

In all, 514 swabbings were made from the total of 131 patients (an average of 3.9 per patient). The numbers of patients swabbed once and more often are summarised in Table 8 where it will be seen that the largest number of swabbings for one person was fifteen.

The 3 patients who were only swabbed once were (1) a patient suffering from tuberculosis who died a few days after

TABLE 9.

Comparing the occurrence of the various organisms amongst the various groups of patients at each swabbing.

	Serial No. of Swabbing.	Total No. Patients Swabbed.	Organism carried. Pneumo. No.	Pneumo. %	S. pyog. No.	S. pyog. %	S. aur. No.	S. aur. %
All patients.	1	131	81	61.8	24	18.3	23	17.6
	2	128	63	49.2	9	7.0	46	35.9
	3	109	67	60.6	11	10.1	44	40.4
Patients with bronchitis or pneumonia.	1	104	69	66.3	13	12.5	16	15.4
	2	104	53	51.0	7	6.7	41	39.4
	3	90	56	62.2	9	10.0	38	42.2
Patients with other conditions.	1	25	12	48.0	11	44.0	7	28.0
	2	23	10	43.5	2	8.7	5	21.7
	3	18	11	61.1	2	11.1	6	33.3
Patients with staphylococcal pneumonia and empyema.	1	2	0		0		1	
	2	1	0		0		1	
	3	1	0		0		0	

admission. (2) The fatal case of staphyloccal pneumonia and empyema and (3) a patient who was transferred to another ward a few days after admission. The majority of the 19 patients swabbed only twice were dismissed one or two days after the second swabbing. A few were not swabbed on dismissal because of pressure of work.

Changes in carrier rates of the various organisms.

Results from the first, second and third swabbings can be compared in Table 9. Later swabbings are not mentioned but are relatively so few in number that comparison would be fallacious. There was considerable difference, from one swabbing to another, in the percentage of patients carrying each organism. The various organisms will be considered separately.

The Pneumococcus.

On admission, 81 of the patients carried a pneumococcus. Despite the fall in the percentage of patients carrying a pneumococcus at the second swabbing, another 14 patients had become carriers of a pneumococcus. A further 10 patients acquired a pneumococcus for the first time at the third swabbing. Two other patients acquired a pneumococcus, one at the fourth and one at the seventh swabbing. Thus, of 131 patients admitted, 107 (81.7%) carried a pneumococcus and of the 104 patients suffering from bronchitis or pneumonia, 88 (84.6%) carried a pneumococcus while in hospital.

When the pneumococci are considered as individual types, one finds that some of the patients carried more than one type while in hospital. Thus, instead of what would appear to be 26 patients acquiring a pneumococcus after admission, one finds that actually 48 patients acquired a new type of pneumococcus. Of these, 14 acquired two new types after admission. Furthermore, one finds that although a patient may appear to have lost a pneumococcus, the same type may reappear in later swabbings. The acquisition of these new types and the probable sources from which they came, are discussed later.

Streptococcus pyogenes.

Although there was a fall in the percentage of patients carrying Streptococcus pyogenes in swabbings after the first, 25 patients acquired Streptococcus pyogenes after admission. In all, 49 patients (38.2%) carried this organism. Thirty-two of these patients were from the group of 104 patients suffering from bronchitis or pneumonia (30.8%). In most of the patients, only a few colonies grew from the swabs, but in 8 patients, a considerable number of colonies were isolated. All of these 8 patients acquired the organism after admission to the ward. In only 2 of these patients were there any symptoms of illness presumably due to the acquisition of Streptococcus pyogenes.

Staphylococcus aureus.

It was in the frequency of isolation of this organism that there was most change in subsequent swabbings. Whereas 23 patients carried this organism when swabbed for the first time, 46 patients were found to be carrying Staphylococcus aureus when swabbed for the second time. Of these, only 4 had previously carried the organism. Thus, 42 (32.1%) of the patients acquired Staphylococcus aureus after being approximately one week in the ward. Twenty of these patients still carried it when swabbed a third time and a further 17 acquired the organism for the first time at their third swabbing. Finally, 3 more patients acquired this organism on the fourth and 2 on the sixth swabbing.

The total number of patients carrying a Staphylococcus aureus at one time or another was 87 (66.4%). Of the 104 patients with bronchitis or pneumonia 71 (68.2%) carried the organism while in hospital, 55 more than on admission. That such a marked increase should occur in hospital suggests either the infection of patients by other patients or by members of the medical, nursing or domestic staff in contact with the patients.

Results of Swabbing when Clinical features suggested a relapse or an acquired infection.

While in hospital, 21 of the patients had a relapse in their clinical condition or showed some feature which suggested an infection, newly acquired.

Three of the patients had 2 such incidents.

On each occasion, swabs were taken from the nose and throat. No change, or at least no appreciable change was found in the bacteriological flora of the nose and throat in 13, that is in the majority of instances. In a further 2, a pneumococcus had been acquired; in 4, Streptococcus pyogenes and in 5, Staphylococcus aureus had been acquired. A detailed description of the various instances suggesting a relapse or an acquired infection is given in Section 3.

(d) The Sites of Isolation of the Organisms.

It was apparent from the bacteriological findings that the various organisms seemed to favour different parts of the nose or throat for their environment. To find out exactly how true this was, the various carrier rates for the organisms under question in a) the anterior nose, b) the posterior nose and c) the throat, were compared. (Table 10).

The pneumococcus was found most often in the posterior nose but also occurred frequently in the anterior nose. The throat was the site of isolation in a smaller number of instances. A considerable proportion of the patients, 40.7 per cent, carried a pneumococcus in both the anterior and the posterior nose, and in 28.4 per cent, the throat was additionally involved.

Streptococcus pyogenes was isolated most frequently from the throat. The nose was less often the source but when it was, the posterior nose was involved more often

TABLE 10.

Showing the number of isolations of the various organisms from the various sites:

(a) Admission Results:

Site of Isolation	Pneumo.		S. pyog.		S. aur.	
	No.	%.	No.	%.	No.	%.
P.N. only.	15	18.5	5	20.8	6	26
A.N. only.	5	6.2	1	4.2	11	48
T. only.	1	1.2	15	62.5	1	4.3
P.N. + A.N.	33	40.7			1	4.3
P.N. + T.	4	4.9				
A.N. + T.			3	12.5	1	4.3
P.N. + A.N. + T.	23	28.4			3	13.1
TOTAL:	81	99.9	24	100.0	23	100.0
P.N.	75	45.8	5	18.5	10	32.3
A.N.	61	37.2	4	14.8	16	51.6
T.	28	17.0	18	66.6	5	16.1
TOTAL:	164	100.0	27	99.9	31	100.0
Isolation rate for 131 admissions:	61.8%		18.3%		17.6%	

P.N. = Posterior nose.
A.N. = Anterior nose.
T. = Throat.

TABLE 10.

(b) Results from all swabbings made:

Site of Isolation	Pneumo.		S. pyog.		S. aur.	
	No.	%.	No.	%.	No.	%.
P.N. only.	74	25.0	16	20.5	18	10.8
A.N. only.	14	4.7	3	3.8	69	41.3
T. only.	3	1.0	52	66.7	6	3.6
P.N. + A.N.	154	52.0			61	36.5
P.N. + T.	9	3.0			4	2.4
A.N. + T.	2	0.7	5	6.4	2	1.2
P.N. + A.N. + T.	40	13.5	2	2.6	7	4.2
TOTAL:	296	99.9	78	100	167	100.
P.N.	277	51.2	18	20.7	90	36.3
A.N.	210	38.8	10	11.5	139	56.0
T.	54	10.0	59	67.8	19	7.7
TOTAL:	541	100.0	87	100.0	248	100.

TABLE II.

Showing the sites of isolation when Streptococcus pyogenes occurred in quantity.

Serial No. of Patient.	Serial No. of Swabbing.	Site of Isolation		
		P.N.	A.N.	T.
71	2			T+++
41	3			T++
42	5	P+	A+	T+++
86	3	P+++		
88	2			T++
98	3	P+++		
	4	P+++		
	5	P++		
	7	P+++		
124	2			T++
128	4			T++

\+ = scanty colonies.
++ = moderate number of colonies.
+++ = large number of colonies.

than the anterior nose. Simultaenous isolation from more than one site seldom occurred.

The posterior nose was heavily infected in only 2 of the 8 patients carrying Streptococcus pyogenes in considerable numbers. In the remaining 6, the throat was the site which was heavily infected with streptococci. (Table II).

In contrast to the other two organisms Staphylococcus aureus favoured particularly the anterior nose as its environment. The posterior nose was involved to a lesser extent and the throat, even less frequently.

When one considers the findings from not only the first but from all swabbings, one finds a similar tendency for each organism to occur especially in one part of the nose or throat. Furthermore, one finds that Staphylococcus aureus also occurred simultaneously in the anterior and the posterior nose in a number of instances. This occurred in 36.5 per cent of all positive swabbings. Although high, the figure was however not as great as the comparable figure for the pneumococcus, 52 per cent.

Sites of Isolation of Multiple types of Pneumococcus.

On 17 occasions throughout the investigation a patient was found, when swabbed, to have more than one type of pneumococcus present. In 16 of these patients, pneumococci were isolated from more than one of the three sites swabbed. The quantities isolated varied from the various sites. Almost each instance varied in the combination of sites involved

and in the numbers of pneumococci of each type present in each site. It would seem, however, that when multiple types occur, more than one site is usually involved and moreover, the use of the anterior nose swab increases the likelihood of a second type being found. Of the 17 occasions in this series where more than one type was isolated, only 9 would have been discovered if the anterior nose had not been examined.

Details of the isolation of an organism from more than one of the three sites examined.

When the results of the swabbings were recorded, a rough estimate was made of the number of colonies of each organism present. If colonies were scanty, the quantity was denoted by "+", if in large amount throughout the plate by "+++", if in only moderate amount by "++". By this means it was possible to compare the number of organisms present in one site with the number in another. (Table 12).

The pneumococcus was the organism most frequently isolated from more than one site. Whether 2 or 3 sites were involved, the usual finding was that no particular site was more heavily infected than another (in 81.5% of all instances). Where there was a difference between the growth from one site and another, the tendency was for the posterior nose to produce a heavier growth than either the anterior nose or the throat.

Little can be said about Streptococcus pyogenes since,

TABLE 12.

Showing the relative number of colonies of each organism obtained from the various sites of isolation when more than one site was involved.

All swabbings:

Sites of Isolation.	Pneumo.		S. pyog.		S. aur.	
P.N. + A.N.	154		0		61	
	No.	%	No.	%	No.	%
P.N. = A.N.	137	89.0			41	67.2
P.N. > A.N.	7	4.5			8	13.1
P.N. < A.N.	3	1.9			12	19.7
+ double types:	+7	+4.5				
P.N. + T.	9		0		4	
	No.	%	No.	%	No.	%
P.N. = T.	3	33.3			2	50.0
P.N. > T.	3	33.3			1	25.0
P.N. < T.	1	11.1			1	25.0
+ double types:	+2	22.2				
A.N. + T.	2		5		2	
	No.	%	No.	%	No.	%
A.N. = T.	1	50.0	5	100.0	2	100.0
A.N. > T.	1	50.0				

TABLE 12 (Continued).

Sites of Isolation.	Pneumo.		S. pyog.		S. aur.	
	40		2		7	
PN+AN+T:	No.	%	No.	%	No.	%
PN=AN=T	26	65.0			2	28.6
PN=AN>T	4	10.0			1	14.3
T>PN=AN			2	100.0	1	16.3
PN>AN=T	2	5.0				
PN<AN=T					2	28.6
(PN>AN)>T					1	14.3
+ double types:	7	17.5				

in only 7 swabbings was the organism present in more than one site. Equal numbers of organisms were isolated from each site in 5 of the 7 instances.

Staphylococcus aureus however occurred in both the anterior and the posterior nose in 61 swabbings. In 67.2 per cent of these, an equal number of organisms was isolated from the two sites. The anterior nose was however more heavily infected in 19.7 per cent and the posterior nose, in the remaining 13.1 per cent. Other combinations of sites of isolation occurred too infrequently for one to form any opinion as to which site was usually most heavily infected.

In conclusion one can say that although the pneumococcus, Streptococcus pyogenes and Staphylococcus aureus can each occur in the anterior nose, the posterior nose and the throat, each has its site of election. When more than one site is involved it is usual for each site to be infected with equal heaviness but if not, the commoner site of isolation is the more heavily infected. This favouring of a particular site has, one feels, an important part to play in the ease with which and the method by which an organism from one individual can pass to another. As such, it is a factor of considerable importance in any study on cross infection involving these particular organisms.

(e) Summary of Findings.

1). The patients involved in this study were the 131 children admitted to a ward, set aside for the treatment of children suffering from pneumonia. They were admitted between October 1949 and April 1950.

2). Of these patients, 104 were found to have an acute respiratory illness and of these, 67 had pneumonia and 37, varying degress of bronchitis. A further 2 patients had pneumonia and empyema due to Staphylococcus aureus. The remaining patients were suffering from a variety of conditions.

3). Of the 131 patients, 61.8 per cent carried pneumococci, 18.3 per cent carried Streptococcus pyogenes and 17.6 per cent Staphylococcus aureus.

4). The pneumococcal types most frequently found were 6 and 23. The fixed types, with the exception of type I were seldom found.

5). Patients suffering from pneumonia as compared with patients suffering from bronchitis had a higher isolation rate of pneumococci.

6). The isolation rate of pneumococci was higher when the patient suffered from lobar or segmental pneumonia than when they suffered from bronchopneumonia.

7). Patients over 1 year, as compared with patients under 1 year of age had a higher isolation rate of

pneumococci and a lower isolation rate of Staphylococcus aureus.

8). Type 6 was the most prevalent type in patients over 1 year and type 23 in patients under 1 year of age. All the type 1 isolations occurred in the older age group.

9). Although the total number of patients carrying pneumococci and Streptococcus pyogenes decreased after the first swabbing there were numerous instances of new types of pneumococci and of Streptococcus pyogenes being acquired after admission to hospital.

10). Staphylococcus aureus was frequently acquired by patients throughout the investigation and 42 patients acquired it during their first week in hospital.

11). It was found that the pneumococcus was most often isolated from the posterior nose, Streptococcus pyogenes from the throat and Staphylococcus aureus from the anterior nose of the patients.

12). The pneumococcus was frequently isolated from more than one site. Usually both the anterior and the posterior nose were involved and in a number of patients, the throat was additionally involved. Staphylococcus aureus was isolated from both nasal sites in a proportion of instances although not as frequently as was the pneumococcus.

* * * * * * *

Chapter 2.

Investigation of the Staff.

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TABLE 13.

Showing the number of times the various members of the Staff were swabbed.

No. of times swabbed.	No. of Staff.	No. of times swabbed.	No. of Staff.	No. of times swabbed.	No. of Staff.
1	9	7	3	13	1
2	1	8	3	14	1
3	3	10	2	17	1
4	4	11	3	22	1
5	4	12	2	23	1
6	3				

(a) The frequency of swabbing the members of staff.

Each member of the staff was swabbed on their first day on duty in the ward under scrutiny, and at weekly intervals thereafter. A total of 285 swabbings was made from the 42 individuals involved in this part of the investigation. The staff were changed frequently so that a considerable number were swabbed on only one occasion. The others however were swabbed anything from 2 to 23 times. Details of the frequency of swabbing are shown in Table 13. The 9 people swabbed on only one occasion included 4 part-time and 3 full-time nurses who were less than a week in the ward and the hospital superintendent and one other doctor who occasionally came into contact with the patients.

(b) The organisms isolated from the members of the staff.

The most frequently found organism in the noses and throats of the members of the staff was Staphylococcus aureus. This was the finding on the first swabbing and also on subsequent swabbings. Streptococcus pyogenes and the pneumococcus were less commonly isolated but were each isolated from a similar number of members of staff at their first swabbing. The figures for subsequent swabbings were rather variable. (Table 14). Frequently more than one of the organisms was found at one swabbing.

TABLE 14.

Showing the number and percentage of members of the Staff carrying each organism at each swabbing.

Serial No. of Swabbing	Total	Pneumo.		S.pyog.		S.aur.	
		No.	%	No.	%	No.	%
1	42	9	21.4	8	19.0	18	42.9
2	33	5	15.2	9	27.3	17	51.5
3	32	8	25.0	7	21.9	15	46.9
4	29	7	24.1	8	27.6	15	51.7
5	25	3	12.0	8	32.0	17	68.0
6	21	4	19.0	7	33.3	13	61.9
7	18	2	11.1	1	5.6	10	55.6
8	15	4	26.7	3	20.0	8	53.3
9	12	6	50.0	4	33.3	7	58.3
10	12	2	16.5	4	33.3	5	41.7
11	10	4	40.0	3	30.0	2	20.0
12	7	0		4		4	
13	5	1		2		1	
14	4	0		2		1	
15	3	1		1		2	
16	3	1		1		1	
17	3	0		2		1	
18	2	0		1		1	
19	2	0		1		0	
20	2	0		1		0	
21	2	0		1		0	
22	2	0		0		0	
23	1	0		1		0	

From studying the results, as tabulated in the appendix, it will be seen that all but two of the members of staff carried at least one of the organisms. Neither of these two exceptions were swabbed more than once. It is also obvious that, as the total number of swabbings for the individuals increased, so did the number of individuals who carried more than one of the three organisms. One might presume that, if swabbing were carried on indefinitely the carrier rate for each organism would approach 100 per cent. That this would be so, is confirmed by the work of Straker, Hill and Lovell (1939), who carried out swabbing of the naso-pharynx in a group of adults over a number of years and stated that, any rate short of 100 per cent understates the actual frequency of the carrier state. Dudley (1932) working with diphtheria carriers found that a carrier rate based upon one swabbing was much less than it would have been if serial swabbings had been taken into account.

Pneumococcus.

A pneumococcus was isolated from the nose or the throat of only 9 of the 42 members of the staff when they were swabbed for the first time. However, in subsequent swabbings other members of the staff were found to have acquired a pneumococcus and by the end of the investigation, pneumococci had been isolated from 21 of them.

TABLE 15.

Showing the incidence of the various pneumococcal types amongst the Staff.

Type of Pneumococcus.	Number of times isolated.	Percentage of all isolations.
6	6	19.4
10	3	9.7
2:3:14:15:22:23:25.	2	6.5
5:11:18:19:20:29:37:Gr.F.	1	3.2

Note: The type of one pneumococcus could not be identified. It did, however, give a reaction with Group F antisera.

Type of Pneumococcus isolated.

The majority of the members of staff carried only one type of pneumococcus. Thus, 13 members of the staff carried one type of pneumococcus, 6 carried two types and 2 carried three types during the investigation. In one person, and on one occasion only, were two types isolated simultaneously from a swabbing.

Of the 31 types of pneumococcus isolated from the staff, 6 were type 6 (Table 15). This type was also the type most frequently isolated from the adult members of the general population as sampled by Landsman (1953) in the "Baby Families" investigation. Other types were isolated less frequently and it was noted that type 2 and type 3, both of which are pathogens of adult pneumonia, each occurred once.

Streptococcus pyogenes.

At the time of their initial swabbing, 8 (19%) of the staff carried Streptococcus pyogenes in the nose or throat, and from 5 of these, the organism was isolated in considerable quantity. A further 19 members of the staff acquired this organism during the period of the investigation and in 5 of them, the organism was acquired in considerable quantity. One thus finds that the total number of members of staff (27) carrying Streptococcus pyogenes at some time during the investigation was almost as great as the number carrying Staphylococcus aureus (32). The majority of acquisitions

occurred in the weeks which followed the development of scarlet fever in one of the patients. There was also an associated increase in the number of patients carrying and acquiring Streptococcus pyogenes in these weeks.

Staphylococcus aureus.

Staphylococcus aureus was isolated from the nose and throat in 32 (76.2%) of the members of staff, 14 of whom acquired the organism after their first swabbing.

Cultures from 26 of the carriers were further examined. As a result of this, 24 of the members of staff were found to carry coagulase positive organisms on at least one occasion during the period of the investigation. Moreover, 19 (45.2% of all members of staff) carried organisms which were not only coagulase positive but also penicillin resistant and 8 of them did so on more than one occasion. The potential danger to the patients was thus considerable, especially as the actual figures in all probability would have been higher had all cultures been tested.

(c) Association of illness with changes in the Bacteriological flora.

Despite the large number of times the members of the staff acquired an organism during the investigation, on only 3 occasions was there any illness associated. One nurse developed tonsillitis and Streptococcus pyogenes was isolated in large amount from the throat. A second nurse

complained of a sore nose and was found to have acquired Streptococcus pyogenes in small amount in the throat. Finally, on one occasion, when a nurse had developed septic spots on her face, it was found that Staphylococcus aureus had been acquired in the nose. Three other members of the staff had coryza when they were swabbed but in none was there any change in the bacteriological flora of the nose and throat.

It therefore appears that the acquisition of these organisms was not usually associated with any clinical manifestation of illness. The acquisition of pneumococci of the higher types was not associated with the development of respiratory illness.

(d) The sites of isolation of the organisms.

Table 16 summarises the sites of isolation of the various organisms. The organisms behaved in similar fashion to those isolated from the patients and again, the pneumococcus favoured the posterior and Staphylococcus aureus the anterior nose whereas, Streptococcus pyogenes favoured the throat. If however, Streptococcus pyogenes was present in considerable quantity, the posterior nose was as often favoured as the throat. (Table 17).

On many occasions, an organism was isolated from more than one site. For both the pneumococcus and Staphylococcus aureus, the most frequent combination of sites was the

TABLE 16.

Showing the number of isolations of the various organisms from the various sites.

(a) 1st Swabbing results:

Site of Isolation	Pneumo.		S.pyog.		S.aur.	
	No.	%	No.	%	No.	%
P.N. only	5	55.6	3	37.5	3	16.7
A.N. only	2	22.2	0	0.0	8	44.4
T. only	0	0.0	5.	62.5	0.	0.0
P.N. + A.N.	2	22.2	0	0.0	7	38.9
TOTAL:	9	100.0	8	100.0	18	100.0
P.N.	7	63.6	3	37.5	10	40.0
A.N.	4	36.4			15	60.0
T.			5	62.5		
TOTAL:	11	100.0	8	100.0	25	100.0
Isolation rate for 42 staff:	21.4		19.0		42.9	

(b) Results from all swabbings:

Site of Isolation	Pneumo.		S.pyog.		S.aur.	
	No.	%	No.	%	No.	%
P.N. only	39	68.4	10	12.7	13	9.4
A.N. only	3	5.3	4	5.1	56	40.6
T. only	1	1.8	52	65.8	0	0.0
P.N. + A.N.	13	22.8	0	0.0	67	48.6
P.N. + T.			7	8.9	1	0.7
A.N. + T.			2	2.5	0	0.0
P.N. + A.N. + T.	1	1.8	4	5.1	1	0.7
TOTAL:	57	100.1	79	100.1	138	100.0
P.N.	53	73.6	21	21.9	82	39.4
A.N.	17	23.6	10	10.4	124	59.6
T.	2	2.8	65	67.7	2	1.0
TOTAL:	72	100.0	96	100.0	208	100.0

TABLE 17.

Showing the sites of isolation of Streptococcus pyogenes when it occurred in considerable quantity.

Serial No.	Serial No. of Swabbing.	Site of Isolation		
		PN.	AN.	T.
3	3			T++
	6	PN+		T+++
7	1			T++
9	11	PN+		T+
13	2	PN+++		AN+ T+
	3	PN++		
	5	PN+++		T+
	6	PN++		
14	14			T+++
23	1	PN++		
28	3		AN+++	T+
36	1			T++
	4			T+++
	5	PN+++	AN++	T+
38	1			T+++
39	1	PN+++		

\+ = scanty colonies.

++ = moderate number of colonies.

+++ = large number of colonies.

anterior and the posterior nose. A similar number of organisms was usually isolated from each site and if not, the posterior nose was more heavily infected with pneumococci than the anterior nose whereas the converse was true for Staphylococcus aureus. Streptococcus pyogenes was seldom isolated from more than one site. When it was, the nose and throat were usually equally infected but if this were not the case, the posterior nose was more heavily infected than the throat. (Tables 16 and 17).

(e) Comparison of the Findings from the Staff with those from the patients and with those from the general population.

Table 18 summarises the frequency with which the various organisms were isolated from these three groups of individuals. The anterior nose findings for the patients and the members of staff were excluded for the purposes of comparison with the other groups, in whom, only the posterior nose and the throat were swabbed. Only the initial swabbing results from patients and staff are considered.

The child, whether healthy or suffering from an acute respiratory infection was frequently a carrier of pneumococci whereas the adults of the general population and the hospital staff seldom carried this organism.

Streptococcus pyogenes was seldom isolated from the patients and never were more than a few colonies isolated. It was isolated with greater frequency from the members of

TABLE 18.

Comparing the number of patients, staff and members of the "Baby Families" carrying each of the organisms.

Group	Total	Carrying Pneumo.		Carrying S.pyog.		Carrying S.pyog.+++		Carrying S.aur.	
		No.	%	No.	%	No.	%	No.	%
Patients	104	65	62.5	12	11.5	0	0	10	9.6
Staff	42	7	16.6	8	19.0	5	11.9	10	23.8
Baby Families aged 15 years and over.	194	28	14.4	45	23.2	1	0.5	32	16.5
Baby Families aged 1-5 years	111	63	56.8	24	21.6	3	2.7	8	7.2

staff and the members of the general population, of all ages.

Staphylococcus aureus occurred more frequently in the hospital staff than in any of the other groups. It was more frequently isolated than was the pneumococcus, in contrast to the findings for the general population of all ages and for the patients. The isolation rate was higher for the hospital staff than for the adult general population and this suggests that the increased rate was an actual result of work in hospital and it is possible that the staff themselves had been infected from the patients.

(f) Summary of the Findings.

(1) The staff were swabbed with sufficient frequency to ensure an adequate knowledge of the organisms present in the nose and throat of members of the medical, nursing and domestic staff coming into contact with the patients.

(2) A pneumococcus was isolated from 21.4 per cent, Streptococcus pyogenes from 19.0 per cent and Staphylococcus aureus from 42.9 per cent. The types of pneumococci isolated were in no way different to those found in the general population.

(3) A considerable number of the staphylococci isolated were coagulase positive and penicillin resistant.

(4) The organisms seemed to favour particular sites in the nose or throat and did so in similar fashion to that found for the patients.

(5) All three organisms were frequently acquired by the staff during the investigation.

(6) The high isolation rate of Staphylococcus aureus from the staff as compared with the adult general population suggests that the association with hospital patients was responsible.

* * * * * *

Chapter 3.

Dust Sampling.

* * * * *

(a) Introduction.

An essential part of this investigation was the examination of the floor dust for the organisms in question. Dust was collected on blood agar plates as described in the opening section and the plates incubated overnight. The plates were examined by a low power lens and the presence of any colonies resembling those of Streptococcus pyogenes or of Staphylococcus aureus was noted. Colonies resembling those of either the pneumococcus or streptococcus viridans were subcultured. If optochin sensitive, the organism was typed, directly or after mouse inoculation of the culture.

The portable dust sampler was that used by Williams (1949) in his investigations on the persistence of Streptococcus pyogenes in floor dust. Most of his samples were, hover, collected by sweeping or by the thimble sampler in order that counts of the organisms in the dust could be made. Only by taking large samples of dust and making counts of the organisms present, can a true estimate of the degree of contamination of the dust be made, but when all that is required is a knowledge of whether or not an organism is present in the dust, the portable dust sampler is adequate.

A series of investigations were done by Lidwell and Lowbury (1950) on the question of survival of bacteria in dust. Lowbury (1950) states that the persistence of

pathogens in the dust cannot be estimated withouttaking into account the viability of the organisms themselves and the rate of turnover of their vechicle. Both writers were particularly interested in the survival of a) the total organisms, b) Streptococcus pyogenes and c) Staphylococcus aureus. The last mentioned organism was found only in occupied rooms and was considered to be an indication of human contamination of the dust.

According to Lidwell and Lowbury, the death rate of the organisms was affected by the humidity of the atmosphere but this must only be considered in conjunction with the effect of light and of heat. The death rate increases with the relative humidity and it can be presumed that the bacteria in the dust, which have survived a drying process, are more liable to damage by substances in solution as the humidity increases. Ultraviolet light not only kills off organisms in the air, dispersed as droplet nuclei, but it also has a lethal effect on bacteria in the dust. Garrod (1944a) showed that there were a smaller number of streptococci in the dust of well, naturally lighted rooms than in those which were poorly lighted. He found that dust near a window was almost sterile. This effect of ultra-violet light was slower in dry conditions and increased with the relative humidity (Lidwell and Lowbury, 1950).

Dunklin and Puck (1948) observed the lethal effect of

humidity, not on organisms in the dust, but on organisms sprayed into the atmosphere. Using a type 1 pneumococcus in a broth, saliva or .5 per cent saline solution, they found a critical mortality in the vicinity of R.H.50 per cent. (In dust there is no critical mortality.) Above and below 50 per cent, the lethal effect decreased. The lethal effect also increased with a rise in temperature. Similar general survival patterns were found with cultures of streptococci and of staphylococci, but the mortality rates were considerably smaller. The above findings have a special bearing on the writer's investigation as it must be presumed that before reaching the dust the organisms have been expelled from the nose or throat of an individual in a saliva suspension.

Finally although it is known that the pneumococcus is a delicate organism and has been shown in the previous paragraph to have a much higher mortality rate than Streptococcus pyogenes and Staphylococcus aureus, it has been shown to persist in the dust of a ward. As long ago as 1917, Stillman found type 1 and type 2 pneumococci in the floor dust of a ward used only for the treatment of patients suffering from pneumonia.

(b) <u>The Isolation of Pneumococci from the Dust.</u>

During the investigation in the ward used for the treatment of children suffering from pneumonia, a total of

275 dust plates was taken. Rather unexpectedly, considering the large number of patients in the ward who carried pneumococci, only 8 (2.9%) of the plates contained pneumococci. An even smaller number of positive plates was obtained from the smaller ward, used for patients suffering from measles. From this ward, 114 plates were examined. Only 2 had pneumococcal colonies (1.75%) but one must remember that these plates were taken in the summer months when the pneumococcus would have less chance of survival in the dust on account of the increased ultra-violet radiation. (Garrod, 1944a). Furthermore, the number of pneumococcal carriers in the ward was much less. In contrast to this are the findings in the small ward where patients suffering from measles were overcrowded. In this ward a total of 46 plates was taken and 5 contained pneumococci (10.9%). The patients however were definitely crowded and the majority carried pneumococci.

Note: The ward used for the treatment of patients suffering from pneumonia will be referred to as "Ward I".

The ward used for the treatment of patients suffering from measles will be referred to as "Ward 2".

The small ward where patients suffering from measles were crowded will be referred to as "Ward 3".

The types of pneumococci found in the dust plates were determined and Table 19 summarises the findings for Ward I and the possibilities of any particular patient being the

TABLE 19.

Ward I.

To relate the time and place of isolation of a pneumococcus from the dust with the position in the ward of patients carrying the same type as was present in the dust.

Type of Pneumo.	Position of Isolation.	Date of Isolation.	REMARKS
33	Cot AI.	21.11.'50.	No patient or member of staff carried type 33 at this time. On 24.11.50 patient in Cot A5 acquired Pneumo. type 33.
23	Cot C13.	29.11.'50.	No member of staff or patient in Division C carried type 23. Carriers of type 23 present in Divisions B and D.
19	Cot B10.	7.12.'49.	On 6.12.49, patient in Cot B10 acquired Pneumo.type 19. No member of staff carried type 19 but two other patients in Division B did so.
Un-known	Cot B10.	14.12.'49.	Patient in Cot B10 carried type 19.
19	Cot D2.	14.12.'49.	Patient in Cot DI carried type 19. No member of staff carried type 19 but patients in Division B carried it.
2	Cot C8.	28.12.'49.	Patient in Cot C8 carried type 2. No member of staff carried type 2.
2	Cot C7.	28.12.'49.	
33	Cot B1.	23. 3.'50.	Patient in Cot BI carried type 33. No member of staff carried type 33.

source of the pneumococcus in the dust or being infected by the pneumococcus in the dust. In none of the instances when a pneumococcus was isolated from the dust, could a member of staff have been the source of infection.

In 4 of the 8 instances, the patient in either the cot associated with the dust sample or in the adjacent cot carried the homologous type. In another instance, 2 patients in the same division carried the homologous type and the patient in the cot associated with the positive dust sample had acquired the type on the day prior to the taking of the dust sample. In yet another instance, a patient in the same division acquired a pneumococcus of the same type as was present in the dust, three days after its isolation from the dust. In the remaining two instances, the source could not be determined since in one, the type was unknown and in the other, any carriers of the homologous type were in other divisions of the ward.

The preceding paragraph shows that there was definite evidence that a) the dust of the ward could be infected with pneumococci from the patients and b) a patient could himself be infected with a pneumococcus known to be present in the dust of the ward. The results from the smaller Wards, 2 and 3, which will now be discussed, confirmed this assumption.

Only type 6 pneumococci were isolated from the dust of Wards 2 and 3. In the small, overcrowded Ward 3, type 6 was

the most prevalent type amongst the patients and on each occasion when the dust contained a type 6 pneumococcus, three of the six patients in the ward, carried it. During this investigation, which lasted seven weeks, 7 of a total of 18 patients were admitted carrying a type 6 pneumococcus and a further 4, acquired it after admission.

In Ward 2, there were only 2 positive dust plates. On both occasions a type 6 pneumococcus was carried by patients in cots near to where the dust was sampled. As was previously stated, the pneumococcal isolation rate was low for the patients suffering from measles but type 6 was the type most frequently carried and acquired by them. Type 6 was acquired 5 times and in 4, this occurred within a few days of the positive dust samples being obtained.

Four patients, also suffering from measles, who were nursed in a cubicle of a ward, were the subjects of yet another subsidiary investigation. These patients were swabbed every second day and the dust was sampled daily. One of these patients acquired a type 5 pneumococcus on her third day in hospital and carried it continuously thereafter. This pneumococcus was isolated from the dust on two occasions, 2 and 6 days respectively after the organism was acquired. No other patient acquired the pneumococcus after it was isolated from the dust. This is yet another example of a patient infecting the ward dust with a pneumococcus.

NOTE: THE INVESTIGATIONS IN WARD 2 AND WARD 3 EACH LASTED 7 WEEKS.
THE INVESTIGATION ON THE 4 PATIENTS IN A CUBICLE OF A WARD LASTED 3 WEEKS.

TABLE 20.

To relate the occurrence of Streptococcus pyogenes in the dust of a division with the number of patients carrying or acquiring the organism for the first time in that division.

Week	DIVISION A			DIVISION B			DIVISION C			DIVISION D		
	Pat - ients Car.	Pat - ients Acq.	Dust	Pat - ients Car.	Pat - ients Acq.	Dust	Pat - ients Car.	Pat - ients Acq.	Dust	Pat - ients Car.	Pat - ients Acq.	Dust
9						1	1					
						Sca.Fever						
10	1		3		1	1				1		
11					1	4		2			1	
12	3	2	1	5	4	1						
13	4	1		2				1	1			
14	4	1	1					2	1			
15	1					1	1					
16	1					1	4	2				
17	1								1			
18	2	1	1	1								
19	2		1				3	2				

(c) Isolation of Streptococcus pyogenes from the dust.

Streptococcus pyogenes was more frequently isolated from the dust plates than was the pneumococcus. In Ward I, 19 of the dust plates (6.7%) contained colonies of Streptococcus pyogenes. In Ward 2, 4 of the plates (3.5%) contained Streptococcus pyogenes and although so few contained the organism, the number was at least greater than for the pneumococcus.

Streptococcus pyogenes was isolated from the dust for the first time in Week 9 and thereafter was isolated each week until Week 19. During these weeks there was an increase in the number of patients and members of staff acquiring the organism and one of the patients developed scarlet fever in Week 10. It is important to notice that, before the patient developed scarlet fever, Streptococcus pyogenes was isolated from the dust, both in Week 9 and in Week 10.

Table 20 summarises the findings in relation to the divisions of the ward from which the dust was obtained and in which the streptococcal carriers were. Streptococci were first isolated from the dust in close proximity to the cot of the patient in division B who later developed scarlet fever. Thereafter streptococci appeared in the dust of both divisions A and B. This occurred prior to the increase in the number of carriers of the organism in these

TABLE 21.

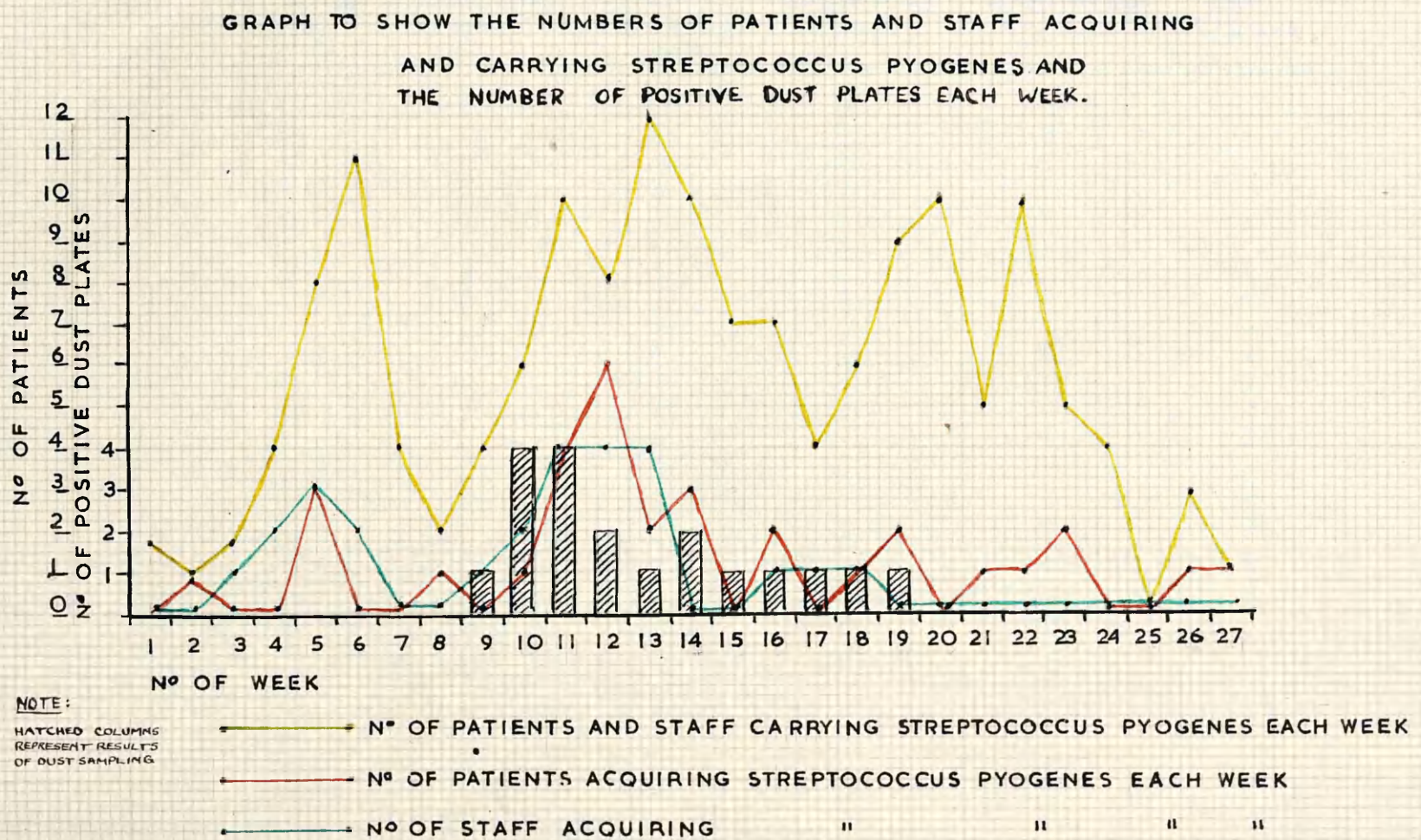
Ward I.

To show for each week the numbers of patients and members of staff carrying or acquiring for the first time, Streptococcus pyogenes. The number of positive dust plates is shown for each week.

Week	Patients carrying S.pyog.	Staff carrying S.pyog.	Patients + Staff carrying S.pyog.	Patients acquiring S.pyog.	Staff acquiring S.pyog.	Positive Dust Plates.
1	0	2	2	0	0	
2	1	0	1	1	0	
3	0	2	2	0	1	
4	0	4	4	0	2	
5	3	5	8	3	3	
6	6	5	11	0	2	
7	3	1	4	0	0	
8	1	1	2	1	0	
9	1	3	4	0	1	1
10	3	3	6	1	2	4
11	4	6	10	4	4	4
12	8	0	8	6	0	2
13	5	7	12	2	4	1
14	6	4	10	3	0	2
15	2	5	7	0	0	1
16	4	3	7	2	1	1
17	1	3	4	0	1	1
18	3	3	6	1	1	1
19	5	4	9	2	0	1
20	5	5	10	0	0	
21	2	3	5	1	0	
22	5	5	10	1	0	
23	3	2	5	2	0	
24	1	3	4	0	0	
25	0	0	0	0	0	
26	3	0	3	1	0	
27	1	0	1	1	0	

Figure 2.

Isolation of Streptococcus pyogenes from the dust.



divisions. The dust of division C was later still in being infected and this was associated with several patients acquiring the organism. The dust of division D was never infected and there were no carriers of Streptococcus pyogenes in this division.

The source of the original streptococcal infection and the resulting infection of other patients is discussed in Section 3. The importance of the ward dust as a medium for infection is emphasised because of two findings a) the presence in the ward floor dust of Streptococcus pyogenes before the patient developed scarlet fever and before other patients began to acquire Streptococcus pyogenes and b) the occurrence of Streptococcus pyogenes in the dust associated simultaneously with a considerable increase in the number of new carriers in the ward, both amongst the patients and the members of staff.

Table 21 summarises the findings and Figure 2 depicts it graphically.

In Ward 2 only 4 dust plates contained Streptococcus pyogenes. One might have expected a higher incidence since 42 per cent of the patients carried Streptococcus pyogenes at least once during their stay in hospital. The corresponding percentage for the patients in Ward I was 36.9 per cent.

The small number of positive plates in Ward 2 as compared with Ward I, could be explained as was the small

TABLE 22.

Ward 2.

To show for each week the numbers of patients and members of staff carrying and acquiring for the first time, Streptococcus pyogenes. The number of positive dust plates is shown for each week.

Week	Patients carrying S.pyog.	Staff carrying S.pyog.	Patients + Staff carrying S.pyog.	Patients acquiring S.pyog.	Staff acquiring S.pyog.	Positive Dust Plates.
1	1	1	2	0	0	1
2	5	3	8	0	1	
3	1	3	4	0	0	
4	5	3	8	1	1	
5	6	3	9	5	0	1
6	4	0	4	1	0	1
7	3	0	3	0	0	1

TABLE 23.

Ward 2.

To relate the site of occurrence of the positive dust plates with the position in the ward of patients carrying Streptococcus pyogenes.

Number of plate.	Week	Site of Occurrence.	Staff carrying S.pyog.	Patients carrying S.pyog.	Patients acquiring S.pyog.
1	1	5A	1	Patient in Cot 2A (admitted 6 days prior to the taking of the dust sample)	0
2	5	3A	3	Patient in Cot IB (admitted befor the other patients acquired S.pyog and 5 days prior to the taking of the dust sample).	Patients in Cots 3A, 4A, 5A, 4B and 5B.
3	6	5C	0	Patient in Cot 6C (admitted 2 days prior to the taking of the dust sample)	Patient in Cot 4C acquired S.pyog ++ the day after the dust sample was taken.
4	7	3C	0	Patients in Cots 5A, 2B and 6B (carried S.pyog prior to the taking of the dust sample).	0

Note: the divisions in this ward were named A, B and C. Each accommodated six patients.

number positive for pneumococci, by the inhibitory action of the increased ultra-violet radiation in the summer months. Moreover, in Ward 2, no scarlet fever incident occurred to increase the streptococcal content of the dust.

The four plates containing Streptococcus pyogenes were obtained in Weeks 1, 5, 6 and 7 of the investigation in Ward 2. Table 22 summarises the numbers of patients and members of staff carrying and acquiring Streptococcus pyogenes each week, and Table 23 relates the site of isolation of the streptococci from the dust with the position in the ward of the patients who carried streptococci.

Two of the positive dust plates (Nos. 2 and 3) occurred prior to patients in nearby cots acquiring the organism. In one of these (No.3) it was likely that the dust was infected by yet another patient in a nearby cot. A patient carrier might also have been the source of the streptococci in plate No.1 but there was no such source for those in plate No.4. In this instance, the organism may have persisted from the previous week.

Such findings as the above are similar to those for Ward I and provide further evidence of the importance of dust as a means of cross infection.

TABLE 24.

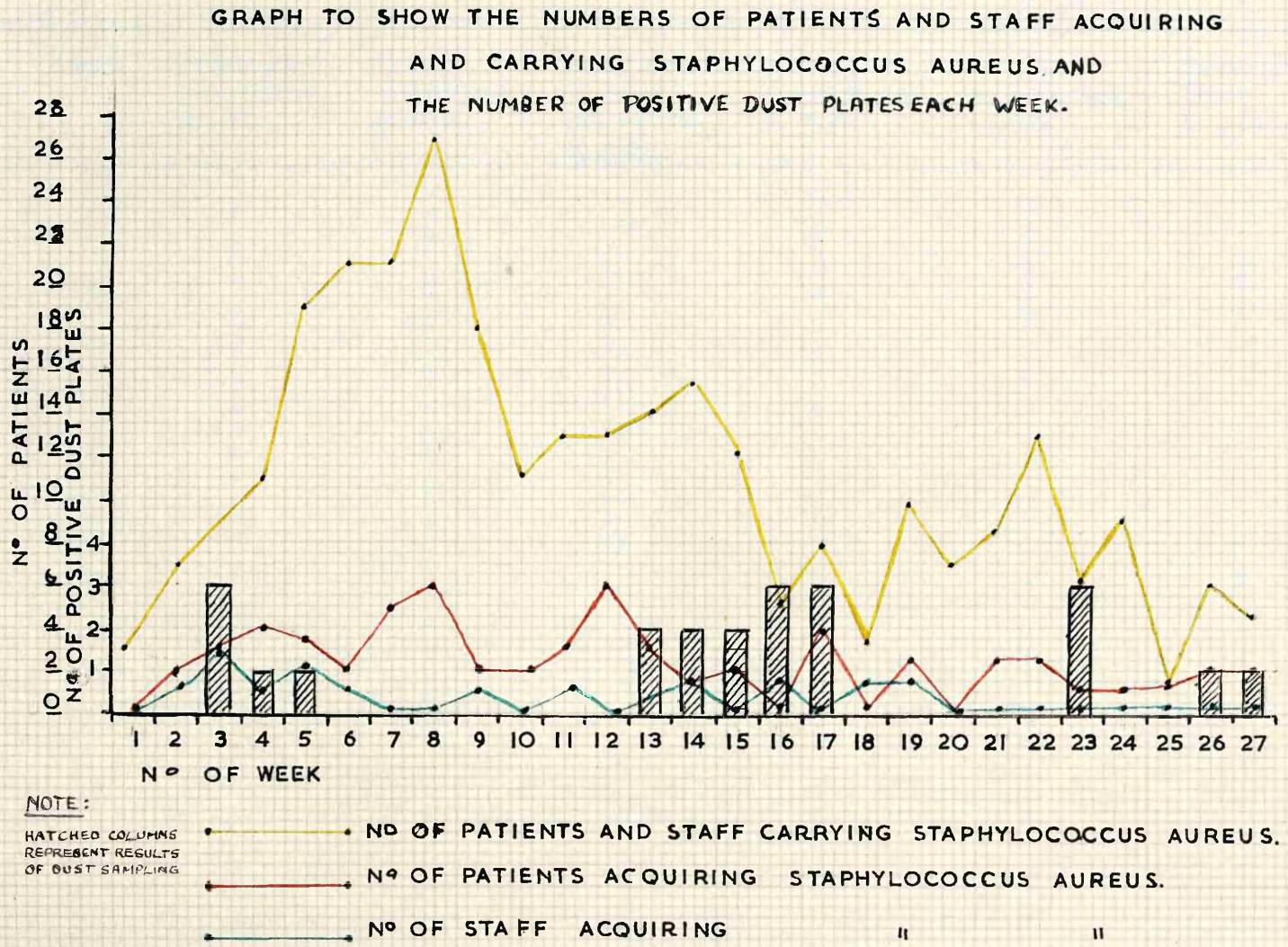
Ward I.

To show for each week the numbers of patients and members of staff, carrying and acquiring for the first time, Staphylococcus aureus. The number of positive dust plates is shown for each week.

Week	Patients carrying S.aur.	Staff carrying S.aur.	Patients + Staff carrying S.aur.	Patients acquiring S.aur.	Staff acquiring S.aur.	Positive Dust Plates.
1	1	2	3	0	0	
2	2	5	7	2	1	
3	4	7	11	3	3	3
4	7	6	13	4	1	1
5	8	11	19	3	2	1
6	10	11	21	2	1	
7	13	8	21	5	0	
8	21	6	27	6	0	
9	9	9	18	2	1	
10	5	5	10	2	0	
11	7	6	13	3	1	
12	12	1	13	6	0	
13	7	7	14	3	0	2
14	7	8	15	1	1	2
15	7	5	12	2	0	2
16	0	5	5	0	1	3
17	5	3	8	4	0	3
18	2	2	4	0	1	
19	4	6	10	3	1	
20	2	5	7	0	0	
21	6	3	9	3	0	
22	5	8	13	3	0	
23	2	4	6	1	0	3
24	4	5	9	1	0	
25	1	0	1	1	0	
26	6	0	6	2	0	1
27	5	0	5	2	0	1

Figure 3.

Isolation of Staphylococcus aureus from the dust.



(d) Isolation of Staphylococcus aureus from the dust.

In Ward I, 22 dust plates contained colonies of Staphylococcus aureus (8% of all plates). Considering the large number of patients and staff who carried this organism an even greater number might have been expected.

The positive dust plates occurred at the beginning, towards the middle and at the end of the experiment with several weeks intervening when no staphylococci were isolated from the dust. In contrast to the findings for Streptococcus pyogenes there was no rise in the number of individuals carrying and acquiring staphylococci in the weeks when the dust contained the organism. The carrier rate remained high at all times and on each occasion that a positive dust plate was obtained, there were several patients and members of staff carrying Staphylococcus aureus. Table 24 summarises the findings and it is of interest to notice that in the period between Week 6 and Week 12, there were no positive dust plates, yet in Week 8, 21 patients and 6 members of staff carried the organism. Furthermore, the positive dust plates did not always occur in divisions where there were carriers of Staphylococcus aureus. Figure 3 depicts the findings graphically.

The isolation of Staphylococcus aureus from the dust did not mirror to any extent the number of carriers of the organism in the ward and in this respect it differed from Streptococcus pyogenes. Its isolation from the dust

TABLE 25.

Ward 2.

To show for each week the numbers of patients and members of staff carrying and acquiring, for the first time, Staphylococcus aureus. The number of positive dust plates is also shown each week.

Week	Patients carrying S.aur.	Staff carrying S.aur.	Patients + Staff carrying S.aur.	Patients acquiring S.aur.	Staff acquiring S.aur.	Positive Dust Plates.
1	0	5	5	0	0	3
2	3	5	8	2	0	1
3	6	4	10	3	0	2
4	12	7	19	5	0	0
5	14	8	22	7	1	5
6	12	4	16	5	0	0
7	6	1	7	1	0	11

TABLE 26.

Ward 2.

Table to relate the division where the positive dust sample was obtained with the presence in that division of patients carrying or acquiring Staphylococcus aureus.

Week	Number of positive dust plates.	Division where positive dust plates occurred.	Staff carrying S. aur.	Patients carrying S. aur.	Patients acquiring S. aur.
1	3	A	5	0	0
2	1	A	5	1 in division A.	2 in division A.
3	2	C	4	3 in division C.	1 in division A. 2 in division B.
4	0	0	7	7	5
5	5	A (3) B (I) C (I)	8	1 in division A. 2 in division B. 4 in division C.	3 in division A. 3 in division B. 1 in division C.
6	0	0	4	7	5
7	11	A (2) B (5) C (4)	1 *	5 in division B.	1 in division C.

* newly arrived in ward. Carrier of S.aur. in large amount.

would thus seem to be more haphazard than that of Streptococcus pyogenes.

The findings for Ward 2 can be more easily analysed because of the smaller nature of the experiment. The writer found that 24 of the 114 dust plates (21.1%) contained colonies of Staphylococcus aureus - a considerably higher percentage than was found in Ward I. Of these plates, 11 were obtained in the last week of the experiment and it was to this week that the higher percentage was due.

Table 25 summarises the findings. The two positive dust plates which were obtained before the first patient was admitted are not mentioned in the table.

The occurrence of Staphylococcus aureus in the dust was not particularly associated with the presence in the ward of a large number of carriers, whether patients or members of staff, and only in one week was it associated with a large number of individuals acquiring the organism for the first time.

Table 26 shows that, whereas in most weeks it was most likely that the staff infected the dust, in some the patients might as easily have been the source of the infection. It is possible that, in Weeks 2, 5 and 7 patients were actually infected by staphylococci in the ward dust.

On consideration of the findings both for Ward I and

Ward 2, it seems likely that infection of the ward dust by Staphylococcus aureus arose from the carriers of the organism amongst the staff rather than from those amongst the patients. There was only slight evidence that the patients actually acquired the organism through the medium of the dust.

(e) Summary of Findings.

(1) Staphylococcus aureus was the organism most frequently isolated from the dust. Streptococcus pyogenes was next in order of frequency of isolation and the pneumococcus was least often isolated.

(2) The number of isolations of pneumococci from the dust was increased in a small ward where the patients were intentionally overcrowded.

(3) The number of isolations of pneumococci and streptococci from the dust was less in the ward used for the treatment of patients suffering from measles than in the main ward, used for the treatment of patients suffering from pneumonia. It is possible that this was due to the warmth and sunlight of the summer months when the former investigation took place.

(4) There was evidence to show that patients infected the dust with pneumococci and that other patients were in turn infected by the pneumococci present in the dust. There was no evidence of any member of staff infecting the dust.

(5) Streptococcus pyogenes was isolated from the dust before the scarlet fever incident occurred and prior to several patients and members of staff acquiring Streptococcus pyogenes.

(6) The number of positive dust plates for Streptococcus pyogenes mirrored the number of carriers of the organism in the ward. There was considerable evidence that infection of the patients and staff occurred through the medium of the dust.

(7) The number of plates containing Staphylococcus aureus did not correspond to the number of carriers in the ward. The organism was present in the dust before any patients were admitted to the ward used for patients suffering from measles.

(8) There was little evidence to suggest that patients were infected with Staphylococcus aureus from the dust. The dust could have become infected from either patients or members of staff, the latter being the more probable source.

* * * * *

SECTION 2.

Chapter 4.

It was by no means the primary object of this investigation to determine the effects of the antibiotics on the bacteriological flora of the nose and throat. However, in any study, such as this, which deals with cross infection and in which the patients have, of necessity, to be treated by antibiotics, one must be fully aware of any changes due to chemotherapy and not the result of their being in hospital. Furthermore, one must determine if the administration of antibiotics did indeed lessen the incidence of cross infection in the ward.

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Chapter 4.

Effects of Chemotherapy.

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(a) The Antibiotics used in the treatment of the patients.

Patients who, on admission, were presumed to have an acute respiratory illness were treated by one or other of the following chemotherapeutic agents; oral sulphadiazine, oral penicillin, or intramuscular penicillin. Unless the patient's illness would have been adversely affected the drug administered to each patient was determined solely by the order of the patient's admission. It was hoped that in this way three groups would be obtained, each containing an approximately equal number of patients. Subsequently comparison could be made between the action of each of the three above-mentioned drugs on the flora of the nose and throat.

If treatment seemed to be unsuitable, or response to treatment inadequate, some other antibiotic or combination of antibiotics was given. The severely ill child was given both sulphadiazine and intramuscular penicillin. Conditions other than an acute respiratory infection, present either on admission or developing later, were treated by the antibiotics indicated. If the antibiotic used for these conditions was intramuscular penicillin, the findings were included with those from the group of patients suffering from an acute respiratory illness who were treated by intramuscular penicillin. Thus this group is a little larger than the other two main groups.

Table 27 summarises the number of patients receiving each antibiotic or combination of antibiotics.

TABLE 27.

Showing the number of patients receiving each antibiotic or combination of antibiotics.

Treatment.	No. of Patients.	
Sulphadiazine	34	
Penicillin Tablets (Oral)	35	
Intramuscular Penicillin	39	42
Intramuscular Penicillin (subsequent to a small dose of penicillin tablets).	3	
Intramuscular Penicillin + Sulphadiazine	11	13
Intramuscular Penicillin + Sulphadiazine (subsequent to a small dose of penicillin tablets).	2	
Intramuscular Penicillin + Sulphadiazine + Streptomycin.	2	
Aureomycin	1	
Streptomycin	2	
No treatment	10	

Ten patients received no chemotherapy either because they did not require it or had been given adequate treatment before admission. For the purposes of completeness there have also been included in the table one patient with septic lesions treated by aureomycin and two tuberculous patients treated by streptomycin.

A number of patients were in more than one treatment group due to:-

(1) inadequate response to the initial treatment, or

(2) the subsequent establishment of a diagnosis requiring a different form of treatment, or

(3) the development of a condition distinct from the original illness.

Dosage of Drugs:

The dose of sulphadiazine given varied with the age of the patient. Thus, 0.125 gm., 0.25 gm. or 0.5 gm. were given every four hours for what remained of the admission day and for six full days thereafter. The totals for a course of chemotherapy were approximately 4.750 G., 10.5 gm. and 21 gm. Using oral tablets of penicillin, containing 20,000 units each, 40,000 units were given every four hours. Later in the investigation, 60,000 units were given every four hours. The total dosage was therefore approximately 1,680,000 units and later 2,520,00 units. Intramuscular penicillin in a dosage of 50,000 units was given every four hours to a total of approximately 2,100,000 units. A higher dose was given to some very ill patients and one of the cases of staphylococcal empyema had a total of 45,000,000 units, while in hospital. When sulphadiazine was given with penicillin, the usual dosage of the drug was given along with penicillin 50,000 or 250,000 units every four hours.

TABLE 28 (a).

Summarising the behaviour of the various organisms:- for all forms of therapy combined.

Organism.	Isolated prior to therapy.	Acquired during therapy.	Isolated prior to and after therapy.		Isolated prior to therapy but not after.			
					Not recurring		Recurring	
	No.	No.	No.	%	No.	%	No.	%
Pneumo-coccus.	81	21	37	45.7	39	48.1	10	12.3
Strepto-coccus pyogenes.	22	6	3	13.6	16	72.7	3	13.6
Staphylo-coccus aureus.	25	41	7	28.0	15	60.0	3	12.0

TABLE 28 (b).

Summarising the behaviour of each organism:- detailed for each form of treatment given.

Organism.	Treatment	Isolated prior to therapy. No.	Acquired during therapy. No.	Isolated prior to and after therapy. No.	%	Isolated prior to therapy but not after. Not recurring No.	%	Recurring No.	%
Pneumo-coccus.	Sulph.	19	6	10	52.6	7	36.8	2	10.5
	Oral pen.	25	8	11	44.0	9	36.0	6	24.0
	I.M.pen.	29	7	14	48.3	17	58.6	1	3.5
	Sulph.+I.M.pen.	8	0	2	25.0	6	75.0	1	12.5
Strepto-coccus pyogenes.	Sulph.	2	1	1	50.0	1	50.0	0	0
	Oral pen.	9	3	1	11.1	8	88.9	0	0
	I.M.pen.	9	2	0	0	6	66.7	3	7.1
	Sulph.+I.M.pen.	2	0	1	50.0	1	50.0	0	0
Staphy-lococcus aureus.	Sulph.	4	18	1	25.0	3	75.0	0	0
	Oral pen.	5	7	2	40.0	3	60.0	0	0
	I.M.pen.	13	13	4	28.6	6	46.2	3	23.1
	Sulph.+I.M.pen.	3	3	0	0	3	100.0	0	0

Abbreviations:
Sulph. = Sulphadiazine.
Oral pen. = Oral penicillin tablets.
I.M.pen. = Intramuscular penicillin.

(b) Changes found in the bacteriological flora of the nose and throat after administration of antibiotics:

Only in a proportion of patients did treatment by the antibiotics bring about any change in the bacteriological flora present in the nose or throat. Moreover, there was no uniformity in the behaviour of the various organisms to the various treatment routines.

Streptococcus pyogenes was the organism most often eliminated as a result of treatment. Of patients carrying Streptococcus pyogenes at the commencement of treatment, 72.7 per cent no longer carried it by the time treatment was completed. A pneumococcus was lost by 48.1 per cent and Staphylococcus aureus by 60 per cent of patients receiving treatment. The findings are summarised in Tables 28a and b.

i). The Pneumococcus: With the exception of the small group treated by combined sulphadiazine and intramuscular penicillin, approximately half the patients in each treatment group carried the same type of pneumococcus before and after the completion of the course of treatment. Furthermore, despite treatment fresh penumococcal types were acquired by approximately a fifth of the patients receiving each antibiotic (16.7% of those receiving intramuscular, and 22.9% of those receiving oral penicillin; 17.6% of those receiving sulphadiazine).

A number of the patients, however, no longer carried a pneumococcus by the time treatment was completed and in most of these it remained absent during the remainder of the patient's stay in hospital. The precentage losing a pneumococcus was similar for the sulphadiazine and the oral penicillin groups (36.8% and 36% respectively), whereas a larger percentage (58.6%) of patients in the intramuscular penicillin group lost their pneumococci. Caution is required in interpreting the figures because of the small numbers of patients involved, but the difference is statistically significant. Very few patients were treated by combined sulphadiazine and penicillin therapy and the results for these patients were therefore of little value.

When particular attention was paid to the nature of the respiratory illness suffered by the patients, i.e. bronchitis or pneumonia, it was found that this had no effect on the behaviour of the pneumococci in the nose and throat as a result of treatment.

Table No.29 summarises the types of pneumococci eliminated from the nose and throat as a result of treatment. All type 19 pneumococci were eliminated, whereas most of type 6 and type 23 pneumococci were still present at the end of treatment. Types 1, 21, 11 and 22 were quite frequently eliminated. It is an interesting fact that type 19 was universally eliminated as a result of treatment. It may be a chance occurrence or the direct

TABLE NO.29.

Showing the types of penumococci eliminated by therapy.

Pneumococcal Type.	Pneumococci present prior to therapy.	Pneumococci eliminated by therapy.	
	No.	No.	%
23	15	4	26.7
6	14	6	42.9
19	7	7	100.0
1	5	3	60.0
14	5	1	20.0
21	5	3	60.0
11	4	3	75.0
7	3	1	33.0
22	3	3	100.0
20	2	1	50.0
18	2	2	100.0
8	2	1	50.0
10:31:Gr.C:U.T.T.	1	1	100.0

result of the intramuscular penicillin which was given to five of the seven patients who lost this type.

Lastly, some patients who carried two types lost one type, whereas the second type persisted despite treatment.

ii). Streptococcus pyogenes:

Unfortunately the number of patients who carried Streptococcus pyogenes in each treatment group was small and the only two which were comparable were the oral and the intramuscular penicillin groups.

As was found with the pneumococcus, treatment did not prevent the acquisition of Streptococcus pyogenes. Although the number of patients acquiring the organism during treatment was small, (6 in number), three of these patients acquired the organism in large numbers. There was one patient in each of the three main treatment groups.

Only 13.6 per cent of the patients still carried Streptococcus pyogenes at the end of the course of treatment, and 72.7 per cent no longer did so. In a further 13.6 per cent the organism was temporarily lost but was again isolated prior to the patient's dismissal. Both oral and intramuscular penicillin in this small number of patients seemed equally effective in clearing the nose and throat of Streptococcus pyogenes.

iii). Staphylococcus aureus:

It so happened that most of the patients carrying Staphylococcus aureus at the commencement of treatment, were

given intramuscular penicillin, and thus comparison between the treatment groups was not practicable. At the end of treatment 28 per cent still carried the organism whereas 60 per cent no longer did so, the remaining 12 per cent lost the organism only temporarily and it recurred while the patient was still in hospital. Taking the intramuscular penicillin group alone Staphylococcus aureus was eliminated from the nose and throat of 46.2 per cent of the patients.

Despite treatment many patients acquired Staphylococcus aureus. Frequently the organism was acquired in large numbers. A difference of some importance was found in the percentage of patients in each treatment group who acquired the organism. Of patients receiving sulphadiazine 52.9 per cent acquired the organism, whereas 20 per cent of those receiving oral and 31 per cent of those receiving intramuscular penicillin acquired the organism.

Coagulase and penicillin sensitivity tests were done on a proportion of the staphylococci isolated from the patients. Staphylococci isolated before and after treatment with penicillin were examined from four of a possible six patients. All had sensitive organisms prior to treatment, and in two the organism remained sensitive, but in the other two the organism isolated after the completion of treatment was penicillin resistant. Acquired staphylococci from 21 patients were examined. Of these patients,four carried coagulase negative and 17 coagulase positive

organisms. Of the latter organisms, 13 were penicillin resistant, two were penicillin sensitive, and in one both sensitive and resistant strains were present. The remaining organism was not tested for sensitivity to penicillin.

There was no tendency for the resistant organism to be especially acquired by patients being treated by penicillin.

(c) Discussion.

It was apparent from the foregoing that in a percentage of patients the administration of the antibiotics, penicillin and sulphadiazine, did bring about the elimination of organisms from the nose and throat. This effect was most marked in the case of Streptococcus pyogenes, and the antibiotic bringing about most change was intramuscular penicillin.

The persistence of organisms in the nose and throat of some patients throughout a course of chemotherapy was no reflection on the results of treatment because clinically all patients responded well to the drugs given. The most probable explanation of this was that penicillin and the sulphonamides were excreted in greater concentration into the actual lung tissue than into the upper air passages.

Changes are known to occur in the bacteriological flora of the mouth when penicillin is given systemically in sufficient dosage (Long, 1946A, 1946B, 1947A). This

change is due to the excretion of penicillin in the saliva and Long (1947B) showed that the threshold for penicillin excretion in the saliva is attained only by doses of 500,000 units and more in the 24 hours. The use of these big doses of penicillin brings about the elimination of penicillin sensitive organisms from the surface of the tonsil and from the nose and pharynx as well as from the mouth. Considering the dosage used by the writer it is little wonder that the changes in the nose and throat were not constant. Furthermore, it was not known whether all pneumococci, streptococci and staphylococci present prior to treatment were in fact penicillin sensitive.

Administration of penicillin locally also brings about changes in the bacteriological flora of the mouth. (Garrod 1944B; MacGregor and Long 1944). Long,(1947B)found that an elimination of penicillin sensitive organisms similar to that mentioned above when intramuscular penicillin was used, occurred when oral penicillin was administered. This time the effect was mainly on the flora of the mouth and there was little effect on the flora of the nose, the tonsils or the pharynx. Moreover, the effect was not maintained throughout the 24 hours without the conscious co-operation of the patient in sucking their pastilles regularly. In the case of both oral and intramuscular penicillin the initial loss of penicillin sensitive organisms was followed by a replacement by organisms of

the Bacterium group. When treatment was discontinued these bacteria gradually decreased in numbers and the normal flora re-established itself.

Similar difference in the excretion into the lungs and into the mouth occurs with the sulphonamides. Landsman et al (1951) showed that sulphonamides had little effect on an acute throat infection, whereas it is well known that the sulphonamides provide adequate therapy for a pneumonic infection. The same authors emphasised the fact that intramuscular penicillin was the treatment of choice in an acute throat infection of streptococcal origin. The writer's findings in this investigation indicate that this might well be the case.

The effect which the administration of the antibiotics has in preventing cross infection must be only of minor importance since many patients acquired pneumococci of other types, streptococci or staphylococci, in the nose or throat while they were receiving one of the antibiotics. It is possible however, that the development of illness due to these acquired organisms may to some extent have been prevented.

It is of interest at this point to consider Table No.30. The members of staff received no chemotherapy and the percentage acquiring a pneumococcus was similar to the percentage of patients so doing. (It is fully realised, however, that the totals involved are very different).

TABLE NO.30.

To show and compare the percentages of patients and staff carrying the various organisms at the first swabbing or acquiring them after the first swabbing.

		Patients	Staff
Pneumococcus	% carrying the organism at the 1st swabbing.	61.8	21.4
	% acquiring the organism after the 1st swabbing.	36.6	38.1
Streptococcus pyogenes	% carrying the organism at the 1st swabbing.	18.3	19.0
	% acquiring the organism after the 1st swabbing.	19.1	45.2
Staphylococcus aureus.	% carrying the organism at the 1st swabbing.	17.6	42.9
	% acquiring the organism after the 1st swabbing.	48.9	13.3
	TOTAL:	131	42

One might well have expected a higher percentage of patients than of staff, since the pneumococcus is such a frequent commensal in the nose and throat of the child. The use of the antibiotics may have to some extent limited the percentage of patients acquiring a pneumococcus, but it more certainly was a limiting factor in the acquisition of Streptococcus pyogenes. Hence the small percentage of patients as compared with that of the staff who acquired the organism.

A much larger percentage of patients than of staff acquired Staphylococcus aureus. Any possible deterrent effect of the antibiotics on the acquisition of this organism would be limited to the occasional instance where a penicillin sensitive organism was acquired by a penicillin treated patient. It was however shown that the majority of the organisms acquired were penicillin resistant. Sulphadiazine could be excluded as a deterrent to acquisition because of its limited action on the staphylococcus.

In conclusion one must refer to the work not only of Long (1947B) but of Haffner, Netter and Rubin (1950), and Lipman, Coss and Boots (1946), who found that the organisms present in the upper respiratory tract were eliminated as a result of treatment by penicillin alone or with the sulphonamides, only to be replaced by organisms, not previously predominant, which were not affected by the drugs

administered. The above writers used greater quantities of antibiotics and found that the replacement was mainly by Bacterium coli and other gram negative organisms, and it is within the realms of possibility that a similar replacement occurred in this investigation. In this instance, however, the replacing organism was the Staphylococcus aureus, usually of a penicillin resistant strain.

(d) Summary of Findings.

1. The pneumococcus in the nose or throat had approximately a 50 per cent chance of surviving a course of chemotherapy. It survived least often when treatment was by intramuscular penicillin.
2. Streptococcus pyogenes was very susceptible to penicillin and most so to oral penicillin.
3. Staphylococcus aureus was almost as susceptible as the pneumococcus to intramuscular penicillin. Its behaviour with other drugs was not determined.
4. All three organisms were acquired by patients undergoing chemotherapy but especially Staphylococcus aureus. Patients receiving treatment by sulphadiazine acquired organisms more often than those receiving either oral or intramuscular penicillin. Many of the acquired strains of staphylococci were coagulase positive and penicillin resistant. It is suggested that this may have been a replacement of the normal flora of the nose and throat

by organisms not affected by the antibiotics used.

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SECTION 3.

SECTION 3.

Evidence of Cross Infection.

Cross infection manifests itself in two ways, clinically and bacteriologically, and there is no better or more simple definition than the following, taken from the recent Medical Research Council memorandum on "The Control of Cross Infection in Hospitals" (1951). "Clinically, cross infection is manifested as respiratory, gastro-intestinal, wound, skin or mucous membrane infection, arising during the course of another disease. Bacteriologically, it implies the acquisition by a patient of pathogenic micro-organisms not present on admission to hospital." Chapter I of this section deals with such clinical evidence of cross infection as was found in the writer's investigation and Chapter 2, with the bacteriological evidence.

For the purposes of this investigation, any relapse in the original respiratory illness was included as a possible clinical manifestation of an acquired respiratory infection. Any sudden elevation of temperature occurring during the period of time in hospital was also considered as a possible clinical manifestation. The occurrence of bacteriological cross infection was ascertained from the examination of the flora of the upper respiratory tract only. Clinical and bacteriological evidence were correlated wherever possible.

Chapter I.

Clinical Evidence of Cross Infection.

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Chapter I.

(a) Evidence of Cross Infection.

During their stay in hospital, 22 of the patients had either a relapse in their clinical condition or developed some clinical feature suggesting the possibility of an acquired infection and therefore the need to investigate it. Three of the patients had two such incidents. On each occasion swabs were taken from the nose and throat and the results were as follows.

In 14 instances, 56 per cent, there was no change, or at least no significant change, in the bacteriological flora of the nose and throat. In only two, had a pneumococcus been acquired whereas in four, Streptococcus pyogenes and in a further five, Staphylococcus aureus had been acquired. (a) The ten patients in whom there was no change in the bacteriological flora, included two patients who had had a relapse in their clinical condition and two who had had an unexplained elevation of temperature but no worsening of their clinical condition. One other patient had an elevation of temperature but this patient developed gastro-enteritis the following day. A further two patients were contacts of scarlet fever and each had symptoms of pharyngitis and an elevation of temperature. One of these latter two patients carried Streptococcus pyogenes in a subsequent swabbing. Finally, there were two patients who developed otitis media

while in hospital, and one who developed gastro-enteritis. It is noteworthy that the first two patients in this group, those with a clinical relapse, responded satisfactorily to a change of treatment from sulphadiazine to intramuscular penicillin, and the reason for the relapse would seem to have been simply, unsuitable treatment.

(b) In the four patients where there was no significant change in the flora of the nose and throat when symptoms suggested an acquired infection, a few colonies of Staphylococcus aureus were isolated from the nose whereas, none had been isolated from the previous swabbing. In three of these four patients, an elevation of temperature, but no change in the clinical picture had occurred, and, in the remaining instance, there was a clinical relapse. It seems improbable that the slight changes in the flora of the nose had been responsible either for the elevations of temperature or for the clinical relapse.

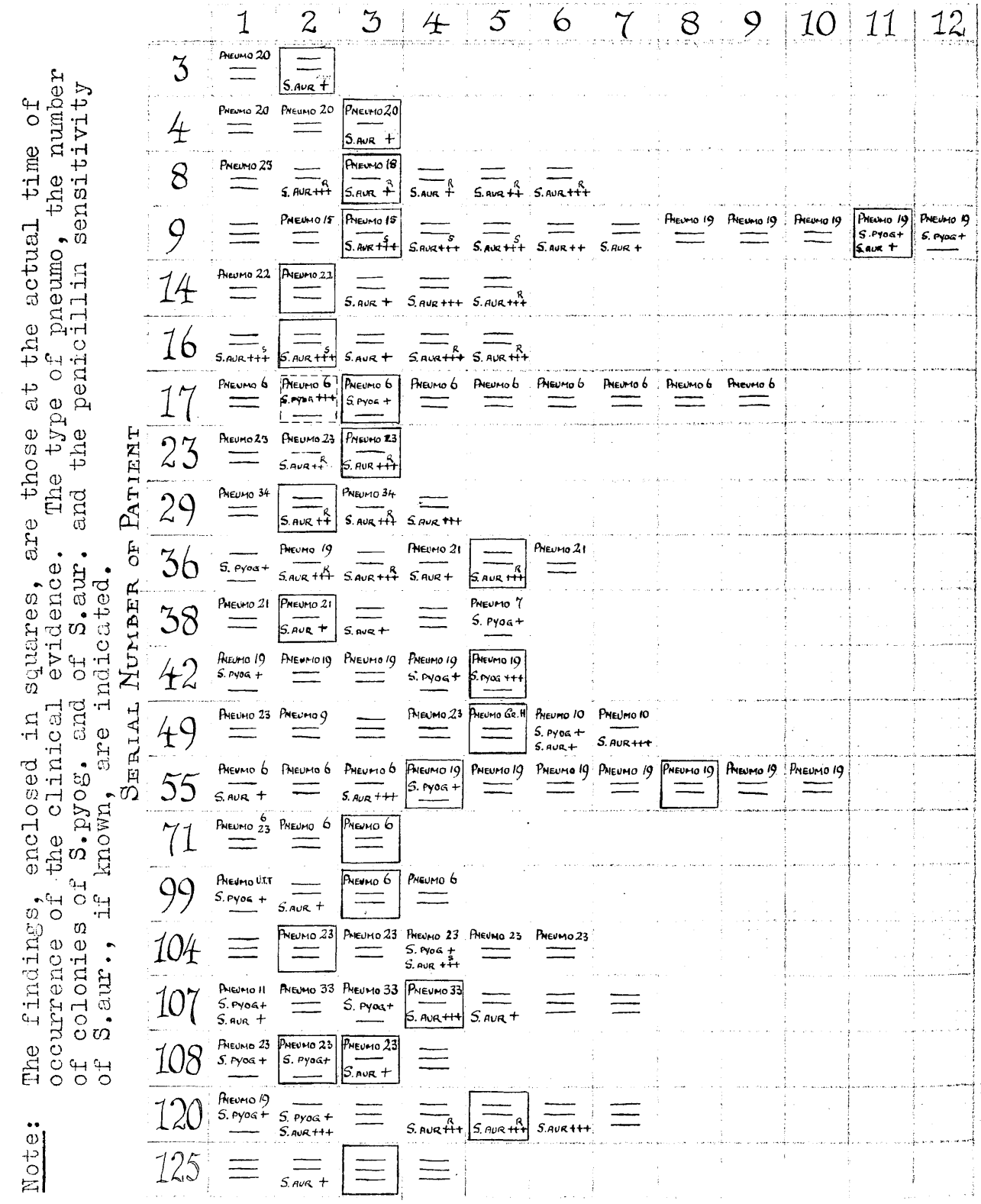
(c) In patient No.99, an elevation of temperature was associated with the acquisition of a type 6 pnuemococcus in the nose. This organism persisted in later swabbings and had not been present in the two previous swabbings. This was the only instance in the whole investigation where there was any clinical upset associated with the acquisition in the nose or throat, of a type of pneumococcus not previously isolated from the patient. In patient No.104 a pneumococcus had been acquired when the patient developed pharyngitis. This

patient had only been swabbed on one occasion before this incident and that was two days previously. It seems unlikely, thought not absolutely impossible, that the acquisition of the pneumococcus was responsible for the pharyngitis.

(d) As would be expected, the occurrence of scarlet fever in patient No.42 was associated with the acquisition of Streptococcus pyogenes in the patient's throat. Patients No.9 and 55 were contacts and each developed tonsillitis. They were also found to have acquired Streptococcus pyogenes in their throats. It seems probable moreover that two other contacts, patients No.49 and 71 who, despite symptoms of pharyngitis were not found to have acquired any organisms in their throats, may in actual fact have had a small number of streptococci, the colonies of which might not have been discernable to the naked eye. Subsequently patient No.49 was found to have acquired Streptococcus pyogenes in the throat when he was swabbed nine days later. The remaining patient was patient No.17 and the manifestation taken as suggesting an acquired infection was the development of a fresh area of consolidation in the contra-lateral lung to that involved by the original consolidation. There had been no relapse in the clinical condition except an elevation of the temperature on the third day in hospital. Streptococcus pyogenes, in considerable numbers, had been acquired in the throat when the patient was swabbed on the eighth day, and were still present in the

Figure 4.

The swabbing results for the patients manifesting clinical evidence of cross infection.



Note: The findings, enclosed in squares, are those at the actual time of occurrence of the clinical evidence. The type of pneumo, the number of colonies of S.pyog. and of S.aur. and the penicillin sensitivity of S.aur., if known, are indicated.

throat when the fresh area of consolidation was discovered on the fourteenth day.

(e) In two of the five patients who had acquired Staphylococcus aureus in moderate or large numbers, septic skin lesions had appeared. In both instances, the septic lesions and the staphylococci in the nose disappeared with treatment and it seemed likely that this organism had not only been acquired in the skin but also in the nose.

A third patient had an unexplained elevation of temperature and a fourth, a relapse in the clinical condition. The former may have been the result of the acquisition of coagulase positive, penicillin resistant staphylococci and likewise the latter, although staphylococci had been present at a previous swabbing. These however, had not been tested for coagulase and penicillin sensitivity.

The fifth patient, No.8, deserves special mention. This patient was found to have a large number of coagulase positive, penicillin resistant staphylococci in the nose when swabbed on his eighth day in hospital at the completion of a course of treatment by intramuscular penicillin. This organism was still present in the nose when the patient's clinical condition became grave and it was at this time isolated from the lung parenchyma by lung puncture, thus proving its etiological significance.

All the above findings are summarised in tabular form in the appendix and Figure 4 summarises the bacteriological

findings for the patients discussed in this chapter.

(b) Discussion.

An extensive investigation into the incidence of cross infection in children's wards was carried out by Watkins and Lewis-Faning (1949). They investigated 9,618 patients and found that 679 (7.1%) had some clinical evidence of cross infection. The danger of cross infection was considerable although the percentage was lower than that found in the present investigation, (16.8%), which however included suggestive evidence as well as actual evidence of cross infection. Watkins and Lewis-Faning also found that 92 of their patients had two incidents and 20 had three or more incidents of cross infection while in hospital.

Melin (1949) studied nursery children and found that they often had protracted infectious lesions with frequent fresh attacks of illness, often due to other types of infecting bacteria. Melin took nasal and throat swabs on any clinical evidence of the development of an upper respiratory infection. The pneumococcus, Streptococcus pyogenes, and Staphylococcus aureus were especially looked for and the antibody titres for these organisms determined. He considered that the acquisition of Staphylococcus aureus had considerable etiological significance as far as the very small infant was concerned, but that the acquisition of a pneumococcus was important in any child under four years of

age. On the other hand, investigations done in a residential nursery by Landsman (1949) did not in particular associate the pneumococcus with the development of upper respiratory infection. Similarly in the "Baby Families" investigations there was no evidence that the occurrence of a fresh respiratory infection was due to the acquisition of a new type of pneumococcus.

The danger, especially to the young infant, of the acquisition of Staphylococcus aureus is recognised by most writers. Melin showed that it was most dangerous as regards the infant under three months of age. The danger is becoming greater because of the prevalence of penicillin resistant strains (Barber and Rozwadowska-Dowzenko, 1948). Patient No.8, in the present investigation was a case in point.

The role of Streptococcus pyogenes as the cause of cross infection in wards for the treatment of measles is well known. Allison (1938) found that 19 per cent of patients suffering from measles, developed otitis media due to acquisition of Streptococcus pyogenes. In an open ward used for the treatment of scarlet fever, 70 per cent of the patients became infected with different types of Streptococcus pyogenes and these cross infections were responsible for many of the second attacks and late complications, (Allison and Brown, 1937). The writer was probably fortunate that the introduction of a virulent

Streptococcus pyogenes into the ward resulted in as little illness as proved the case.

In comparison with the findings of the above mentioned writers, the results of cross infection, found by the present writer, were not very serious. Gastro-enteritis and otitis media each occurred only twice. Septic lesions of the skin or mucous membrane occurred twice and the introduction of Streptococcus pyogenes into the ward was responsible for the development of scarlet fever in one patient and of tonsillitis in another two.

The only indication of acquired respiratory infection was the occurrence of five instances of relapse in the original respiratory infection and the development in one instance of fresh consolidation. Even this is an over-statement since two of these relapses were the result of unsuitable treatment.

The etiology of the seven unexplained recurrences of fever is uncertain. Only two were associated with the acquisition of an organism, in one, a pneumococcus, in the other, Staphylococcus aureus.

On the whole then, the incidence of clinical evidence suggesting cross infection and especially that suggesting respiratory cross infection was limited. Being so, the over all picture was complimentary to the nursing staff, although disappointing from the point of view of the writer who wished to investigate the cause of acquired respiratory

infection. Moreover the use of antibiotics was probably of some preventative value.

(c) Summary.

(1) During the investigation 22 patients manifested signs suggesting an acquired infection, three had two such episodes. Included as a sign of probable acquired infection was the occurrence of an unexplained rise in temperature of which there were seven instances. There were only four instances of a probable acquired respiratory infection.

(2) In the majority of instances there was no change in the flora of the nose and throat. On one occasion only was an elevation of temperature associated with the acquisition of a pneumococcus and on no occasion was a relapse in the respiratory infection due to a similar acquisition.

(3) Scarlet fever occurred once and on several occasions a throat infection developed when Streptococcus pyogenes was acquired.

(4) The acquisition of Staphylococcus aureus was associated with the occurrence of septic lesions in two patients, an elevation of temperature in one patient and a relapse in the clinical condition in two others. In one of the latter the relapse was severe and the organism was isolated from the lung parenchyma.

(5) Gastro-enteritis, otitis media and a non streptococcal throat infection accounted for only five of the instances suggesting acquired infection.

Chapter 2.

Bacteriological evidence of Cross Infection.

In this chapter the writer considers such evidence as was found of bacteriological cross infection either between one patient and another or between a member of staff and a patient. It has been shown in previous chapters that frequently, organisms were acquired by patients or staff and usually without any clinical upset. In the following pages the results of tracing the source of these organisms are studied.

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Bacteriological evidence of cross infection.

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a) The Pneumococcus.

The ease with which the pneumococcus could be accurately typed rendered this organism very suitable for any investigation into its epidemiology. It was found that during the entire period of the investigation, 62 types of pneumococci were acquired by 48 patients. Of these, 34 acquired one type whereas the remaining 14 acquired two types of pneumococci during their stay in hospital. More than half of these patients had already carried a pneumococcus, of a different type, on admission to hospital.

Having found that there were 62 instances of a pneumococcus being acquired while in hospital, the question then arose as to the sources of the acquired pneumococci. If indeed one patient did infect another, the writer wished to determine if the patients needed to be in adjacent cots for this to occur, or if, in fact, one patient could infect another at some greater distance. To facilitate answering these questions diagrams were drawn showing the positions in the ward of patients carrying each specific type. These, and a summary of the findings in each instance of the acquisition of a pneumococcus, are shown in the appendix. The diagrams and summary also take into account those members of staff carrying the type in question. The possibility that a member of staff might infect a patient

was in this way determined.

Despite careful search it was found that in 24 of the 62 instances no other patient or member of staff carried the homologous type of pneumococcus acquired by the patient. Thus, in 38.7 per cent, the writer's methods failed to trace the source of the acquired pneumococcus. In 61.3 per cent, however, there existed a possible source, or possible sources, amongst the other patients and/or the members of staff.

In 14 instances there was evidence that the source of the acquired pneumococcus was the patient in the adjacent cot. In some, the evidence was more conclusive than in others, where the period of contact between the two patients was shorter. In yet another 12 instances, the type of pneumococcus acquired was, or had recently been, carried by another patient not in the adjacent cot, but in the same division of the ward. It is suggested that infection occurred between the patients in a division of the ward and this postulated some vehicle for infection, either the ward air, the ward dust or the clothing or hands of the nursing or medical staff.

The type of pneumococcus acquired by a patient in a further 10 instances, was carried by a patient in another division of the ward. Although it may seem unlikely that

infection could occur between patients so far distant as this from each other, in 5 of the 10 instances, the type involved was one of the more unusual types and carried by so few other patients throughout the investigation, that it made the possibility of transferred infection more real. Moreoever in none of these 10 instances was there any possibility of a member of staff being the source of infection.

In the remaining 26 instances, no other patient, at or near the time when a pneumococcus was acquired, carried the type in question.

Much less frequently involved as probable sources of the acquired pneumococcus, were the members of staff. In only 10 of the 62 instances was there a carrier, amongst the members of staff, of the type of pneumococcus acquired by the patient. In another five instances a member of staff had carried this type at one time while in the ward but not actually at the time of acquisition. In all the other 47 instances there was no possible source of the acquired type amonst the members of staff.

The position was further complicated by the fact that in 11 of the 15 instances where there was some indication that a member of staff might have infected a patient, there was also evidence that the patient might

have been infected by another patient in the ward at that time. When the types of pneumococci involved were taken into account, it was found that types 6 and 23 were the types acquired when either a patient or a member of staff could be the possible infecting source. These types were at all times frequently carried by both patients and members of staff, and therefore, on any one occasion when a patient acquired either of them, there were several carriers of these types in the ward. There thus frequently existed several possible sources of infection. In the remaining four instances such evidence as existed to point to the source of infection involved members of staff only. However, in only one instance could it be said with any degree of certainty that a member of staff was the source of the acquired pneumococcus.

One may conclude from the foregoing that it was seldom possible to trace an acquired pneumococcus to a source amongst the members of staff.

Table 31 summarises the types of pneumococci acquired by the patients and the frequency with which each type was acquired. Types 6 and 23 were the most frequently acquired. This was not unexpected considering the over-all frequency of occurrence of these types. Several possible sources of infection usually existed. Other types were less frequently

TABLE 31.

Showing the frequency with which each type was acquired.

(a) Patients.

Number of times each type was acquired.	TYPES.
1	1:2:9:11:16:18:24:42:(U.T.T.)
2	17
3	15:21.
4	7:10:19:22:33.
6	4.
7	23.
12	6.
TOTAL:62	

(b) Staff.

Number of times each type was acquired.	TYPES.
1	2:3:10:11:18:21:29:37.
2	14:15:22:23:35.
4	6
TOTAL:22	

acquired and usually with these types, the source of infection could be postulated with some degree of certainty. The adult pathogens, types 1 and 2, were seldom acquired, but type 4, which is also a pathogen of adult pneumonia, was acquired six times. After one patient in Week 7 acquired this type, a further four patients acquired it in the following weeks. It would appear that this type, which is not a common commensal or a common pathogen in the young child, easily passed from patient to patient when once introduced into the ward. On the one occasion when type I was acquired, no other patient or member of staff carried this type, and thus the source of the organism could not be suggested.

Members of staff themselves, acquired pneumococci on 22 occasions. These instances were dealt with in similar fashion to those occurring amongst the patients, and an attempt was made to determine from which individual the pneumococcus had been acquired. In only five was there a possibility that the pneumococcus was acquired from another member of the staff, but in four of these there was also a patient in the ward who carried the homologous type at the time of acquisition and could thus also be the source of infection. There were in all nine instances where a patient provided a probable source, the patient carrying the acquired type at the time of its acquisition by a member of staff. In a further two instances a patient carried the

TABLE 32.

To compare the findings for patients with those for the staff when a pneumococcus was acquired.

	Staff		Patients	
	No.	%.	No.	%.
Source traceable to a patient.	12	54.6	36	58.1
Source traceable to a member of staff.	5	22.7	15	24.2
Source traceable to a patient only.	8	36.4	25	37.1
Source traceable to a member of staff only.	1	4.5	4	6.4
Source traceable to a patient and a member of staff.	4	18.2	11	17.7
Source not traceable either to a patient or a member of staff.	9	40.9	22	38.7.
TOTAL:	22	100.0	62	99.9

acquired type in the previous week but not at the actual time of acquisition by the member of staff. In one other instance both a patient and a member of staff were found to have acquired pneumococci of the same type in the same week. Either may have infected the other. The source of the acquired type could not be traced to either a patient or a member of staff in nine instances. The above findings can be summarised and compared with those for the acquisition of pneumococci by patients. (Table 32). The figures are almost identical although the totals (22 and 62) are so dissimilar.

One may say in conclusion that when either a patient or a member of staff acquired a pneumococcus, in just over half the instances one could find the possible source of the acquired pneumococcus amongst the patients and in just under a quarter, amongst the staff. One could determine the possible source of infection with some ease when the type acquired was one of the less frequently occurring. On the other hand, the source was difficult to determine when type 6 or type 23 was acquired since several possible sources usually existed amongst both patients and staff.

No definite statements can be made regarding the findings for the acquisition of pneumococci by members of staff because of the large number of their contacts outwith the hospital who provided possible sources for acquired pneumococci.

b) Streptococcus pyogenes.

The question of transference of streptococci from patient to patient and from member of staff to patient, can be dealt with in a similar manner to that used for pneumococci. A summary of the findings and the explanatory diagrams are shown in the appendix.

There were 32 instances of acquisition of Streptococcus pyogenes by patients. Twenty-five patients acquired Streptococcus pyogenes after admission to the ward, a further three patients appeared to have acquired the organism, but one had carried Streptococcus pyogenes five weeks beforehand and the other two had carried it seven weeks beforehand. Finally, from four patients a heavy growth of streptococci was isolated, whereas in the week previous, only a few colonies had been isolated.

Of the 32 instances, it was found that in six, infection could have arisen from the patient in the adjacent cot, in 14 from a patient in the same division, and in four, from a patient in another division. In the remaining eight, the possibility that a patient was the source of the acquired organism could be excluded.

In three instances, a member of staff was the likely source and in another 21 instances members of staff could not be excluded because carriers of streptococci were present amongst them at the time of acquisition of the organism by a patient. The remaining eight instances

occurred in weeks 12, 26 and 27, when the members of staff were not swabbed.

Combining the above findings one finds that, in the eight instances where the members of staff were not swabbed, a patient provided a likely source of the acquired organism and in the eight where there was no possibility of a patient constituting the infecting source, possible sources existed amongst the members of staff. In all other instances, although in all probability a patient was the infecting source, the members of staff could not be completely excluded from being possible sources of the acquired streptococci.

Comparing the above findings with those for the pneumococcus one notes that (a) a member of staff formed the probable infecting source more frequently; (b) in no instance was it impossible to find a probable source as occurred in 38.7 per cent of the instances when a pneumococcus was acquired; (c) in the case of acquisition of the streptococcus, not only was the percentage where the source was traceable to a patient higher, but the patient could more certainly be considered as the infecting source since in the majority, infection could be traced to a patient in the adjacent cot or at least in the same division.

There were only five instances of Streptococcus pyogenes being acquired before week 10. Thereafter there were 16 instances between week 10 and week 14. Until the

end of the investigation in week 27, there were only another 11 instances. The occurrence of this period of increased rate of acquisition merits further comment and the details will now be considered.

In week 10 patient No.42 developed scarlet fever and in week 11 four patients acquired Streptococcus pyogenes. One of these patients was in the adjacent cot to patient No.42, the other three in other divisions. Also in this week, six members of staff carried Streptococcus pyogenes, four of them for the first time. These patients and members of staff acquired the streptococci almost simultaneously and this suggested a common source of infection. Patient No.42 was in all probability that common source of infection and the floor dust the probable vehicle of infection since it contained the organism at this time and since the organism was acquired by patients in more than one division at one time. It was rather more difficult to find the source of the Streptococcus pyogenes which patient No.42 acquired and which resulted in the development of scarlet fever. There was a possibility that the infection originated from staff No.13. This nurse was found to have acquired many streptococci in her throat when she complained of a painful throat in week 3. It does, however, seem unlikely that a patient would acquire a virulent, streptococcus from this nurse seven weeks later, by which time the nurse was much less heavily infected.

It was more likely that patient No.42 was infected through the medium of the floor dust which contained Streptococcus pyogenes in week 9 and again in week 10 before the patient developed scarlet fever. One was now faced with the task of finding out how the dust became infected in the first place. Firstly, the dust might have been infected by patient No.41 who carried many streptococci in week 8, prior to dismissal, but had also carried streptococci in small number in week 7. Another and more likely explanation was that the source was the Christmas decorations which had been hung in the ward in week 9. These decorations had once been in a scarlet fever ward but were reported to have been fumigated. It is possible that a few streptococci remained in the dust of these decorations and then infected the floor dust, and thus the patient, since it is well known that streptococci can persist in dry dust for many months. (Medical Research Council,1951, White 1936). Lastly, the organism may simply have been brought into the ward on the clothes or shoes of a member of staff.

On the five occasions before week 10 when a patient acquired Streptococcus pyogenes, a member of staff was the probable source of infection. After the development of scarlet fever in patient No.42 in week 10, four patients in week 11, six patients in week 12, two in week 13 and three in week 14 acquired Streptococcus pyogenes. In all but one,

infection could be traced to a patient in the same division of the ward. The exception could possibly be traced to a patient in the adjacent division. All but one were associated with the presence of streptococci in the dust of the division in which the patient was. The simultaneous presence of carriers of Streptococcus pyogenes amongst the members of staff prevented those members from being excluded as possible sources of infection.

In each of the 11 instances occurring after week 14, a patient in the adjacent cot or at least in the same division, was considered the possible source although again, members of staff could not be excluded. Only one instance, in week 14, was associated with the presence of the organism in the dust of the same division. In a further four, dust from another division only was found to contain the organism.

Acquisition of Streptococcus pyogenes by members of the staff also occurred. On 24 occasions it was found that a member of staff had acquired this organism. This includes three instances where a large number of organisms was isolated whereas on some former occasion a few organisms had been isolated, and a further two instances where members of staff were again found to be carrying streptococci after several weeks in which they had been free of the organism. This figure of 24 for the 42 members of staff represents a higher rate of acquisition than occurred with the 131 patients, amongst whom there were 32 instances. This could

be explained by the fact that they, on the average, were examined more frequently than the patients and were longer exposed to infection with more chance of acquiring the organism. Moreover, the use of penicillin must have had some effect on the rate of acquisition of Streptococcus pyogenes by the patients. (Section 2; Chapter 3).

The acquisition of Streptococcus pyogenes by members of staff occurred with the same increase in frequency in certain weeks as had been found on studying the findings for the patients. In the initial weeks of the experiment eight instances occurred and the rest between weeks 9 and 18. Of these, 12 occurred in the period, week 10 to week 13, which followed the development of scarlet fever in patient No.42. There were no instances after week 18 and thus the majority of the instances occurred when the organism was present in the floor dust.

It was difficult to decide with any great degree of certainty whether the members of staff acquired the organism from other members of staff or from the patients. During the period, weeks 10 to 13, so many patients and members of staff carried Streptococcus pyogenes that many possible sources of infection were present. In the period, weeks 3 to 6, staff No.13, a nurse, was probably responsible. She acquired Streptococcus pyogenes and developed tonsillitis in week 3. Although the illness cleared up quickly she continued to carry large numbers of streptococci in her

throat for several weeks. In the instances occurring after week 10, although members of staff could not be excluded, it was more likely that the patients in the ward were responsible for the infection. In one of these instances the member of staff was complaining of a painful throat when the organism was found to have been acquired.

To summarise, in 14 of the 24 instances, a patient was the possible source of the acquired streptococcus, in a further three, one could not exclude the possibility of a patient having been the source and in the remaining seven, patients could be excluded. In eight of the instances it was possible that another member of staff provided the probable source and in the remaining 16, although a most unlikely source, it was not possible to completely exclude another member of staff. From the foregoing one finds that in only three instances was it possible that both a patient and a member of staff could be the source and in 14 a patient was the more likely source although members of staff could not be completely excluded. It was found that, in five of the seven instances where infection from a patient could be excluded, there was a likely source of infection amongst the members of the staff.

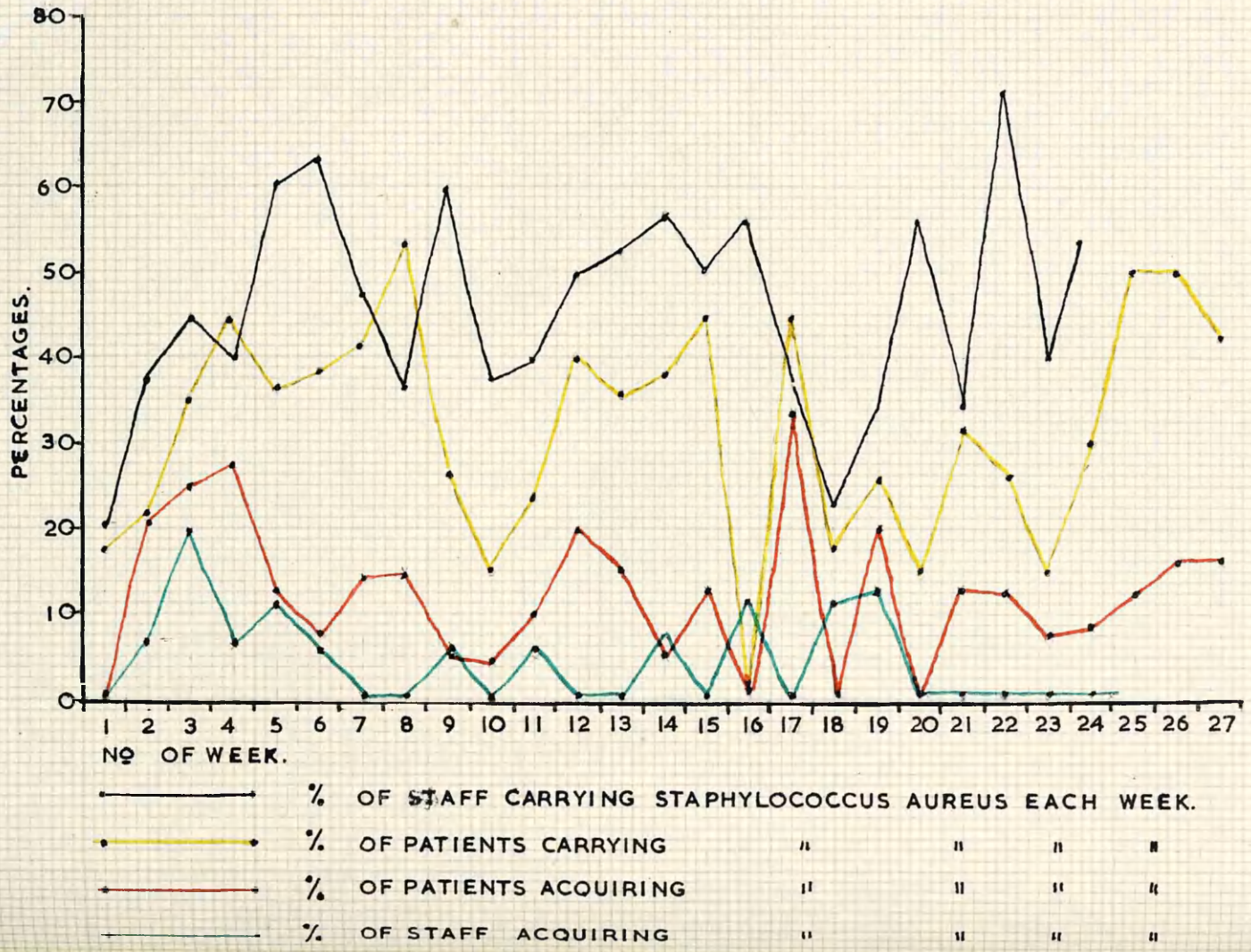
In conclusion one may say that when Streptococcus pyogenes was acquired by either a patient or a member of the staff, the source from which they were presumed to have been acquired was more frequently a patient than a member of

the staff. The findings, associated with the occurrence of scarlet fever in the ward, make one feel that the ward dust was closely connected with cross infection by streptococci, and was the actual vehicle of infection and not merely affording an indication that cross infection was going on, by virtue of it containing the organism. This surmise is in contrast to a statement made by Williams (1954) who said that in the investigation of an outbreak of infection by streptococci, "their presence in the dust may be no more than a byproduct of dispersal from a carrier to new host by much more direct means."

Figure 5.

The acquisition of Staphylococcus aureus.

GRAPH TO SHOW PERCENTAGES OF PATIENTS AND STAFF CARRYING AND ACQUIRING STAPHYLOCOCCUS AUREUS EACH WEEK.



c) Staphylococcus aureus.

This organism was carried by 87 (66.4%) of the patients and 32 (76.2%) of the staff at some time during the investigation. Sixty-four (48.8%) of the patients and 14 (33.3%) of the staff acquired the organism after their first swabbing. At all times, therefore, the ward was heavily populated by carriers of Staphylococcus aureus and thus on all occasions, when the organism was acquired, there were several possible sources of infection amongst both the staff and the patients. Since the actual source of infection would have been impossible to determine in almost all instances, one could only attempt to find out whether infection arose more frequently from a patient or from a member of staff. As was stated in a previous chapter, the dust did not seem to play the important part it did in the transference of Streptococcus pyogenes. The occurrence of Staphylococcus aureus in the dust did not come particularly at those times when there were many carriers in the ward or when most instances of acquisition of the organism occurred.

Although a large percentage of the patients acquired Staphylococcus aureus, a considerable proportion of the staff themselves acquired the organism. The graph, figure 5, however, shows that on all occasions (a) the percentage of staff carrying Staphylococcus aureus was greater than that of the patients so doing; and (b) the percentage of patients acquiring Staphylococcus aureus was greater than

that of the staff so doing.

It is possible then that members of staff infected the patients, since originally and at all times, the percentage of carriers amongst the staff was higher. Patient to patient infection was less likely since it has been suggested in an earlier chapter, that transference of staphylococci probably took place in the majority of the instances by direct contact, because of the preponderance of anterior nasal carriers. The patients were certainly not in direct contact and also rather far apart for direct droplet infection. Dust-borne infection was possible but if patient to patient infection did occur the most likely method would be by mediate contact by means of the hands or uniform of the members of staff, or occasionally by toys, thermometers, etc. If the former were the case the members of staff may easily themselves have become infected with the organism, unless already carriers. Finally, a member of staff could easily have infected another by direct or mediate contact.

It is thus likely that in the majority of instances, infection of the patients came from the staff, although on some occasions, infection may have arisen from another patient with a member of staff or the floor dust acting as the vehicle of infection. One other point in favour of staff to patient infection is the fact that patients admitted to the ward in the early weeks of the investigation

acquired Staphylococcus aureus as frequently as did those admitted later, when the total number of carriers amongst the patients was greater, thereby rendering the risk of infection from another patient the greater. Members of staff could as easily have acquired the organism from patients as from other members of staff, by direct or mediate contact.

Definite conclusions are, however, impossible without the aid of Bacteriophage typing.

d) Summary and Conclusions.

1. The Pneumococcus:

Pneumococci were acquired by 48 patients but this was an underestimate of the total number of times a pneumococcus was acquired. When the type was ascertained, it was found that there were 62 instances of a pneumococcus being acquired by a patient during his stay in hospital. The members of staff acquired pneumococci less frequently, 22 instances in all. It was to be expected that any source of infection existing in the ward would be determined fairly accurately because of the distinct serological types, and in this respect the pneumococcus was a more satisfactory organism to deal with than the streptococcus or the staphylococcus, which were not so typed.

In a considerable proportion of instances (38.7%), the source of the pneumococcus acquired by the patient could not be found. In 54.9 per cent, a probable source of infection was found amongst the patients and in a smaller number, (24.2%), amongst the members of staff. Similarly, in instances of acquisition of a pneumococcus by a member of staff, the source of the acquired organism could not be determined in a considerable proportion, but when it was determined, a patient formed that source more frequently than did a member of staff.

When the acquired type was one of the less commonly occurring types one could often say with some confidence

from which individual the organism came, but not so with the more frequently occurring type 6 and type 23. When these types were involved there were usually several possible sources from which a patient or a member of staff might acquire the organism. The acquired types were on a few occasions isolated from the floor dust of the ward.

A patient therefore, did seem capable of infecting another patient or a member of staff with a pneumococcus. In a few instances the infecting and the infected patients were in adjacent cots. In some, the patients were at least in the same division but on other occasions, the infected patient and the patient from whom infection was presumed to have come were in different divisions. If the latter were the case there is some doubt as to whether infection did occur from one to the other unless one accepts that the ward dust was definitely the medium of infection. When one takes into account the rapid death rate of the pneumococcus when exposed to conditions of drying such as would be found in the ward dust, the isolation of pneumococci from it even occasionally, suggests that the dust was infected by the patients or members of staff. It is probable that the dust could be the actual vehicle of infection from patient to patient and therefore also from patient to member of staff, since (a) the patients were far enough apart to make direct droplet infection from one to the other difficult if

not impossible, and (b) direct contact between patients did not occur. Mediate infection, however, could not be excluded since it did occur through the communal use of toys and thermometers and by way of the hands and uniform of the nurses who attended one patient after another. (Green and Penfold: 1947; Hare: 1941).

2. Streptococcus pyogenes.

Streptococcus pyogenes was acquired by the patients much less often than was the pneumococcus. The staff, however, acquired the organism as often as did the patients. The writer's findings suggested that usually the source of infection for patient and member of staff alike was a patient in the ward. The development of scarlet fever in a patient and the subsequent happenings helped to substantiate this view. The ward floor dust contained Streptococcus pyogenes especially at the time when most of the instances of acquisition of this organism occurred. The dust was thus the most likely vehicle of infection from patient to patient or from patient to member of staff. The dust would become infected from the droplets expelled from the patients and members of staff. The smaller droplets remain suspended for some time but the larger ones fall to the floor rapidly. (Hare: 1940; Bourdillon and Lidwell: 1941). Direct droplet infection from patient to member of staff or from member of staff to patient could easily have occurred although it would have been difficult between patient and

patient. Direct contact between patients and members of staff did occur as also did mediate contact between patients and therefore they must be included as possible methods of cross infection. Transmission by means of hands and clothing, contaminated by nasal carriers, serve as the principal means of contaminating the environment of other individuals in the opinion of Rubbo and Benjamin , 1951. There were, however, very few nasal carriers amonst the individuals examined by the present writer and the occurrence of mediate contact by fomites, between patients at least, was limited. The main thing in favour of dust borne infection was that the patients acquiring the streptococci were scattered throughout the various divisions and a positive dust sample was usually obtained from any division in which a patient acquired streptococci. Moreover, the dust carried Streptococci pyogenes before scarlet fever developed in one of the patients.

3. Staphylococcus aureus.

Staphylococcus aureus was acquired by a large number of patients and by a lesser number of members of staff. Owing to the fact that the organism was not typed and that there was a large number of possible sources of the organism amongst patients and staff alike, no attempt was made to determine the exact source of the acquired organism. It was however, more than likely that the staff infected the patients rather than that one patient infected another. Members of staff acquiring the organism might have acquired

it from either another member of staff or a patient. In view of the high nasal carrier incidence, infection by direct or mediate contact was more probable than by air borne droplets or by floor dust. (See dust sampling findings). In connection with this assumption one might mention a recent publication by Marsh and Rodway (1954). They found that dust suppressive measures had no effect in lessening the number of times Staphylococcus aureus was acquired by babies in neonatal nurseries. This finding bears out the writer's surmise regarding the method of transfer of these organisms.

Again may it be stressed what a danger the nursing staff constituted for the patient in the wards, especially as many of the staphylococci carried by the staff were penicillin resistant pathogens and at the time of these investigations the newer antibiotics were in very short supply.

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SECTION 4.

SECTION 4.

Discussion and General Conclusions.

Chapter 1.

Discussion

a) Presumed Etiology of Pneumonia:

As long ago as 1886, pneumonia was found to be associated with the organism, now known as the pneumococcus, (Frankel, quoted by Heffron, 1939). It was subsequently shown that the majority of instances of pneumonia in the adult were caused by a pneumococcus and at the present time, the pneumococcus is still responsible for most instances of lobar pneumonia in adults. In an investigation by the Medical Research Council in 1951, in which the writer took part, it was found that 69-79 per cent of cases of pneumonia in adults, from several hospitals throughout the country, were pneumococcal in origin.

With the development of pneumococcal typing, it was found that the pneumococci, most often associated with pneumonia in the adult, were types 1, 2 and 3. Other types were less frequently found. Pneumonia in the infant and child was also presumed to be due to the pneumococcus, but, many writers found that it was the higher types of pneumococci which were associated with the illness. (Tables 33 and 34). These higher types were also the types of pneumococci found in the healthy population of all ages (Park, 1930; Gundel, 1933; Straker, Hill and Lovell, 1939; Guthrie and Montgomery, 1948; Mendez and Likar, 1953).

Table 33.

Types of Pneumococci isolated by various authors from pneumonia patients and healthy carriers.

Pneumo-coccal Type.	Pneumonia in adults.		Pneumonia in infants and children.		Healthy Carriers.	
	No.	%	No.	%	No.	%
1	2,573	21.4	384	15.3	42	1.2
2	888	7.4	59	2.4	36	1.0
3	1,480	12.3	103	4.1	529	14.9
4	643	5.3	122	4.9	233	6.6
5	809	6.7	134	5.3	12	0.3
6 & 26	330	2.7	309	12.3	368	10.4
7	1,116	9.3	99	4.0	71	2.0
8	980	8.2	40	1.6	134	3.8
9	266	2.2	43	1.7	80	2.3
10	131	1.1	21	0.8	168	4.7
11	129	1.1	46	1.8	133	3.8
12	168	1.4	11	0.4	38	1.1
13	138	1.1	23	0.9	20	0.6
14	512	4.2	434	17.3	11	0.3
15 & 30	106	0.9	64	2.6	62	1.8
16	91	0.8	36	1.4	5	0.1
17	147	1.2	33	1.3	54	1.5
18	317	2.6	70	2.8	280	7.9
19	298	2.5	187	7.5	99	2.8
20	192	1.6	33	1.3	64	1.8
21	72	0.6	42	1.7	66	1.9
22	105	0.9	38	1.5	144	4.1
23	90	0.7	66	2.6	46	1.3
24	62	0.5	18	0.7	18	0.5
25	114	0.9	8	0.3	25	0.7
27	26	0.2	1	0.1	17	0.5
28	46	0.4	18	0.7	60	1.7
29	107	0.9	31	1.2	105	2.9
31	32	0.3	17	0.7	22	0.6
32	22	0.2	4	0.2	8	0.2
Not 1-32	49	0.4	12	0.5	593	16.7
TOTAL:	12,049	100.0	2,506	100.0	3,543	100.0

(From Finland, 1942a)

Table 34.

Types of pneumococci reported by various authors in pneumonia in children.

Country and Authors.	No.of Cases.	Type 1	2	3	Group 4.
America					
1916 Wollstein & Benson.					
1916 Pisek & Pease.					
1917 Mitchell					
1922 Lyon					
1923 Wollstein					
1926 Westlund					
1930 Trask et al.					
1930 Cooper					
1930 & 1931 Plummer et al.	958	128 13.4%	89 9.3%	54 5.6%	687 71.7%
Germany.					
1932 Gundel & Schaefer	52	6 11.5%	3 5.8%	3 5.8%	40 76.9%
Poland.					
1932 Stankiewicz et al.	64	23 36.0%	5 7.8%	6 9.4%	30 46.9%
Japan.					
1930 Ishida	123	22 17.9%	46 37.4%	11 9.0%	44 35.8%
Australia.					
1924 Webster	120	47 39.2%	6 5.0%	7 5.8%	60 50.0%
Scotland.					
1933 Blacklock & Guthrie	140	9 6.4%	3 2.1%	1 0.7%	127 90.7%
TOTAL: 14 authors	1457	235 16.1%	152 10.4%	82 5.6%	988 67.8%

(From Blacklock and Guthrie (1933))

Pneumonia in the infant differs from that in the adult not only in the type of pneumococcus associated with it but also in the distribution of the consolidation. In the infant, broncho-pneumonic or scattered lobular consolidation is characteristic and only in the older child does one find the lobar consolidation characteristic of pneumonia in the adult. (Sheldon, 1951). Pneumonia of a scattered lobular distribution is also found in the adult and is often caused by pneumococci of the higher types (Marshall and Perry, 1952). It is however, almost impossible to distinguish such cases from cases suffering from a diffuse aspiration pneumonia.

The epidemiology of pneumonia has been studied by many writers and was reviewed by Finland (1942b). The contagiousness of adult pneumonia is in little doubt but the factors underlying the susceptibility of one individual, so that he develops pneumonia, instead of remaining a healthy carrier, are not clearly understood. Pneumococci were presumed to pass from one individual to another by means of the expulsion, from the nose and throat, of droplets of discharge and the inhalation, by another, of these droplets. There was however, very little direct or positive proof that pneumonia was acquired through airborne droplet infection. (Finland, 1942b).

The high incidence of pneumococci of types 1, 2 and 3 as compared with the low incidence of these types in the healthy population was thought to indicate an exogenous

infection, whereas, the association of pneumonia in the infant and child with pneumococci of the higher types common in the healthy population of all ages was thought to be evidence of an endogenous infection.

It is frequently noted that, in the two or three days preceding the onset of pneumonic symptoms, the child shows symptoms and signs of an upper respiratory infection (Sheldon, 1951), and it could in all probability be this preceding respiratory infection which initiates the endogenous infection by pneumococci already present in the upper respiratory tract. (Dochez, Mills and Kneeland, 1933). Factors such as poor social status, environment, climate, are often blamed for the occurrence of pneumonia in the infant, but Landsman in 1953 showed that this had comparatively little effect and she incriminated some other factor in the initiation of the pneumonic process. She suggested that this factor might be viral in nature.

Alternatively, it may be that pneumonia in the young child or infant, is due to the acquisition of a pneumococcus, of one of the higher types, which the child has not already carried and which is of higher virulence to the child than to the adult. Thus it may be an acquired infection which causes the pneumonia but this may only develop when the respiratory tract mucosa has already been damaged by some other factor.

Even in the adult, the exogenous nature of pneumococcal

pneumonia is obscured by certain features. Many writers have found an increase in the carrier rate of the pneumococcal type causing the pneumonia, in the contacts of the patient (Stillman, 1917; Rosenau, Felton and Atwater, 1926; Jacobson, 1927; Smillie, 1933; Smillie and Leeder, 1934). This would substantiate the exogenous nature of the infection but it is difficult to understand why only one member of a group develops pneumonia.

The greater virulence of some pneumococcal types may disturb the random distribution of types in pneumococcal pneumonia and it may well be that a highly virulent and resistant type spreads in a small group or family and the occurrence of some factor reducing the resistance of one member will cause the development of pneumonia, in that individual (Jordan and Burrows, 1947). The writer has seen an adult suffering from pneumonia due to a type 2 pneumococcus suddenly relapse with a consolidation on the contra-lateral side due to a type 7 pneumococcus, a patient suffering from a type 7 pneumonia being at this time in the opposite bed. This would seem to be evidence of the exogenous nature of lobar pneumonia in the adult but the writer admits some other factor which rendered the patient susceptible to the acquired infection by the type 7 pneumococcus. The possibility that allergy may have been partially responsible cannot be excluded. (Sharp and Blake, 1930; Lindau, 1933; Fried, 1933).

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b) The Present Investigation and the Relationship of this Study on Cross Infection by the pneumococcus to the Etiology of Respiratory Infection.

One of the main reasons for undertaking this present investigation was to see if the exogenous type of infection, mentioned in the last paragraph, did occur in the infant. The writer wished to ascertain how great was the occurrence of cross infection by pneumococci in an open ward, and whether cross infection by pneumococci ever caused illness in the patient. The ward used for the investigation was at all times fully occupied by patients, nearly all of whom were suffering from bronchitis or pneumonia. These patients were exposed to the pneumococci and other organisms carried by the members of staff who looked after them. Each was also continually in the presence of the other patients, 61.8 per cent of whom were found to be carrying pneumococci. What is more, if one presumes that these pneumococci were the causal organisms of the respiratory illnesses it meant that the pneumococci to which the patients were exposed were virulent and capable of causing bronchitis or pneumonia.

On many occasions throughout the investigation a patient was cross infected by a pneumococcus. On only one occasion, when a pneumococcus was acquired, was there any suggestion of a constitutional upset; on this occasion, an elevation of temperature. On the few occasions when there was some clinical evidence suggestive of an acquired respiratory

infection, such as recrudescence of temperature or clinical relapse, there was never an associated acquisition of a pneumococcus.

The source of the acquired pneumococcus was looked for on every occasion with as much care as possible. In a small proportion it was traced to the patient in the adjacent cot. In a similar number of instances it could be traced to another patient in the same division of the ward and in several other instances it could be traced to a patient in another division of the ward. On some occasions the acquired pneumococcus was found in the dust of the ward. On other occasions, however, the pneumococcus could not be traced to any other patient in the ward nor was it found in the dust of the ward. This happened in 38.7 per cent of instances. A final, extremely important point was that there were few convincing instances of a member of staff forming the source of the acquired pneumococcus although the nursing staff was in very close contact with the patients. The nursing, medical and domestic staff were all however, in good health. If they had had upper respiratory infections, with the increased dissemination of the secretions of the upper respiratory tract which would result, one might have found some more convincing incidents of staff to patient infection.

If, as the writer's findings suggest, patients infected other patients with pneumococci, the question arises as to

how this infection took place. As was remarked in a previous chapter, the patients were rather far apart for direct droplet infection to occur. They were not in direct contact although some mediate contact did occur by means of toys and the nurses' hands and uniform. The remaining possibility was that the pneumococcus was transferred from one patient to another through the medium of the floor dust and pneumococci were found in the floor dust on several occasions. They were more frequently isolated when there was overcrowding of the patients and thus more possible sources of pneumococci to infect the dust. Transference of pneumococci by this vehicle would account for the fact that, on some occasions, the patient presumed to be the source of the infecting pneumococcus and the patient acquiring the pneumococcus were separated by the breadth of the ward or were actually in different divisions of the ward.

The pneumococcus is a delicate organism and one might presume would not survive as long in the dust as would Streptococcus pyogenes, but the information already known about this latter organism indicates what probably happens with the pneumococcus. (Thomas and van den Ende, 1941; Wright, Cruickshank and Gunn, 1944). In the first place the pneumococci are probably expelled in droplets by the patients. Many of the droplets fall on bed-clothes and floor, where they dry. At the times of bed making and floor

sweeping these particles are again dispersed into the air and as such may be inhaled by other patients. Fine droplets, however may remain suspended for some considerable time (Wells and Wells, 1936), and may also be inhaled by some person at some distance from the source. In all probability, what happens in the child's home is that dust and droplets containing pneumococci are inhaled and lodge in the nose or throat. No illness occurs and, more and more types of pneumococci are acquired until, by the time adult life is reached, a considerable number of the pneumococcal types have been carried at one time or another (Straker, Hill and Lovell, 1939).

It is of interest, at this point, to draw attention again to the findings of Melin (1949), who studied children in a residential nursery. (See Section 3; Chapter I). He presumed that the occurrence of respiratory incidents was due to the acquisition of pneumococci. There was no such evidence in the present investigation and although there were a few examples of clinical relapse or recrudescence of temperature, in none was there a new type of pneumococcus acquired. Unless one had some allergy to the type of pneumococcus acquired, it would seem then that the acquired pneumococcus would remain as an innocuous inhabitant of the nasopharynx until some other factor should cause downward spread of the organism to the endotracheal regions. These were the findings of Blake and Cecil in 1920, working with

Phillipine monkeys. Small amounts of a virulent pneumococcal culture injected intratracheally produced a disease similar to lobar pneumonia whereas spraying of large amounts into the nasopharynx failed to produce the disease, even although the pneumococci might be carried for some considerable time in the nasopharynx. The writer's own investigation, in the wards for the treatment of patients with measles, further confirms this assumption, since although pneumococci were acquired frequently by the patients, the acquisition did not bring about the development of a respiratory infection as a complication of measles. Although patients suffering from measles, as well as those suffering from bronchitis or pneumonia received chemotherapy, many of the pneumococci were acquired after therapy had been discontinued or were acquired by patients receiving no chemotherapy. In such patients, the possibility of chemotherapy having a preventive action on the development of a respiratory infection, did not exist.

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c) Comparison of the Writer's Findings regarding Cross-infection by the Pneumococcus, Streptococcus Pyogenes, and Staphylococcus Aureus.

At this point one must digress to consider the findings for the other two organisms, Streptococcus pyogenes and Staphylococcus aureus, for they differ in considerable measure from the pneumococcus. The acquisition of either of these two organisms was on occasion associated with the occurrence of illness on the part of the patient. In contrast to what was found regarding the pneumococcus there was definite evidence of members of staff infecting patients. Streptococcus pyogenes was readily acquired by patients and staff after introduction into the ward. Staphylococcus aureus was acquired by a large proportion of the patients and was the organism most frequently acquired. The writer's findings suggest that in the majority of instances a member of staff infected the patient with this organism. There was more or less conclusive proof that the dust was the vehicle of transference of Streptococcus pyogenes and that dust was seldom involved in the transference of Staphylococcus aureus.

Regarding the difference in the ease with which, and the method by which these organisms were passed from one individual to another, an interesting point was brought to light by the writer's investigations. The pneumococcus, Streptococcus pyogenes and Staphylococcus aureus each

occurred in the anterior nose, the posterior nose, and the throat but each had its site of election. The pneumococcus favoured the posterior nose, the streptococcus the throat, and the staphylococcus the anterior nose. The pneumococcus and the staphylococcus were frequently found in the alternative nasal site but seldom in the throat.

That there was such a difference in the behaviour of the three organisms must have had some bearing on the method of passage of the organisms from one individual to another. The pneumococcus and Streptococcus pyogenes were less commonly acquired than was Staphylococcus aureus. The former two organisms, occurring as they did in the posterior nose and throat, could only pass to another individual by droplet infection or by droplet infected dust. Unlike viruses, bacteria are less certainly transferred by this method than by contagion. The latter was the probable method of transference of the more frequently acquired Staphylococcus aureus. The more hardy nature of Streptococcus pyogenes would allow of its persistence in the dust whereas the pneumococcus must have frequently died. In this way a streptococcus introduced into the ward would spread rapidly and with more certainty than a pneumococcus. That this might have occurred is shown by the work in wards for the care of patients with measles or diphtheria.(Allison, 1938; Cruikshank and Godber, 1939).

Contagion is suggested as the most likely method of

transference of Staphylococcus aureus because of its frequent presence in the anterior nose, which part is in contact with the fingers of the individual very frequently. It was thus a very easy procedure for a member of the nursing staff to infect the patient whom she was nursing. Hamburger and Green (1946) found that chronic dangerous carriers of virulent streptococci were often heavy anterior nasal rather than heavy throat carriers. The anterior nose was thus a potential danger as a source of pathogenic organisms such as Staphylococcus aureus as well as Streptococcus pyogenes. The writer also found a considerable number of individuals who were anterior nasal carriers of the pneumococcus and it was possible that a member of staff might also have infected a patient with a pneumococcus by direct contact. Since there were few convincing examples of staff to patient infection, this could not have happened very frequently.

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d) The part played by the pneumococcus in the etiology of Pneumonia as based on the Writer's Bacteriological Findings for the Patients on Admission to Hospital.

The writer considered that although the numbers involved were small, an analysis of the initial bacteriological findings of the patients admitted to the ward, suffering from bronchitis or pneumonia, might elucidate some points regarding the part played by the pneumococcus in the etiology of bronchitis and pneumonia in the child.

Firstly the writer assumed that the organisms present in the nose and throat were the organisms involved in the pulmonary pathology. The basis of such an assumption is found in the work of Blacklock and Guthrie (1933) who, in agreement with work done by Kelly and Gussin (1924), Sutliff (1928), Park (1930), Plummer et al. (1930 and 1931), found that in 85 per cent of cases the type of pneumococcus isolated by a throat swabbing was in fact the causative organism of the pathogenic process. One might conclude from their findings that the same would apply for organisms in the throat other than the pneumococcus, should they be the organisms causing the pneumonia. Blacklock and Guthrie also suggest that, since the higher types of pneumococcus were frequently found as commensals in the throat, confirmation of their etiological significance be obtained by lung puncture or from autopsy specimens. The writer did

in fact perform lung punctures on several of the patients with almost uniformly unsuccessful results. The exception was that of a patient who carried a type 5 pneumococcus in the posterior nose and from whom the same type of pneumococcus was isolated by lung puncture. The reason for the "sterile lung punctures" obtained on other occasions might arise from the fact that those punctures were done after chemotherapy had been commenced and also from the fact that it was difficult to be sure that the puncture was being made into actual consolidated lung tissue.

Staphylococcal pneumonia and empyema was recognised as a distinct entity and the two cases of this which occurred in the writer's series of patients were considered separately from the others, the etiology of which the writer was trying to determine.

When an attempt was made to compare the present findings with those of other writers, it was found that nearly all reported work took place in the days before efficient chemotherapy. Many of the pneumococci isolated were from empyema pus or from autopsy specimens. This indicated a far more advanced disease process than the writer met with in the present series of patients. The incidence and type of pneumococci isolated from those above-mentioned sources can not be compared satisfactorily with the incidence and type of pneumococci isolated from the nose and throat.

The findings in the literature vary considerably from

country to country (Möller, 1942 and Mørch, 1943), especially as regards the prevalent types of pneumococci in the pneumonia of children. The prevalence of Type 14 is noted in American series (Table 35) whereas this type was seldom isolated from the patients suffering from pneumonia in Glasgow. (Guthrie and Montgomery, 1948). These writers, in agreement with the earlier writer Hendry (1942), found that in Glasgow, Types 6 and 19 were the most frequently isolated. These also were the types most frequently isolated from the noses and throats of the writer's patients.

Numerous writers have noted the influence of the age of the child suffering from pneumonia, on the type of pneumococcus isolated. Nemir, Andrews and Vinograd (1936) in America, Friederichsen (1939) in Denmark and Montgomery and Guthrie (1948) in Glasgow found different prevalent types of pneumococci in children over and in children under two years of age. Similar age influence in type prevalence was noted in the present writer's series of patients. In the child under one year of age Type 23 occurred most often, whereas in the older child, Type 6 was most frequent. When Type I occurred, as it did on a few occasions only, the child from whom it was isolated was always over one year in age.

It is thus obvious from the preceding paragraphs that comparison of the present writer's bacteriological findings,

with those of other writers is rendered difficult because of differences in several factors, including, the age of the patient, the severity of the disease suffered and the country in which the investigation took place. In view of this and the fact that the pneumococcus is such a frequent commensal in the nose and throat of the healthy child, it was considered that a more profitable source of information would be the comparison of the writer's findings with those for healthy children of similar age to the patients. In this way, one would have evidence against the acquired pneumococcus being a cause of the pneumonia if the pneumococcal carrier incidence amongst healthy and amongst ill children were similar.

Extensive studies were done in 1939 by Straker, Hill and Lovell, who found a considerable variation in the incidence of pneumococci from one healthy community to another and also a considerable seasonal variation. Therefore, to make a satisfactory comparison with the writer's findings a community similar to that from which the patients came, had to be found and the seasonal influence had to be obviated.

Figures for healthy carriers of pneumococci are available from the literature, Glynn and Digby in Liverpool, (1923); Park in America (1930); Gundel in Germany, (1933); Straker, Hill and Lovell in England,(1939); and Guthrie and Montgomery in Glasgow, (1948). The communities sampled

Table 35.

Comparing the isolation of the various organisms from certain groups of individuals.

Group of Individuals.	Total	Pneumococcus.		Streptococcus pyogenes.		Staphylococcus aureus.	
		No.	%	No.	%	No.	%
Patients suffering from bronchitis or pneumonia aged 0 - 6 years.	104	64	61.5	12	11.5	10	9.6
Patients with other conditions.	25	12	48.0	11	44.0	1	4.0
Baby Families aged 1 - 5 years.	111	62	55.0	24	21.6	8	7.2
Random Families aged 0 - 5 years.	70	46	65.7				

and the ages of the individuals examined make these findings unsuitable for comparison. More recently Mendez and Likar (1953), reported figures for families in the Paddington area. They found that 38 per cent of children under one year of age and 40 per cent of those over one year of age, carried pneumococci and the types in order of frequency were: 6, 19, 8, 14, 23 and 11. These results are more suitable for comparison with the present findings than those of the writers mentioned above, but in preference to them the findings from the 'Baby Families' and 'Random Families' investigations, described in the opening section, will be used. The former mentioned investigation was carried out concurrently with the writer's investigations, the latter, two years previously. The most important feature about these investigations, in rendering them suitable for comparative purposes, was that the healthy children examined, lived in a similar family environment to that from which the hospital patients came and should therefore provide an indication of the bacterial environment of the patients before they became ill.

As only the posterior nose and the throat of the healthy children were swabbed the writer's anterior nose findings have been excluded for the purposes of comparison. It will be noted from the tables, that, unfortunately, none of the children of the 'Baby Families' were aged under one year, (Tables 35 and 36). If, however, one again refers

Table 36.

Comparing the pneumococcal types isolated from the members of the Baby Families and the patients.

Baby Families - All Members (398)			Baby Families - aged 1-5 years (111)			Patients with bronchitis or pneumonia aged 0-6 years (104)		
Type Isolated	Isolations No.	%	Type Isolated	Isolations No.	%	Type Isolated	Isolations No.	%
6	34	24.3	6	21	33.3	23	16	19.8
23	22	15.7	19	12	19.0	6	15	18.5
19	20	14.3	23	8	12.7	19	8	9.9
11:22	7	5.0	11	4	6.3	1:14:21	5	6.2
14	5	3.6	14	3	4.8	11	4	4.9
10:18:33	4	2.9	4:15:18:22:33	2	3.2	7:22	3	3.7
13:15:29:41	3	2.1	5:13:16:29:U.T.T.	1	1.6	8:18:20	2	2.5
1:3:4:5:12:20:34	2	1.4				2:5:10:12:17:29:31:33:34:U.T.T.:Gr.A.	1	1.2
2:8:16:21:25:39:U.T.T.	1	.7						
No.of types isolated: 140			No.of types isolated: 63			No.of types isolated: 81		

to the work of Mendez and Likar (1953) one finds that the pneumococcal carrier incidence in children under one year and in those aged between one and five years were virtually the same.

Pneumococci were isolated from 61.5 per cent of the patients, from 55 per cent of the children of the 'Baby Families' and from 65.7 per cent of the children of the 'Random Families'. There was no difference in the isolation rates for Staphylococcus aureus from the three groups, but Streptococcus pyogenes was isolated more frequently from the healthy children than from the children with a respiratory infection, but not as frequently as from the group of 25 patients with other conditions. The difference between the percentage of patients and that of either group of healthy children, who carried pneumococci, is not statistically significant.

From all groups of individuals, adult and child, pneumococci of types 6, 19 and 23, were the most frequently isolated. Types 6 and 23 were more frequently isolated than type 19 from the patients, whereas, from the healthy children, types 6 and 19 were more frequently isolated than type 23. No significance is attached to this difference since in patients under one year of age, type 23 was more common than type 6, whereas in patients over one year of age type 6 was more common than type 23. The type prevalence was similar for the adult, the adolescent and

the child members of the 'Baby Families'.

Type 1 occurred only in patients. Type 21, although only accounting for .7 per cent of all types isolated from all members of the 'Baby Families', was responsible for 4.9 per cent of the isolations from the patients if one excludes the anterior nose findings. Type 1 and type 21 were thus the only two types of pneumococci which appeared to be pathogenic in the pneumonia of young children and they accounted for only a few instances.

As was previously stated, the subjects examined in the 'Baby Families' investigation were all aged over one year. The pneumococcal carrier incidence amongst patients suffering from either bronchitis or pneumonia who were aged over one year was 72.1 per cent (anterior nose findings excluded), and this is considerably higher than 55 per cent, the corresponding incidence amongst healthy children. The difference is, however, not quite statistically significant but is at least suggestive of a correlation between the pneumococcal carrier incidence and the occurrence of bronchitis or pneumonia. For the patients under one year of age the pneumococcal carrier incidence was 54.1 per cent. If one, however, considers separately patients suffering from bronchopneumonia and those suffering from a segmental or lobar pneumonia and compares the pneumococcal carrier incidence in these two groups, 51.4 per cent and 84.4 per cent respectively, with that in healthy children,

55 per cent, one finds that it was those patients with a segmental or lobar pneumonia who had a higher pneumococcal carrier incidence than did healthy children. The difference is statistically significant. It was these latter patients who were mainly aged over one year and thus accounted for the higher carrier incidence in patients over one year.

To summarise, it was only when patients with a segmental or lobar pneumonia were considered, that any significant difference was found between the pneumococcal carrier incidence amongst patients suffering from pneumonia and that amongst healthy children. The only pneumococcal types which appeared to be of particular pathogenicity for patients were Type 1, which is one of the pathogens of pneumonia in adults, and Type 21, which accounted for only a few instances.

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Chapter 2.

General Conclusions regarding the Etiology of Pneumonia and Bronchitis in the Infant and young Child.

On the evidence gained from the investigations there is little reason to believe that either bronchitis or pneumonia in the infant under one year of age is due to an exogenous infection by a pathogenic type of pneumococcus. This statement is made because (a) the rates of isolation of pneumococci and of the various types taken individually, from the noses and throats of patients and of healthy children, were similar; (b) a considerable percentage of patients suffering from bronchitis or pneumonia did not carry pneumococci in their noses or throats, and (c) the acquisition of similar types of pneumococci to those isolated from patients on admission, occurred frequently during the investigation and was not associated with respiratory illness.

In the child over one year of age there was some evidence that the respiratory illness might be due in some instances to the acquisition of a pneumococcus of a type common in the general population, and not usually pathogenic. Suggesting this was the increased frequency of isolation of pneumococci from these children compared with that from healthy children, and also the x-ray appearance which was somewhat reminiscent of that found in the pneumococcal pneumonia of the adult. This surmise may, however, be

erroneous for two possible reasons. Firstly, the increase in carrier rate may only have been the result of the respiratory infection causing an increase in the number of pneumococci present in the nose and throat, and thus rendering the isolation of pneumococci from a nose or throat swab, the more likely. Secondly, those types of pneumococci which one is presuming were acquired by the patients were also carried by healthy children and were frequently acquired by other children while in hospital, without the development of illness.

The foregoing statements make one wonder regarding several points. Firstly, if in some older children, the pathogenic organism of the bronchitis or the pneumonia, was a newly acquired pneumococcus, one wonders why this organism should have been pathogenic to one child and not to another. Secondly, in the child under one year of age, one wonders why organisms already present in the nose and throat should suddenly become pathogenic.

The former question also, of course, applies to pneumonia occurring in the adult, since some individuals may acquire a type 1 or a type 2 pneumococcus and develop pneumonia whereas other contacts may carry these types and develop no illness. One usually finds, however, that when a type 1 or a type 2 pneumococcus is introduced into a small community only one individual will develop pneumonia but that the incidence of upper respiratory illness amongst the

others is high. (Smillie, 1933; and Smillie and Leeder, 1934). This association with an upper respiratory infection is important but it also seems to be essential that there should exist an especial susceptibility of the individual himself before the acquired pneumococcus will cause pneumonia. It is very likely that allergy plays a considerable part in the development of this susceptibility. It is also possible that the same may occur in the child who has become allergic to a certain type of pneumococcus which may have caused some previous infection. The presence of an upper respiratory infection in the family may predispose to the overgrowth of pneumococci in the nose and throat and thereby increase the ease of passage of a pneumococcus from one individual to another. The patient may acquire the pneumococcus and the upper respiratory infection, already present, may predispose to the organism spreading downwards along the respiratory tract. An allergic response may lead to the development of pneumonia due to the acquired pneumococcus. Alternatively, the patient may acquire the pneumococcus and because of the concomitant upper respiratory infection, may aspirate catarrhal discharge, infected with the pneumococcus, and thereby develop pneumonia.

Although types I and 2 account for the majority of pneumococcal pneumonias in the adult, they are seldom associated with pneumonia in the child. This may be due to the greater virulence of type I and type 2 for the adult than for the child. On the other hand, the writer found

that type 21 was the only type which seemed to be specifically pathogenic for the young child. Types I and 2 were however also isolated from the patients on a few occasions. It is of interest that the one patient in the writer's series who suffered from pneumonia due to a type 2 pneumococcus, infected the patient in the adjacent cot with the organism. Furthermore, the organism was also present in two dust samples, taken from the vicinity of the patient. This is remarkable considering the more frequent isolation from the patients of other types of pneumococci which were seldom found in the floor dust. It rather suggests that the increased virulence of type I and type 2 pneumococci is in some way associated with an increase in the ability of the organisms to withstand the unfavourable conditions in the dust and therefore to persist longer and have more opportunity of infecting other individuals.

In the infant under one year and also in the older child whose infection is endogenous and not acquired, the factors which initiate the pathogenicity of the higher types of pneumococci present in the nose and throat, may be peculiar to the environment of the child. What seems more likely is that there is some factor which causes damage to the mucous membrane of the upper respiratory tract, and thus encourages the increased colonisation of the respiratory tract by the organisms present in the nose and throat and their downward spread to the endotracheal region.

The increase in colonisation may give rise to the impression, which the writer had, of increase in the number of pneumococci present in the nose and throat. Furthermore, the anatomy of the respiratory tract of the child is such as to facilitate the downward spread of infection. (Blacklock and Guthrie, 1933). It seems possible that this unknown damaging factor is viral in nature and perhaps similar to the common cold or influenza virus.

In connection with this theory one might mention the results of swabbing the contacts of the patients in this investigation who suffered from bronchitis or pneumonia. These were reported by Anderson (1953). Contacts, in the adult age group, had a higher pneumococcal carrier rate than did adult members of the general population. The increase was not due to the homologous type isolated from the patient. Furthermore, the incidence of non-pneumonic upper respiratory infection was high amongst these contacts. Anderson suggests that acute respiratory infection in the older members of the family leads to an increase in the number of pneumococci in the respiratory tract and therefore to an increased 'dose' of organisms to the infant who develops pneumonia. There is however, another possibility, that the same acute upper respiratory infection affects the infant, damages the mucous membrane and leads, by virtue of the anatomy of the infant's

respiratory tract to increased colonisation of the mucous membrane by the pneumococci and other organisms already present in the nose and throat. Once the pneumococci or other organisms became endotracheal, they could easily go on to produce bronchitis, bronchiolitis or pneumonia.

In the report by Landsman (1953) on the 'Baby Families' it is shown that the newborn babies acquired pneumococci, presumably from the older members of the family, and although those babies were subjected to conditions of over-crowding and under-nutrition, respiratory infection did not develop in a large number. This finding does much to disprove the usual theories that social and environmental factors are of considerable, if not prime importance, in the etiology of pneumonia in infants and young children. When an upper respiratory infection is introduced into an overcrowded family, in poor conditions, it is likely that the infection is more liable to spread to all members including the infant, who may subsequently develop pneumonia.

Finally, the importance in all age groups, of aspiration as the cause of pneumonia of an atypical character is being realised. Marshall and Perry, (1952), as mentioned before, have suggested that the pneumonia associated with the higher types of pneumococci in the adult is often accompanied radiologically by a diffuse lobular picture, likely to be due to the aspiration of infected material. The writer has suggested in the previous paragraphs that an

upper respiratory infection damages the mucous membrane and allows a downward colonisation by the organisms already present in the nose and throat. The writer is of the opinion that, rather than, or as well as, colonisation, there may be an aspiration into the lower air passages of the infected discharge in the nose and throat, while the young child is asleep. The influence noted by other writers of poor environment may be due to the fact that the child of the better class home who is having constant care and attention, has less chance of aspirating this infected discharge.

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and Cooper, G. (1930). Table 36, which is from Blacklock and Guthrie, (1933), quoted this reference amongst others. It has not been possible to trace the original reference.

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SUMMARY OF THE WRITER'S FINDINGS

Summary of the Writer's Findings.

(1) Amongst infants and children, in an open ward, cross infection by pneumococci occurred frequently. It did not constitute a danger to the children and did not lead to respiratory complications. It did not lead to any lengthening of the period in hospital as may occur with cross infection by Streptococcus pyogenes or Staphylococcus aureus. The writer did not find any clinical evidence to suggest that either a newly acquired respiratory infection or a recrudescence of the original respiratory infection was associated with the acquisition of a pneumococcus.

(2) Acquired infections with Streptococcus pyogenes or Staphylococcus aureus were frequently found amongst children in the open ward. Although in the majority of instances there was no clinical upset, in a definite proportion illness was caused by the acquisition of either of these organisms. Staphylococcus aureus was the organism most frequently acquired by patients while in hospital.

(3) The writer found evidence that Streptococcus pyogenes was transferred from individual to individual through the medium of the floor dust. In all probability, this was the method of transference, in the majority if instances.

(4) The writer found evidence to suggest that the pneumo-

coccus was transferred by means of the floor dust. Direct droplet infection, however, could not always be excluded.

(5) There was no evidence to suggest that Staphylococcus aureus was transferred by floor dust. The writer's findings suggested direct or mediate contact as the method of transference.

(6) It is suggested that the different methods of transference of these three organisms were, to some extent, due to the fact that each organism occurred particularly in one part of the nose or throat. Organisms such as the pneumococcus and Streptococcus pyogenes, which were found in the posterior nose and throat would be, by virtue of their site of occurrence most easily transferred by droplets which might infect the dust. Organisms in the anterior nose, for example, Staphylococcus aureus, would be transferred most easily by contagion.

(7) Chemotherapy did not prevent the acquisition of organisms in the nose or throat. During treatment, however, some organisms did disappear and this was most marked when a patient carrying Streptococcus pyogenes was treated by penicillin.

(8) There was little evidence to show that the nursing, medical or domestic staffs infected patients with pneumococci but there was evidence that they infected

them with Streptococcus pyogenes and Staphylococcus aureus.

(9) The staff, in their turn, apparently acquired streptococci, and on a few occasions, pneumococci, from the patients. Without the aid of bacteriophage typing it was impossible to prove that a member of staff ever acquired Staphylococcus aureus from a patient.

(10) Some conclusions are put forward as regards the etiology of pneumonia in the infant and young child. Staphylococcal pneumonia with empyema was looked upon as a distinct entity and is not considered here. In the older child, the acquisition of a type of pneumococcus, not already present in the upper respiratory tract, may cause the development of pneumonia with a radiological appearance similar to that in the adult. Apart from this, the association of the pneumococcus with pneumonia or bronchitis in the infant and young child would seem to be purely fortuitous. There was no evidence to suggest that either Staphylococcus aureus or Streptococcus pyogenes was responsible for more than a few of the instances of pneumonia in the infant or young child. It is suggested that the true nature of the disease process is an upper respiratory infection favouring an increase in the numbers of pneumococci and other organisms present in the nose

and throat, and a subsequent downward colonisation of the mucous membrane by these organisms causes the development of pneumonia or bronchitis. Alternatively, the upper respiratory infection may be followed by aspiration of infected discharge into the lower respiratory tract. The aspiration of secretions normally present in the mouth and throat may also occur during sleep in the infant, without any preceding upper respiratory infection and lead to the development of pneumonia. Even in the older child, where the picture may suggest the pneumococcal pneumonia of the adult, the assumption of pathogenic powers by pneumococcal types newly acquired in the nose or throat and usually innocuous, may be due to an allergic response on the part of the patient. A preceding respiratory infection may also be necessary and it is noteworthy that the incidence of respiratory infection amongst contacts is high.

Included amongst the pneumococci isolated from the patients, were some of intrinsically high virulence, for example, types 1 and 2. The patients from whom these types were isolated were all in the older age group. It is however possible that some other types are virulent, especially for young patients, and the writer found evidence that type 21 was one of these. It is suggested that types of

pneumococci, for example type 2, which are pathogens of pneumonia, are also more resistant to the drying conditions of the floor dust and therefore, more certain of being transferred from one individual to another.

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APPENDIX

APPENDIX.

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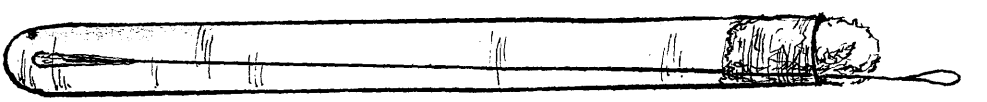
Figure 6.

THROAT SWAB

Figure 7.



PER-NASAL SWAB

(1) Methods of Swabbing and Dust Sampling.

(a) Taking the Throat Swab:

The swab used by the writer is depicted in Figure 6. Whilst the tongue was held down by a spatula the swab was inserted into the mouth and rubbed over the fauces, that is, over one tonsillar bed, over the back of the soft palate and over the other tonsillar bed. The swab was then replaced in its sterile glass container.

(b) Taking the Anterior Nose Swab:

The swab depicted in Figure 6 was also used for swabbing the anterior nose. The swab was inserted for about half an inch into one of the nostrils and rubbed round the walls of this nostril, using a circular movement.

(c) Taking the Posterior Nose Swab:

A per-nasal swab was used and this is depicted in Figure 7. The main characteristic of this swab was the fineness of the nicrome wire used, 32 gauge, and the smallness of the pledget of cotton wool carried by it. The swab was inserted through one nostril and passed along the floor of the nose until it reached the postnasal space. When the end of the swab impinged on the posterior wall of the naso-pharynx, it was turned quickly through 180 degrees and then rapidly withdrawn and replaced in its sterile glass container.

Showing blood agar plate in position and the direction of the jet of air which blows the dust onto the exposed plate

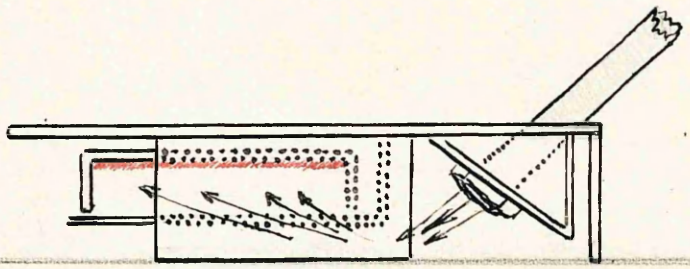


Figure 8.

PORTABLE DUST SAMPLER

This swab was described by Bradford and Slavin in 1940, and found by these authors and Miller, Leach, Saito and Humber in 1943 to be very successful in isolating Haemophilis pertussis from the postnasal space of patients suffering from whooping cough. Landsman 1953 and Anderson 1953 found it equally successful in isolating pneumococci from the post-nasal space.

(d) Taking the Sample of Floor Dust:

The dust sampler used was similar to that devised and constructed by Mr. T. Nash of the Central Public Health Laboratory, Colindale, London, and used in studies reported by Williams in 1949. Mr.McLean of Knightswood Hospital laboratory made the sampler used in the writer's investigations and a drawing of it is shown in Figure 8. The sampler, which was made in perspex, carried a petri dish containing blood agar on which the dust was collected. A jet of air was puffed on to the floor under the blood agar plate and the floor dust blown up on to the exposed surface of the blood agar plate. Six puffs had to be given to obtain a sufficient sample of dust. The plate was then removed, covered with its lid and a fresh plate inserted for the next dust sample.

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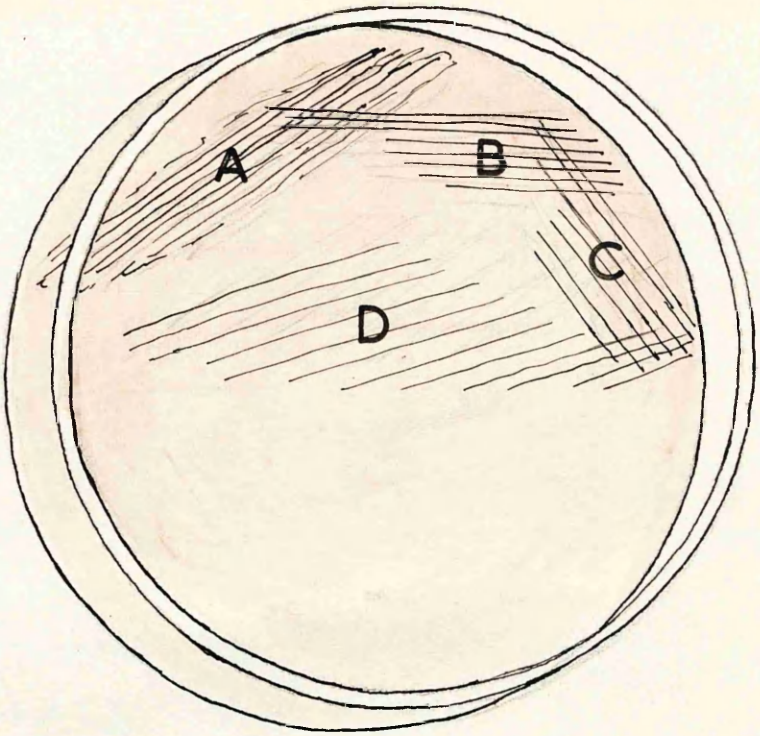
Figure 9.



Method of Plating:

The swab is first rubbed over area A. The platinum loop is sterilised in a flame and the plate inoculated as shown, using parallel strokes over area B. After sterilising once more, the loop is stroked over area C, then turned, and stroked over area D. In this way, a graded density of growth is obtained with the production of single colonies.

(2) Bacteriological Procedures with the Swabs and Dust Plates.

As soon as possible after being obtained, each swab was plated out on half of a blood agar plate, and on half of a similar plate containing 1:1,000,000 gentian violet. The blood agar were made by adding 7.5 millilitres of horse blood to 100 millilitres of melted nutrient agar. Plating was done by means of a platinum loop in such a fashion as to get a graded density of growth with the production of single colonies. The method is shown in Figure 9.

The plates were incubated overnight at 37° centigrade. Dust plates were also placed in the incubator without delay and incubated overnight at 37° centigrade.

(a) Procedure with Cultures obtained from Plating out the Swabs.

After overnight incubation, the culture plates were inspected through a low power lens. The blood agar plate showed the range of bacteria present. The gentian violet blood agar plate inhibited the growth of some organisms, especially staphylococci but allowed the growth of pneumococci and streptococci.

(i) The identification of Streptococcus pyogenes:

Small, translucent, convex and slightly granular colonies were recognised as those of Streptococcus pyogenes when each was surrounded by a clear zone due to the haemolysin produced by the organism, diffusing into the

blood agar. No further identification procedure was carried out. The approximate number of colonies present was noted. "Streptococcus pyogenes +++" referred to culture plates where the streptococcus was the predominant organism. The presence of a few scattered colonies was denoted as "Streptococcus pyogenes +" and of an intermediate number of colonies, as "Streptococcus pyogenes ++".

(ii) Identification of Staphylococcus aureus:

The colonies of Staphylococcus aureus were recognised as circular discs, considerably larger than the colonies of Streptococcus pyogenes. The colonies were opaque but glistening and moist and presented a golden yellow colour, some paler than others. The majority had a zone of complete haemolysis around each colony. The number of colonies present was noted in similar fashion to that used for Streptococcus pyogenes.

A considerable number of the staphylococci isolated were examined for coagulase production. Organisms, which were found to be coagulase positive, were also tested for penicillin sensitivity.

Method of Testing for Coagulase Production:

In the first instance, a simple slide test was used. (Cadness-Graves, Williams, Harper and Miles: 1943). A sweep from the staphylococcal colonies or a single colony if the growth was scanty, was suspended in a drop of saline

on a glass slide. To this heavy bacterial suspension, a drop of undiluted human plasma was added. There was almost immediate macroscopic clumping of the bacteria if the strain of Staphylococcus aureus being tested was coagulase positive.

The above method is efficient and simple, but may on occasion give a false negative result. To obviate this, any strain found to be negative by the slide test was further tested by the tube test. A sweep of the staphylococcal growth, or a single colony when the growth was scanty, was suspended in 5 millilitres of a nutrient broth and incubated overnight. Human plasma was diluted 1:10 with normal saline. To a test tube containing 1 millilitre of the diluted plasma, 10 drops of the broth culture were added. The tube was incubated for six hours at 37° centigrade, and coagulation noted (after Fisk: 1940). If the staphylococcus being tested was coagulase positive, the contents of the test tube clotted. As controls, broth cultures of a known coagulase negative and of a known coagulase positive staphylococcus were added to diluted plasma and incubated in similar fashion.

Method of Testing for Penicillin Sensitivity:

Each coagulase positive staphylococcus was tested for penicillin sensitivity. A broth culture, made as described for tube testing for coagulase production, was used and a drop of this plated onto half of a penicillin ditch plate.

The method used was that advised by Barber in 1947 and detects penicillinase production. The plate used was a blood agar plate from which a ditch had been cut. The ditch was filled with agar containing 10 units of penicillin per millilitre. Plating was done, using a platinum loop, in such a manner as to obtain a growth of graduated density from heavy to light. A standard penicillin sensitive, Oxford Staphylococcus was used as a control.

Growth was inhibited for half to one inch from the ditch if the strain was penicillin sensitive. Resistant strains grew up to and over the ditch. With penicillinase producing resistant strains, the growth grew over the ditch where it was heaviest but gradually became inhibited as the heaviness of the plating decreased. All resistant strains isolated by the writer were found to produce penicillinase.

Photographs of typical plates are shown in Figures 10a and 10b.

(iii) Identification of the Pneumococcus:

A photograph of typical colonies of pneumococci is shown in Figure 11. The colony is flat and smooth and often umbilicated. It is semi-transparent and often described as "dewdrop" like. The colonies on blood agar are each surrounded by a greenish zone said to be due to peroxide formation, resulting in the production of

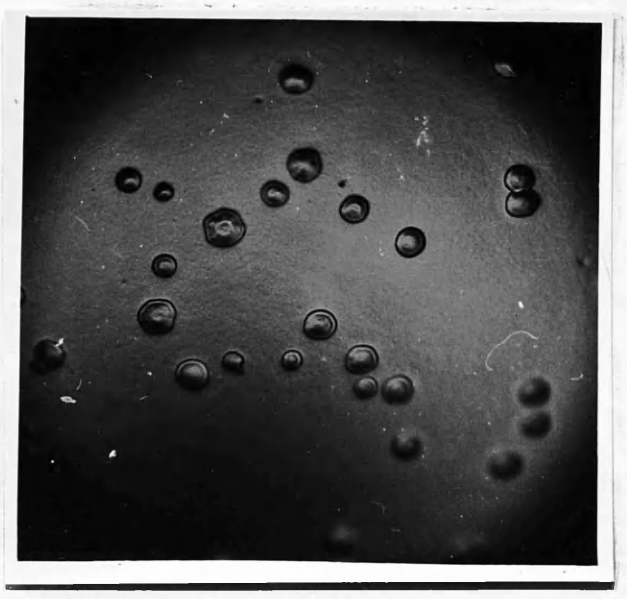
Figure 10a.

Photograph showing above, a penicillin sensitive and below, a penicillin resistant, penicillinase producing, Staphylococcus aureus. A standard penicillin sensitive, Oxford staphylococcus is shown.

Figure 10b.

Photograph showing two strains of penicillin resistant, penicillinase producing Staphylococcus aureus. A standard penicillin sensitive, Oxford staphylococcus is shown.

Figure II.

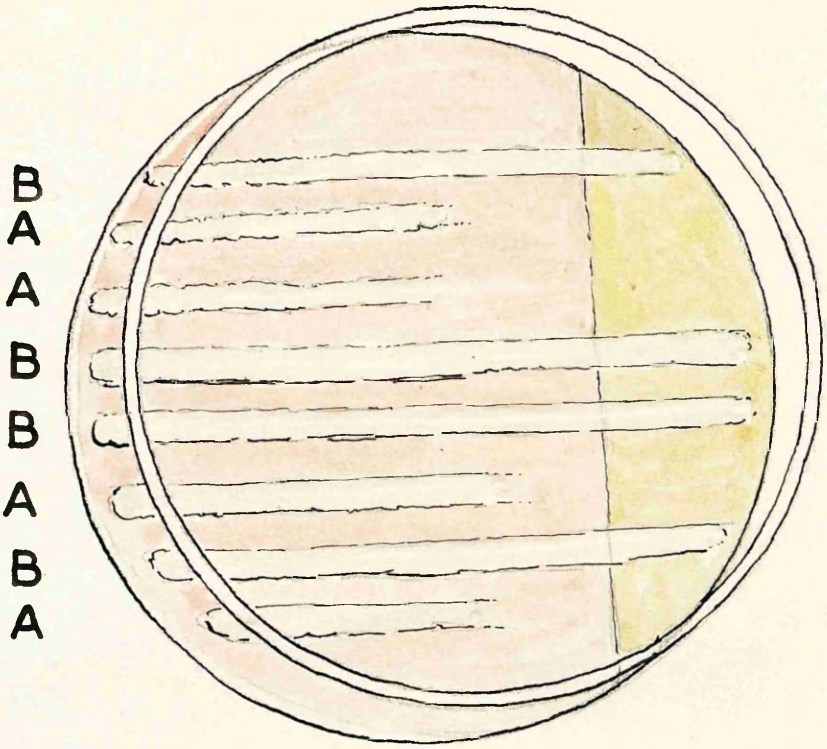


Photograph showing typical colonies of pneumococci on blood agar.

methaemoglobin. If colonies on the blood agar or gentian violet blood agar plates showed the cultural characteristics of the pneumococcus, they were subjected to a capsule test. If positive, they were directly typed. Colonies which were not quite typical, colonies resembling Streptococcus viridans, and colonies which had been tested for the presence of a capsule and found to be capsule negative were subcultured onto optochin (ethyl hydrocupreine) ditch plates and tested for sensitivity to this substance. Morgenroth and Levy in 1911 (quoted by Heffron, 1939) found that optochin had a specific bacteriocidal effect on pneumococci in test tube preparations. Mørch in Copenhagen (1943) found that in fact, optochin was bacteriostatic and she used it as a means of distinguishing pneumococci from other viridans colonies.

Optochin-sensitive organisms were considered to be pneumococci and, if capsule positive, were typed. If capsule negative, they were first subjected to mouse inoculation in an endeavour to induce capsule formation. On a very few occasions this did not occur and the pneumococcus could not be typed. Mouse inoculation was also carried out on any pneumococcus on which the first effort of typing was unsuccessful. Occasionally, a pneumococcus had to be inoculated into a mouse on more than one occasion if difficulty was experienced in typing

Figure 12.



Method of plating on optochin ditch plates:

A sweep from the pneumococcal growth to be tested is stroked across the plate using a platinum loop. Several strains were tested on one plate. The appearance of the resultant growth of an optochin sensitive organism is shown at A, and of an optochin resistant organism at B.

In the diagram, the blood agar is coloured pink, the optochin containing agar, yellow, and the growth of pneumococci white.

the pneumococcus or if the capsule formation was poor. Optochin sensitive, capsule negative strains which did not produce a capsule on mouse inoculation were considered to be "rough" strains.

Method of Testing for Presence of a Capsule:

This method was used in Knightswood laboratory and is a modification of Fleming's Nigrosin method (Mackie and McCartney, 1942). A drop of 1 per cent aqueous rose-bengal and a drop of 1 per cent aqueous nigrosin were mixed on a slide. A sweep from the pneumococcal colonies present on the culture plate was suspended in a drop of saline on the same slide. The two drops were mixed and the slide allowed to dry in air without heat and then examined under the microscope. The rose-bengal stained the organism and the nigrosin, the background. The capsule, if present, was unstained and appeared as a clear lacuna around the organism.

Method of Testing for Optochin Sensitivity:

Mørch (1943) used solid media containing optochin, or as a more delicate method broth containing optochin, to identify pneumococci. Anderson and Landsman (unpublished) at Knightswood Hospital used a modification of Mørch's method. A blood agar ditch plate was used, the ditch containing a 1:50,000 concentration of optochin in agar. Two or three sweeps of the organism to be tested were plated separately across the optochin ditch plate using a platinum loop. Organisms from several culture plates

could usually be tested on the one optochin ditch plate. Resistant strains grew up to and over the optochin ditch whereas the growth of sensitive strains was inhibited for a half to one inch from the ditch. Figure 12 indicates the method of plating and the appearances of sensitive and resistant strains.

Mouse Inoculation:

Intra-peritoneal injection of a broth suspension of the organism was used. The growth on the optochin ditch plate or the original growth on the blood agar, or gentian violet blood agar plate was utilised. If the original growth was not pure a subculture on blood agar was first made. A portion of the agar carrying the colonies of the organism was emulsified in 5 millilitres of broth in a virus mortar and 2 millilitres of the emulsion injected into the mouse by the intra-peritoneal route. If the strain of pneumococcus was mouse-virulent, as was usual, the mouse died within 24-48 hours. If they lived longer the mice were killed. An autopsy of the mouse was carried out. The skin was reflected to expose the abdominal and chest muscles. The peritoneal cavity was punctured by a sterile capillary pipette carrying a rubber teat and the peritoneal cavity washed out with 2 millilitres of broth. A capsule test was done on the peritoneal washings. If capsulated diplococci were seen, typing was carried out forthwith. If no such organisms were seen, a drop of the

peritoneal washings was plated on to a blood agar plate. The thoracic cage of the mouse was then opened and the heart exposed. A sterile capillary pipette was passed through the heart wall and the heart blood withdrawn. This also was plated on to the blood agar plate.

If the growth on the blood agar plate after overnight incubation was capsule positive, the pneumococcus was thereupon typed. If negative, further mouse inoculation was carried out. Occasionally the pneumococcus was lost if the mouse died from some other cause and occasionally the pneumococcus was overgrown by other organisms which were probably bowel contaminants. If such were the case, further mouse inoculation was carried out from the original culture.

Method of Typing the Pneumococcus:

The capsule of the pneumococcus becomes swollen when acted upon by its specific anti-serum. (Neufeld, 1902). This capsule swelling reaction forms the basis of the method of typing of the pneumococcus used by the writer.

The specific rabbit anti-sera used were obtained from the "State Serum Institute" in Copenhagen. Anti-sera for types 1 to 42 were available, but to facilitate typing, use was made of anti-sera, pooled into groups of which there were eight, labelled A to H. Type 3 anti-serum was not included in any of the groups and all pneumococci were tested with this anti-serum separately.

For the identification of type, a sweep of the pneumococcal colonies on the culture plate was used rather than a single colony. The reason for this was to obviate missing the presence of more than one type of pneumococcus. The pneumococci were tested with all the group anti-sera and type 3 anti-serum in the following manner. On each of three slides were placed three separate drops of saline and to each of these nine drops, a drop of 1 per cent aqueous methylene blue was added. The sweep from the culture plate was inoculated into each of the mixtures of saline and methylene blue. A drop of each group anti-serum was added to each preparation of the pneumococcus. To the ninth preparation was added type 3 anti-serum. Each preparation was covered by a glass cover slip, allowed to stand for five minutes and then examined under the microscope. All preparations were examined since, if there were more than one type present, capsule swelling would be present with more than one group anti-serum, unless all types present were in the one group. Capsule swelling was seen as a sharply outlined, unstained zone with a ground glass appearance, surrounding each blue-stained diplococcus.

Having thus assigned a pneumococcus to a group of types it was then tested with each individual anti-serum in the group for capsule swelling. Thus the type or types present were ascertained.

(b) Procedure with Dust Plates:

After overnight incubation, the dust plates were examined using a low power lens. Figure 13 shows photographs of two of these dust plates and depicts the usual range of colonies found.

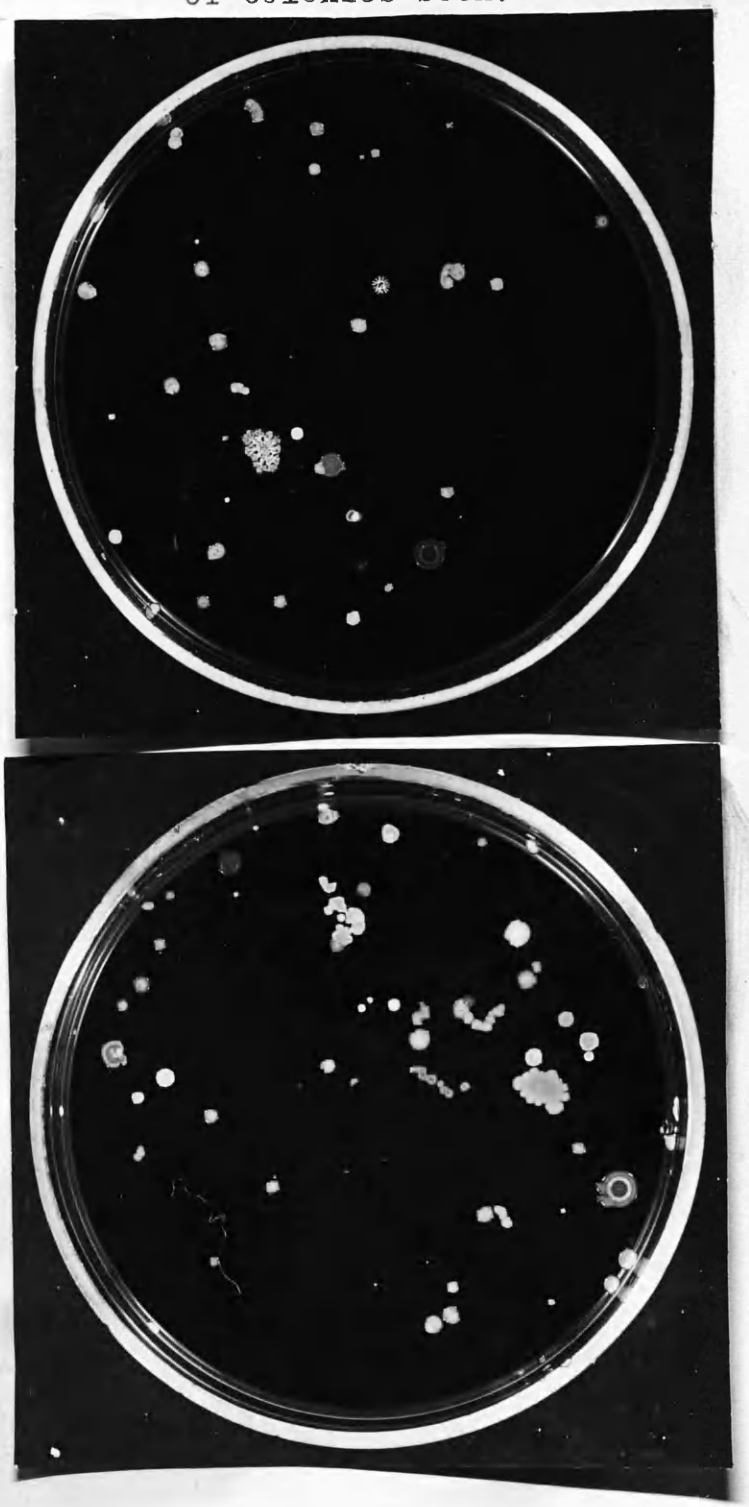
Colonies of Streptococcus pyogenes and Staphylococcus aureus were recognised by their typical naked eye appearance. A film was made of these colonies to assist in the identification of the organisms. If there was any doubt about the nature of any colony, this was subcultured and identified from the subculture by its cultural characteristics.

Typical colonies of pneumococci were seldom found and in order to detect the presence of pneumococci in the dust, any viridans producing colony, in any way resembling that of a pneumococcus, was tested for optochin sensitivity. If optochin sensitive and capsule positive the pneumococcus was thereupon typed. If capsule negative or if the capsule production was poor, mouse inoculation was first carried out and typing carried out on any pneumococci obtained from the peritoneal washings or the heart blood. The writer was fortunate in that on only one occasion did a capsule fail to appear after mouse inoculation of an optochin sensitive, capsule negative organism.

On a few occasions the dust plates were overgrown by a fungus, Bacillus proteus or subtilis, and the identification

Figure 13.

Photographs showing two typical dust culture plates and the usual range of colonies seen.



of the other organisms present was thus rendered impossible.

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3. Tables summarising the results of all swabbings.

3. Tables summarising the results of all swabbings.

A. Results of Swabbing the Patients.

B. Results of Swabbing the Members of Staff.

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Note: The type of pneumococcus is shown. The heaviness of the growth of Streptococcus pyogenes and of Staphylococcus aureus is indicated. If coagulase production and penicillin sensitivity tests were carried out, the results are also shown.

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ABBREVIATIONS

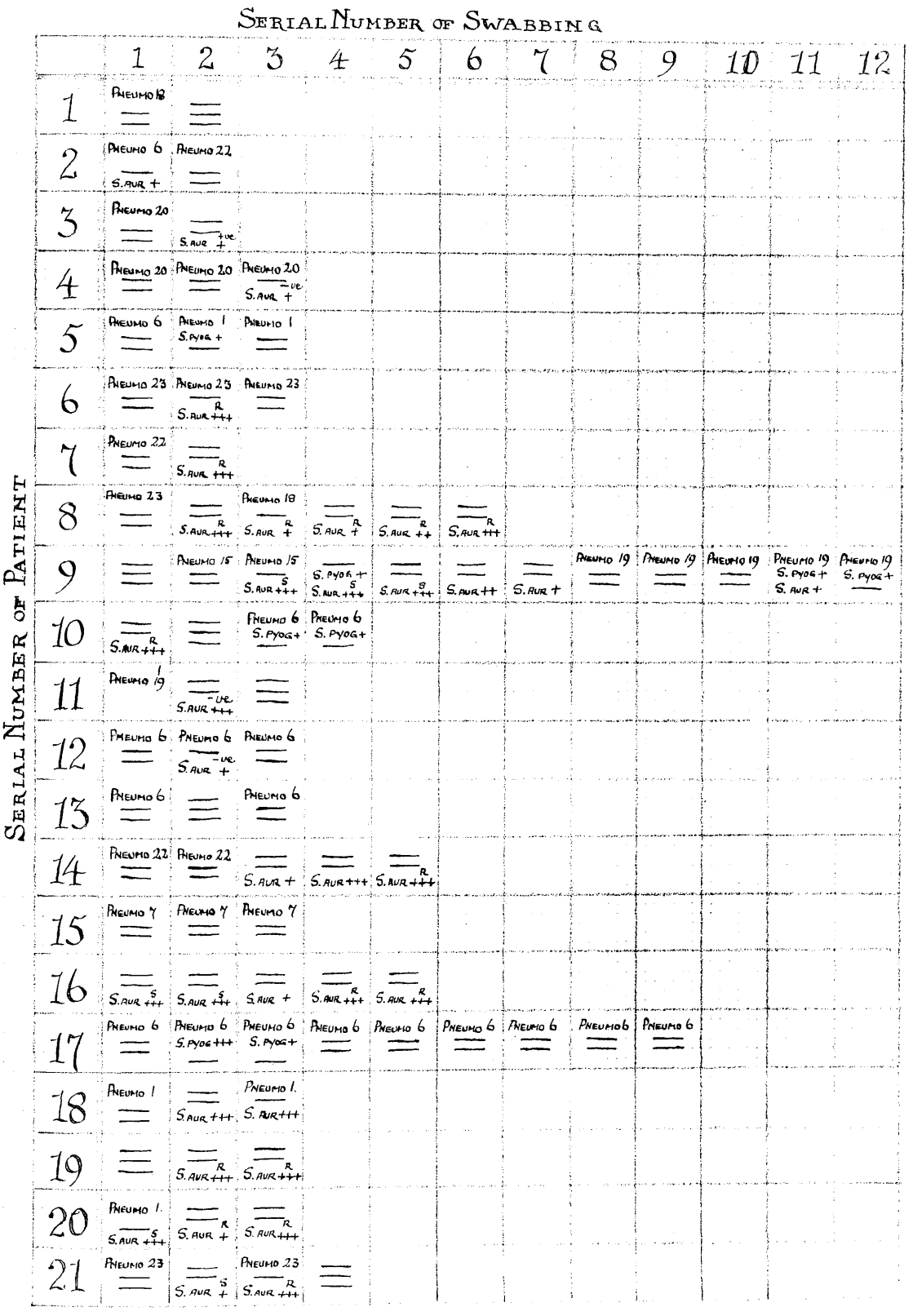
PNEUMO.	~	PNEUMOCOCCUS
S.PYOG.	~	STREPTOCOCCUS PYOGENES
S.AUR.	~	STAPHYLOCOCCUS AUREUS.
+	~	A FEW COLONIES ISOLATED
++	~	A MODERATE NUMBER OF COLONIES ISOLATED.
+++	~	NUMEROUS COLONIES ISOLATED.
+ve	~	COAGULASE POSITIVE } PENICILLIN SENSITIVITY NOT DETERMINED
−ve	~	COAGULASE NEGATIVE }
R	~	COAGULASE POSITIVE, PENICILLIN RESISTANT.
S	~	COAGULASE POSITIVE, PENICILLIN SENSITIVE

FOR EXAMPLE :

PNEUMO 6
S.PYOG ±
S.AUR +++

~ PNEUMO. TYPE 6 PRESENT, S.PYOG. PRESENT IN SMALL NUMBERS, S.AUR PRESENT IN LARGE NUMBERS, COAGULASE POSITIVE, PENICILLIN RESISTANT.

A. Results of Swabbing the Patients



A. CON'D.

SERIAL NUMBER OF SWABBING

SERIAL NUMBER OF PATIENT

	1	2	3	4	5	6	7	8	9	10	11	12
22												
23	PNEUMO 23	PNEUMO 23 / S. AUR R ++	PNEUMO 23 / S. AUR R +++									
24		PNEUMO 2	PNEUMO 4									
25	PNEUMO 19	PNEUMO 6	PNEUMO 6									
26	PNEUMO 6		PNEUMO 6									
27	PNEUMO 21		PNEUMO 21									
28	PNEUMO 23	PNEUMO 23	PNEUMO 23 / S. AUR -VE +++									
29	PNEUMO 34	S. AUR R ++	PNEUMO 34 / S. AUR R +++	S. AUR +++								
30		PNEUMO 7	PNEUMO 33									
31	S. PYOG +		PNEUMO 6 / S. AUR +									
32			S. AUR +									
33	PNEUMO 19	PNEUMO 19 / S. AUR +++	PNEUMO 19 / S. AUR +									
34		S. AUR R +++	PNEUMO 4 / S. AUR +									
35	PNEUMO 14 / S. PYOG +	PNEUMO 14 / S. AUR +++	PNEUMO 14	PNEUMO 14 / S. PYOG +								
36	S. PYOG +	PNEUMO 19 / S. AUR R +++	S. AUR R +++	PNEUMO 21 / S. AUR +	S. AUR R +++	PNEUMO 21						
37	PNEUMO 5 23 / S. PYOG + / S. AUR +++		S. AUR +++	S. AUR +++	PNEUMO 4	PNEUMO 4 / S. AUR S +++	PNEUMO 4					
38	PNEUMO 21	PNEUMO 21 / S. AUR +	S. AUR +		PNEUMO 7 / S. PYOG +							
39	PNEUMO 7	PNEUMO 4	PNEUMO 4 / S. AUR R +++	PNEUMO 4 / S. AUR +	PNEUMO 4	PNEUMO 4 23	PNEUMO 4	S. AUR -VE +				
40	PNEUMO 19 / S. PYOG +											
41	PNEUMO GR. C. / S. PYOG +	PNEUMO 7	PNEUMO 24 / S. PYOG ++									
42	PNEUMO 19 / S. PYOG +	PNEUMO 19	PNEUMO 19	PNEUMO 19 / S. PYOG +	PNEUMO 19 / S. PYOG ++							
43	PNEUMO 6 22	PNEUMO 6 / S. AUR +	PNEUMO 6									

SERIAL NUMBER OF SWABBING

SERIAL NUMBER OF PATIENT

	1	2	3	4	5	6	7	8	9	10	11	12
44	Pneumo 19	Pneumo 19	Pneumo 19	Pneumo 19	Pneumo 19							
45	Pneumo 6 S. pyog +	Pneumo 6	Pneumo 6	Pneumo 6	Pneumo 6	Pneumo 6 S. pyog +						
46	Pneumo 1/14		Pneumo 23 S. aur +									
47			Pneumo 6/22 S. aur +									
48	S. aur ++	Pneumo 4	Pneumo 4									
49	Pneumo 23	Pneumo 9		Pneumo 23	Pneumo 23	Pneumo 10 S. pyog + S. aur +	Pneumo 10 S. aur +++					
50	S. aur 6/++											
51	Pneumo 23 S. aur +	Pneumo 23 S. aur +	Pneumo 23 S. aur +									
52	Pneumo 6	Pneumo 6										
53	S. aur +			S. aur R/+	S. aur R/++	S. pyog +	Pneumo U.T.T. S. pyog +	S. aur ++				
54	Pneumo 23 S. aur +		Pneumo 16 S. aur +++									
55	Pneumo 6 S. aur +	Pneumo 6	Pneumo 6 S. aur +++	Pneumo 19 S. pyog +	Pneumo 19	Pneumo 19	Pneumo 19	Pneumo 19	Pneumo 19	Pneumo 19	Pneumo 19	
56	S. aur +	S. aur ++										
57	Pneumo 12 S. aur +											
58	S. aur R/+++	Pneumo 42		S. pyog + S. aur R/+++								
59	S. aur ++	Pneumo 10/23	Pneumo 10									
60	S. aur ++		Pneumo 23									
61	Pneumo 11 S. aur +	Pneumo 11										
62		S. aur +++										
63	Pneumo 19 S. aur +											
64	Pneumo 21 S. aur ++		Pneumo 15 R/S. aur +++	Pneumo 15 S. pyog +	Pneumo 19 S. pyog + S. aur +							
65												
66			Pneumo 6									

SERIAL NUMBER OF SWABBING

SERIAL NUMBER OF PATIENT

	1	2	3	4	5	6	7	8	9	10	11	12
67	Pneumo 6/19	Pneumo 6	Pneumo 6 S. pyog +	Pneumo 6 S. pyog +								
68	Pneumo 19	Pneumo 19	Pneumo 6/19	Pneumo 19 S. pyog + S. aur +++	Pneumo 19 S. pyog +							
69	Pneumo 23	Pneumo 23	Pneumo 23									
70	Pneumo 23	Pneumo 23 S. aur ++	Pneumo 23	Pneumo 23 S. aur +	Pneumo 23	Pneumo 23						
71	Pneumo 6/23	Pneumo 6	Pneumo 6									
72		S. aur +++	S. aur ++	S. aur +								
73	Pneumo 6	Pneumo 23										
74	Pneumo 23	Pneumo 23										
75	S. pyog +											
76	Pneumo 33	Pneumo 6/33	Pneumo 6									
77	Pneumo 23		S. aur R ++									
78	Pneumo 19/23	Pneumo 23	Pneumo 23 S. aur +									
79		Pneumo 33 S. aur +										
80	S. pyog +		Pneumo 23 S. aur +++									
81		S. aur R +++	S. aur +++									
82	Pneumo 11	Pneumo 11 S. aur R +++	Pneumo 17 S. aur R +++									
83		Pneumo 17	Pneumo 21									
84		S. pyog + -ve S. aur +++	-ve S. aur +++	Pneumo 6 S. aur +	Pneumo 6 S. pyog +	R S. aur +++	R S. aur +++	R S. aur +	R S. aur +	R S. aur +++	Pneumo 11 R S. aur ++	Pneumo 11 S. pyog + R S. aur +++
85	Pneumo 2	Pneumo 2		Pneumo 2 S. pyog +		Pneumo 2 S. pyog + S. aur +				13	14	15
86	Pneumo 29	S. aur +	S. pyog +++ S. aur +	Pneumo 29					84	Pneumo 11 S S. aur +++	S. pyog + S. aur +++	Pneumo 11 R S. aur +++
87	S. pyog + S. aur +		S. pyog +									
88	S. aur +	Pneumo 22 S. pyog ++ S. aur +	R S. aur +++	Pneumo 22 S. aur +								

SERIAL NUMBER OF SWABBING

SERIAL NUMBER OF PATIENT

	1	2	3	4	5	6	7	8	9	10	11	12
89												
90	PNEUMO 21	PNEUMO 22 S. AUR +	S. AUR R +++	S. AUR R +++	S. PYOG +		S. PYOG +	PNEUMO U.T.T. S. PYOG +		PNEUMO U.T.T. S. AUR +		
91		PNEUMO 7	PNEUMO 7 21	PNEUMO 7 S. PYOG +								
92		S. PYOG +	PNEUMO 10									
93	PNEUMO 8 S. PYOG +	PNEUMO 8 S. AUR +	PNEUMO 8									
94	PNEUMO 14 23	PNEUMO 6	PNEUMO 23	PNEUMO 14	PNEUMO 23							
95	PNEUMO 6	PNEUMO 6 S. AUR +										
96												
97	PNEUMO 21	PNEUMO 21	PNEUMO 21 S. AUR R +	PNEUMO 21								
98	S. PYOG + S. AUR S +	PNEUMO 6 S. PYOG +	S. PYOG +++	S. PYOG +++	S. PYOG ++		S. PYOG +++					
99	PNEUMO U.T.T. S. PYOG +	S. AUR +	PNEUMO 6	PNEUMO 6								
100	PNEUMO 31	PNEUMO 15 S. AUR +	PNEUMO 10	PNEUMO 15	PNEUMO 15	PNEUMO 15						
101		S. AUR S +					S. PYOG +					
102			S. PYOG +									
103			S. AUR R +++									
104		PNEUMO 23	PNEUMO 23	PNEUMO 23 S. PYOG + S S. AUR +++	PNEUMO 23	PNEUMO 23						
105	PNEUMO 12	PNEUMO 12 S. AUR R ++										
106	S. PYOG +	PNEUMO 6										
107	PNEUMO 11 S. PYOG + S. AUR +	PNEUMO 33	PNEUMO 33 S. PYOG +	PNEUMO 33 S. AUR +++	S. AUR +							
108	PNEUMO 23 S. PYOG +	PNEUMO 23 S. PYOG +	PNEUMO 23 S. AUR +									
109	PNEUMO 8 14	PNEUMO 14	PNEUMO 14 22									
110			PNEUMO 4 S. AUR +++									

Serial Number of Swabbing

Serial Number of Patient	1	2	3	4	5	6	7	8	9	10	11	12
111	Pneumo 14 S. Pyog +		Pneumo 14	Pneumo 14	Pneumo 14	Pneumo 14	Pneumo 14					
112	S. Pyog +	S. Aur + (-ve)	S. Aur + (-ve)									
113	Pneumo 33	Pneumo 33	Pneumo 33 S. Pyog +	Pneumo 33								
114	S. Aur R +++		Pneumo 33	Pneumo 33	Pneumo 33	Pneumo 33 S. Pyog + S. Aur +++	Pneumo 33 S. Aur S +					
115												
116	Pneumo 11	Pneumo 11										
117												
118	Pneumo 10	S. Aur R +++										
119	Pneumo 21	S. Aur +++										
120	Pneumo 19 S. Pyog +	S. Pyog + S. Aur +++		S. Aur R +++	S. Aur R +++	S. Aur +++						
121	Pneumo 11 S. Pyog +			S. Aur S +++								
122												
123	Pneumo 14	Pneumo 14	Pneumo 14									
124	Pneumo 6 S. Pyog +	S. Pyog ++ S. Aur R +++	S. Pyog + S. Aur R +++									
125		S. Aur +										
126	Pneumo 11 S. Pyog +											
127	Pneumo 18		S. Aur +									
128	Pneumo 18		Pneumo 18	Pneumo 18 S. Pyog ++								
129	Pneumo 6	S. Aur R +++	S. Aur +++	S. Aur R +++								
130		S. Aur S +++										
131	S. Pyog +	S. Aur S +++										

B. Results of Swabbing the Members of Staff.

Serial Number of Swabbing

Serial Number of Member of Staff.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
1	S. AUR +	S. AUR +++	Pneumo 10 S. AUR +++	S. AUR +++	S. AUR +++ R		S. AUR +++ S	S. AUR +++	Pneumo 6 S. PYOG + S. AUR +++ S	S. AUR ++ S													
2		S. AUR +++	S. AUR ++ -UE																				
3			S. PYOG ++ -UE S. AUR —	S. PYOG +	S. PYOG + S. AUR +	S. PYOG +++ S. AUR +++ S	S. PYOG + S. AUR ++ S	S. PYOG + S. AUR + S																
4			S. AUR +++ S	S. AUR +++ R	S. AUR ++ S	S. AUR +++ R	S. AUR ++	S. AUR	S. AUR +++	S. AUR +++	S. AUR +++ R	S. PYOG + -UE S. AUR +	S. AUR ++										
5	S. AUR +	S. AUR +++		S. PYOG +																			
6			S. AUR ++	S. PYOG + -UE S. AUR +++	S. AUR ++ R	S. AUR ++ -UE			S. AUR +			S. PYOG +			Pneumo 22 S. AUR +++ R	Pneumo 22 S. AUR +++ R	S. PYOG + S. AUR + R						
7	S. PYOG ++	S. PYOG + S. AUR +++ R	S. AUR -UE +++		S. PYOG + S. AUR +																		
8					S. AUR -UE +++	S. PYOG + S. AUR -UE ++	S. AUR -UE +++		S. AUR +			S. PYOG + S. AUR +	S. PYOG +	S. PYOG +	S. AUR +	S. PYOG +	S. PYOG + S. AUR + R		S. PYOG +				
9	Pneumo 2				S. PYOG +	S. PYOG +			Pneumo 2 S. PYOG +	Pneumo 29	Pneumo 29 S. PYOG ++	S. PYOG +			S. PYOG +		S. PYOG +		S. PYOG +		S. PYOG +		S. PYOG +
10	S. PYOG +		S. PYOG +	S. PYOG + S																			
11			Pneumo 6					Pneumo 18		S. AUR +	Pneumo 18 S. PYOG +												
12		Pneumo 14	Pneumo 14	Pneumo 14 S. PYOG +	Pneumo 14 S. AUR +	Pneumo 14 S. AUR ++																	
13		S. PYOG +++ S. AUR + R	S. PYOG ++ S. AUR ++ R	S. PYOG + S. AUR +++ R	S. PYOG +++ S. AUR ++	S. PYOG +++ S. AUR +	S. AUR +	Pneumo 6 S. PYOG + S. AUR +	S. AUR +++	S. PYOG + S. AUR ++ R	S. AUR -UE +												
14	Pneumo 6 S. AUR +	Pneumo 6		Pneumo 6 S. AUR + R	Pneumo 6 S. AUR -UE ++	Pneumo 6 S. AUR -UE +++	Pneumo 6 S. AUR +	Pneumo 6 S. AUR +++	Pneumo 6 S. PYOG +	Pneumo 6 S. PYOG +	Pneumo 6	S. AUR +++ R	Pneumo 6 S. PYOG +	S. PYOG +++ S. AUR ++ R									
15	S. AUR -UE +++	S. AUR +++ S	S. AUR +++ R	S. AUR + S	S. AUR ++ S	Pneumo 23																	
16	S. AUR ++ S																						
17			Pneumo 15				Pneumo 23		Pneumo 23	S. PYOG +	S. PYOG +												
18		Pneumo 3 S. PYOG +			Pneumo 6																		
19																							

B. CON'D.

SERIAL NUMBER OF SWABBING

SERIAL NUMBER OF MEMBER OF STAFF.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
20	S. AUR ++ R	S. AUR +++	S. AUR +++ S	S. AUR +	S. PYOG + S. AUR +- inc	S. PYOG + S. AUR +++ S		S. AUR + S	S. AUR +++ R	S. AUR +++		S. AUR + S											
21	S. AUR ++ -inc	S. PYOG + S. AUR +++ -inc																					
22	S. AUR + S R	S. AUR +++ R		S. AUR ++	S. AUR +++	S. AUR +++ R																	
23	S. PYOG ++			PNEUMO 11 S. AUR +																			
24	PNEUMO 6 S. AUR + -inc		PNEUMO 22	PNEUMO 22	S. AUR ++ R	S. PYOG + S. AUR ++ -inc	S. AUR +++ S	PNEUMO 35 S. PYOG +	PNEUMO 35 S. PYOG +	S. PYOG +	PNEUMO 35												
25		S. PYOG +			S. PYOG +																		
26	PNEUMO 5	S. AUR +++ R	S. PYOG + S. AUR +++ S	S. AUR +++ S	S. PYOG + S. AUR +++ R	S. AUR +++ R	S. AUR +++ R	S. AUR +++ S															
27	PNEUMO 3																						
28	S. AUR + S	S. PYOG + S. AUR +	PNEUMO 35 S. PYOG +++ S. AUR +++ -inc	S. PYOG + S. AUR +++ R																			
29	PNEUMO 10																						
30	S. AUR +++																						
31		PNEUMO 34	PNEUMO 34 S. AUR ++	PNEUMO 34 S. AUR + S		PNEUMO 34																	
32	S. AUR +	S. AUR +++ R																					
33	PNEUMO 19 S. AUR +++ R	PNEUMO 2 S. AUR +++ R	PNEUMO 2 14 S. AUR +	PNEUMO 2 S. AUR + R		S. AUR +++ R	S. AUR +++ R	S. AUR +++ R	PNEUMO 2 S. AUR + R														
34	PNEUMO 10 S. AUR +++ R																						
35	S. AUR +	S. AUR + R		PNEUMO 20	S. AUR +++ S		S. AUR ++																
36	S. PYOG ++	S. PYOG + S. AUR +++	S. PYOG + S. AUR ++ S	S. PYOG +++	S. PYOG +++ S. AUR +++ R	S. PYOG +																	
37	S. PYOG + S. AUR + S	S. PYOG + S. AUR + S		S. AUR +++ S	S. AUR + S	S. AUR +++ S																	
38	S. PYOG +++ S. AUR +++																						
39	S. PYOG +++ S. AUR +++ S	S. AUR ++ S	S. PYOG + S. AUR +++ R																				
40	S. PYOG +	S. PYOG +																					
41																							
42	PNEUMO GR. F.																						

4. Summary of Findings for Section 2, Chapter 1.

Summary of Findings when there was Clinical Evidence of Cross-infection.

(Note:- temperature figures are in degrees Fahrenheit)

A. No associated change in the naso-pharyngeal flora.

Serial No. of Patient.	Nature of original illness.	Clinical Manifestations indicating Relapse or an Acquired Infection.	Results of Swabbing Nose and Throat.
14	Pneumonia.	On 4th day, patient became very ill with elevation of temperature to 104.8°. Treatment changed:- satisfactory result.	No change.
16	Pneumonia.	On 7th day, cough much increased, temperature 99.4°. Treatment changed: satisfactory result.	No change.
23	Severe Bronchitis.	On 14th day, elevation of temperature to 100°. No change in clinical condition.	No change.
36	Pneumonia.	On 17th day, gastro-enteritis developed, temperature elevated to 102°.	No change.
49	Pneumonia.	On 26th day, elevation of temperature to 100°. Complained of pain in throat. Contact of scarlet fever.	No change. A few S.pyog. found in throat 9 days later.
55	Collapse left lower lobe, probably tuberculous.	Ears started to discharge in 8th week of hospitalisation.	No change.

A. (Contd.)

Serial No. of Patient.	Nature of original illness.	Clinical Manifestations indicating Relapse or an Acquired Infection.	Results of Swabbing Nose and Throat.
71	Mild Bronchitis.	On 12th day, elevation of temperature to 99.2°. Throat was injected. Contact of scarlet fever.	No change.
91	Pneumonia.	Symptoms of gastro-enteritis developed.	No change.
108	Pneumonia.	On 8th day, otitis media developed. Treatment changed: satisfactory result.	No change.
125	Pneumonia.	On 17th day, elevation of temperature to 99.4°. No change in clinical condition.	No change.

B. Virtually no change in the naso-pharyngeal flora.

Serial No. of Patient.	Nature of original illness.	Clinical Manifestations indicating Relapse or an Acquired Infection.	Results of Swabbing Nose and Throat.
3	Pneumonia.	On 6th day, elevation of temperature to 101°. No change in clinical condition.	A few S.aur. had been acquired in P.N. and A.N.
4	Severe Bronchitis.	On 13th day, elevation of temperature to 102°. No change in clinical condition.	A few S.aur. had been acquired in A.N. Organisms coag.+ve; pen. sens.
38	Pneumonia.	On 9th day, patient became ill and breathless. Chest very moist.	A few S.aur. had been acquired in A.N.
108	Pneumonia.	On 19th day, elevation of temperature to 99°. No change in clinical condition.	A few S.Aur. had been acquired in A.N.

C. Pneumococcus acquired.

Serial No. of Patient.	Nature of original Illness.	Clinical Manifestations indicating Relapse or an Acquired Infection.	Results of Swabbing Nose and Throat.
99	Severe Bronchitis.	On 9th day, elevation of temperature to 101°. No change in clinical condition.	Type 6 pneumo. had been acquired in A.N. and persisted in later swabbings.
104	Pneumonia.	On 3rd day, throat injected, a little exudate on tonsils.	Type 23 pneumo. had been acquired in P.N., A.N. and T. and persisted in later swabbings.

D. Streptococcus pyogenes acquired.

Serial No. of Patient.	Nature of original Illness.	Clinical Manifestations indicating Relapse or an Acquired Infection.	Results of Swabbing Nose and Throat.
9	Primary Tuberculosis Complex.	Patient developed pain in throat, elevation of temperature to 99.8°, after the 10th swabbing. Tonsils injected and enlarged. Contact of scarlet fever.	On 11th swabbing, S.pyog.+ found to be present in throat for first time.
17	Pneumonia (left base)	X-ray on 14th day showed consolidation in right mid zone and left basal consolidation had resolved. On 3rd and 4th days temperature was elevated.	On 8th day patient acquired S.pyog +++ in throat. Still present on 16th day.
42	Primary Tuberculosis Complex.	On 27th day, patient developed scarlet fever.	On 27th day, patient had acquired S. pyog.+++ in throat. S.pyog. + had been isolated on 2 occasions before.

D. (Contd.)

Serial No. of Patient.	Nature of original Illness.	Clinical Manifestations indicating Relapse or an Acquired Infection.	Results of Swabbing Nose and Throat.
55	Collapse left lower lobe, probably tuberculous.	On 23rd day, elevation of temperature. Throat injected, exudate on tonsils. Contact of scarlet fever.	On 23rd day, patient had acquired S.pyog.+

E. Staphylococcus aureus acquired.

Serial No. of Patient.	Nature of original Illness.	Clinical Manifestations indicating Relapse or an Acquired Infection.	Results of Swabbing Nose and Throat.
8	Pneumonia. Fibrocystic disease of pancreas.	On 10th day, relapse in clinical condition associated with high temperature. Had intramuscular penicillin until 7th day.	On 8th day, patient acquired S. aur.+++ in A.N. and P.N. Organisms were coag. +ve; pen.res. S. aur.+++ still present on 10th day with a newly acquired type 18 pneumo. Lung puncture:-S.aur. in pure growth; coag +ve, pen.res.
9	Primary Tuberculosis. Complex.	Two weeks after admission temperature elevated. Many septic spots present. No previous treatment.	S.aur +++had been acquired in A.N. Organisms were coag +ve, pen.sens.
29	Pneumonia.	On 6th day temperature elevated to 100°. No change in clinical condition. Receiving treatment by oral penicillin.	S.aur.+++ had been acquired in P.N., A.N. & T. Organisms were coag. +ve, pen.res.

E. (Contd.)

Serial No. of Patient.	Nature of original Illness.	Clinical Manifestations indicating Relapse or an Acquiring Infection.	Results of Swabbing Nose and Throat.
107	Herpes Labialis.	In 3rd week, septic lesions became superimposed on a gross herpetic infection.	S.aur.+++ had been acquired in P.N. and A.N.
120	Pneumonia.	On 22nd day clinical condition relapsed, no elevation of temperature. Treatment by I.M. penicillin and sulphadiazine had been discontinued on 7th day.	S.aur.+++ had been acquired in P.N. and A.N. on 19th day. Organisms were coag.+ve. pen.res. Still present on 22nd day. On 8th day S.aur.+++ was present but not tested for pen. sensitivity.

Abbreviations:

P.N. = Posterior nose.

A.N. = Anterior nose.

T. = Throat.

\+ = A small number of organisms.

+++ = A large number of organisms.

Coag.+ve = Coagulase positive.

Coag.-ve = Coagulase negative.

Pen.sens. = Penicillin sensitive.

Pen.res. = Penicillin resistant.

Pneumo. = Pneumococcus.

S.pyog. = Streptococcus pyogenes.

S.aur. = Staphylococcus aureus.

5. Summary of Findings for Section 2, Chapter 2.

5) Summary of Findings for Section 2, Chapter 2.

A. Acquisition of fresh pneumococcal types by ward patients.

B. Acquisition of fresh pneumococcal types by members of staff.

C. Acquisition of streptococcus pyogenes by ward patients.

D. Acquisition of streptococcus pyogenes by the members of staff.

* * * * * * *

SUMMARY OF FINDINGS

A). Acquisition of fresh pneumococcal types by ward patients.

Patients acquiring one type of Pneumococcus only.

Serial Number of Patient.	Type of Pneumo-coccus acquired.	Week when pneumo-coccus acquired.	Diagram.
2	22	2	15
5	1	2	1
8	18	3	4
10	6	5	19
25	6	6	19
31	6	8	19
34	4	8	17
37	4	10	17
38	7	11	12
46	23	9	18
48	4	8	17
53	U.T.T.	14	-
54	16	10	6
55	19	11	14
58	42	9	8
60	23	10	18
66	6	11	19
68	6	11	19

Patients acquiring one type of Pneumococcus only. (Contd.)

Serial Number of Patient.	Type of Pneumo-coccus acquired.	Week when Pneumo-coccus acquired.	Diagram.
73	23	11	18
76	6	11	19
79	33	11	16
80	23	12	18
82	17	13	9
88	22	13	15
92	10	5	13
94	6	14	19
98	6	17	19
99	6	17	19
104	23	17	18
106	6	19	19
107	33	19	16
109	22	21	15
110	4	21	17
114	33	22	16

Patients acquiring two types of Pneumococcus.

Serial Number of Patient.	Type of Pneumo-coccus acquired.	Week when Pneumo-coccus acquired.	Diagram.
9	15	3	10
	19	9	14
24	2	6	2
	4	7	17
30	7	6	12
	33	7	16
36	19	7	14
	21	8	11
39	4	7	17
	23	11	18
41	7	8	12
	24	9	7
47	6	9	19
	22	9	15
49	9	8	5
	10	12	13
59	10	9	13
	23	9	18
64	15	11	10
	19	14	14

Patients acquiring two types of Pneumococcus. (Contd.)

Serial Number of Patient.	Type of Pneumo-coccus acquired.	Week when Pneumo-coccus acquired.	Diagram.
83	17	12	9
	21	13	11
84	6	15	19
	11	22	3
91	7	14	12
	21	15	11
100	15	17	10
	10	18	13

Summary of results of tracing the source of the acquired type of pneumococcus to the other patients or to the members of staff.

Group 1. Source traced to patient in adjacent cot.

Group 2. Source traced to a patient in the same division of the ward, but not in adjacent cot.

Group 3. Source traced to a patient in another division of the ward.

Group 4. Source could not be traced to any of the patients in the ward.

Group A. Source could be traced to a member of staff.

Group B. Source unlikely to be a member of staff, but not impossible.

Group C. Source could not be traced to any of the members of staff.

Number.	Diagram No.	Serial No. of Patient.	Type acquired.	Week of acquisition.	GROUPS							REMARKS
					Patients				Staff			
					1	2	3	4	A	B	C	
1	1	5	1	2				*			*	
2	2	24	2	6				*		*		Staff No.9 in contact with the patient. Staff No.9 carried type 2 in Week 1 and again in Week 9.
3	3	84	11	22	*						*	Patient No.116 in the adjacent cot carried type 11 in Weeks 21 and 22.
4	4	8	18	3	*						*	Patient No.1 was in the adjacent cot for 1 day in Week 2. Patient No.1 carried type 18 in Week 1.
5	5	49	9	8				*			*	
6	6	54	16	10				*			*	

Number.	Diagram No.	Serial No. of Patient.	Type acquired.	Week of acquisition.	GROUPS							REMARKS
					Patients				Staff			
					1	2	3	4	A	B	C	
7	7	41	24	9				*			*	
8	8	58	42	9				*			*	
9	9	82	17	13			*				*	Patient No.83 in the adjacent division carried type 17 in Week 12. Perhaps No.82 and 83 were infected from the same unknown source.
10	9	83	17	12				*			*	
11	10	9	15	3				*			*	
12	10	64	15	11				*			*	
13	10	100	15	17				*			*	
14	11	36	21	8			*				*	Patients Nos.27 and 38, in divisions B & C respectively, carried type 21 in Week 8. Type 21 was also carried by No.38 in Week 7.
15	11	83	21	13				*			*	
16	11	91	21	15			*				*	Patient No.97 carried type 21 when admitted to the adjacent division. Patient No.91 acquired type 21 two days after No.97 was admitted.
17	12	30	7	6	*						*	Patient No.15 carried type 7 in Week 5. Although in same division as Patient No.30, he was dismissed the day after that on which No.30 was admitted.
18	12	38	7	11				*			*	

Number.	Diagram No.	Serial No. of Patient.	Type acquired.	Week of acquisition.	GROUPS Patients 1	2	3	4	Staff A	B	C	REMARKS
19	12	41	7	8	*						*	Patient No.30 was in the adjacent cot to No.41 in Week 7 for 4 days. Type 7 was carried by No.30 in Week 6 but not in Week 7.
20	12	91	7	14				*			*	
21	13	49	10	12				*		*		Staff No.29 was in contact with the patient but was swabbed only once (in Week 11) when he carried type 10. Therefore it is possible that he carried type 10 in Week 12.
22	13	59	10	9				*		*		Staff No.29 was in contact with the patient but was swabbed only once (in Week 11) when he carried type 10. Therefore it is possible that he carried type 10 in Week 9.
23	13	92	10	15				*			*	Staff No.29 still in occasional contact with the patients although not being swabbed.
24	13	100	10	18				*			*	Staff No.29 still in occasional contact with the patients although not being swabbed.
25	14	9	19	9	*						*	Patient No.44 in the adjacent cot carried type 19 in Weeks 7, 8 and 9. Patient No.42 in the same division also carried type 19 in Weeks 7, 8 and 9.
26	14	36	19	7			*				*	Patient No.33 in the adjacent division carried type 19 in Weeks 6 and 7. Patients Nos. 40 and 44 in the other adjacent division carried type 19 in Weeks 6 and 7 respectively.

Number.	Diagram No.	Serial No. of Patient.	Type acquired.	Week of acquisition.	GROUPS							REMARKS
					Patients				Staff			
					1	2	3	4	A	B	C	
27	14	55	19	11	*						*	Patient No.42 in the adjacent cot carried type 19 in Weeks 8, 9 and 10. Patients Nos. 9 and 44 in the same division carried type 19 in Weeks 9, 10 and 11.
28	14	64	19	14			*				*	A patient in each of two other divisions carried type 19; Patient No.68 in Week 13 and Patient No.55 in Weeks 13 and 14.
29	15	2	22	2		*					*	Patient No.7 in the same division was admitted carrying type 22 in Week 2. Patient No. 2 acquired type 22 two days after No.7 was admitted.
30	15	47	22	9				*	*			Staff No.24 acquired type 22 in Week 9. It is therefore possible that Staff No.24 infected the patient. (Both swabbed on the same day).
31	15	88	22	13			*				*	Patient No.90 in the adjacent division carried type 22 both in Week 12 and Week 13.
32	15	109	22	21				*			*	
33	16	30	33	7				*			*	Type 33 pneumococcus was isolated from the dust near this patient's cot in Week 7.
34	16	79	33	11			*				*	Patient No.76 in the adjacent division A in Week 10 and in division B in Week 11, carried type 33 in both weeks.
35	16	107	33	19				*			*	

Number.	Diagram No.	Serial No. of Patient.	Type acquired.	Week of acquisition.	GROUPS Patients 1	2	3	4	Staff A	B	C	REMARKS
36	16	114	33	22	*						*	In Week 21 Patients No.114 and 107 were moved to adjacent cots. Patient No.107 carried type 33 in Week 21 although not in Week 22.
37	17	24	4	7				*			*	
38	17	34	4	8	*						*	Patients No. 24 and 39 in the two adjacent cots to No.34 carried type 4 in Week 7 and No.39 still carried type 4 in Week 8.
39	17	37	4	10			*				*	Patient No.39 in the adjacent division carried type 4 in Weeks 8 to 12 inclusive.
40	17	39	4	7		*					*	Patient No.24 in the same division acquired type 4 five days before type 4 was isolated from No.39. Therefore Patient No.24 may have infected No.39 or both may have been infected from the same unknown source.
41	17	48	4	8			*				*	In division A (not adjacent) Patient No.39 in Weeks 7 and 8, No.24 in Week 7 and No.34 in Week 8 carried type 4.
42	17	110	4	21				*			*	
43	18	39	23	11	*				*			Patient No.74 in the adjacent cot and Patients No. 69 and 70 in the same division carried type 23 in Weeks 10 and 11. Staff No.17 carried type 23 in Week 11.
44	18	46	23	9		*				*		Patient No.51 in the opposite cot (division D) carried type 23 in Weeks 7, 8 and 9. Staff No.17 acquired type 23 in this week.

Number.	Diagram No.	Serial No. of Patient.	Type acquired.	Week of acquisition.	GROUPS Patients 1	2	3	4	Staff A	B	C	REMARKS
45	18	59	23	9	*					*		Patient No.54 in the adjacent cot carried type 23 in Week 8 although not in Week 9. Previous occupant of No.59's cot also carried type 23. Staff No.17 acquired type 23 this week.
46	18	60	23	10		*			*			Patient No.71 in the opposite cot (division D) carried type 23 when No.60 was transferred to this division. Previous occupant of No. 60's cot also carried a type 23. (Not infected in division A - see dates in diagram). Staff No. 17 was in contact with the patient and carried type 23 in Weeks 9 and 11.
47	18	73	23	11		*					*	Patient No.71 in the adjacent cot carried type 23 in Week 9 but not in Week 10 or 11. Patient No.60 in the opposite cot carried type 23 in Week 10. There were numerous carriers of type 23 in the adjacent divisions.
48	18	80	23	12	*						*	Patient No.70 in the adjacent cot carried type 23 in Weeks 11 and 12. In Week 11 also, Patients No. 39, 69 and 74 in the same division carried type 23.
49	18	104	23	17			*				*	Patient No.104 was swabbed in Week 17 on the day of transfer to division C from division A where he must have acquired the type 23 pneumo. There were no carriers of type 23 in division A.

Number.	Diagram No.	Serial No. of Patient.	Type acquired.	Week of acquisition.	GROUPS							REMARKS
					Patients				Staff			
					1	2	3	4	A	B	C	
50	19	10	6	5		*			*			Patient No.17 in the same division carried type 6 in Weeks 4 and 5. Patient No. 12 in the adjacent division carried type 6 in Weeks 4 and 5. Staff No.14 carried type 6 in Week 5.
51	19	25	6	6		*			*			Patients No.12 and 26 in the same division carried type 6 in Week 5. Patient No.13 in the opposite cot carried type 6 in Weeks 3 and 6. Staff No.14 carried type 6 in Week 6.
52	19	31	6	8		*			*			Patient No.17 in the opposite cot carried type 6 in Weeks 7 and 8. In the same division No.26 carried type 6, three days, and No.55, two days before No.31 acquired type 6. Staff No.14 carried type 6 in Week 8.
53	19	47	6	9	*				*			Patient No.52 in the adjacent cot carried type 6 in Weeks 7 and 8. Patient No.45 in the same division carried type 6 in Weeks 8 and 9. Staff No. 14 carried type 6 in Week 9.
54	19	66	6	11		*			*			Patient No.67 in the same division carried type 6 in Week 10. In the other divisions there were several carriers of type 6. Staff No.14 carried type 6 in Week 11.

Number.	Diagram No.	Serial No. of Patient.	Type acquired.	Week of acquisition.	GROUPS Patients 1	2	3	4	Staff A	B	C	REMARKS
55	19	68	6	11	*				*			In Week 10 the other three occupants of division D (No. 67, 71 and 73) carried type 6. In Week 11 No.67 in the adjacent cot and No.71 in the same division carried type 6. Staff No.14 carried type 6 in Week 11.
56	19	76	6	11	*				*			Patient No.45 in the adjacent cot carried type 6 in Weeks 10 and 11. In Week 10, Patients No.17 and 55 in the same division carried type 6. Staff No.14 carried type 6 in Week 11.
57	19	84	6	15		*					*	Patient No.95 in the opposite cot carried type 6 in Weeks 14 and 15. In Week 14 Patient No.94, also in the same division, carried type 6.
58	19	94	6	14	*						*	Patient No.95 in the adjacent cot carried type 6 when admitted 4 days prior to No. 94 acquiring type 6.
59	19	98	6	17			*				*	Patient No.84 in the adjacent division carried type 6 in Week 16 but not in Week 17. Patient No.99 in the adjacent division carried type 6 in Week 17.
60	19	99	6	17		*					*	Patient No.84 in the opposite cot carried type 6 in Week 16 but not in Week 17.
61	19	106	6	19				*			*	Patient No.99 in the adjacent division carried type 6 but was dismissed before No.106 was admitted.
62	Patient No.53 acquired a pneumococcus which was un-typable. No source could therefore be found for it.											

SUMMARY OF FINDINGS. (Contd.)

B). Acquisition of fresh pneumococcal types by members of the staff.

Serial Number of Staff.	Type of Pneumo-coccus acquired.	Week when Pneumo-coccus acquired.	Diagram.
1	10	3	13
1	6	10	19
6	22	22	15
9	29	10	20
11	6	4	19
11	18	9	4
12	14	3	20
13	6	9	19
14	15	15	10
15	23	7	18
17	15	5	10
17	23	9	18
18	3	5	20
18	6	8	19
23	11	9	3
24	22	9	15
24	35	15	20
28	35	13	20
31	37	13	20

SUMMARY OF FINDINGS. (Contd.)

Acquisition of fresh pneumococcal types by members of the staff.

Serial Number of Staff.	Type of Pneumo-coccus acquired.	Week when Pneumo-coccus acquired.	Diagram.
33	2	15	2
33	14	16	20
35	21	21	11

Summary of results of tracing the source of the acquired type of pneumococcus to the patients or to other members of staff.

Group 1. Source probably a patient.

Group 2. A patient could be excluded as the source.

Group A. Another member of staff might be the source.

Group B. A member of staff could be excluded as the source.

Number.	Diagram No.	Serial No. of Staff.	Type acquired.	Week of acquisition.	GROUPS				REMARKS
					Patients		Staff		
					1	2	A	B	
1	2	33	2	15	*			*	Patient No.85 carried type 2 in Weeks 12 to 15 inclusive.
2	3	23	11	9	*			*	Patient No.61 carried type 11 in Weeks 8 and 9.
3	4	11	18	9		*		*	
4	10	17	15	5	*			*	Patient No.9 carried type 15 in Weeks 3 and 4 though not in Week 5.
5	10	14	15	15		*		*	
6	11	35	21	21		*		*	Patient No.119 carried type 21 but was admitted after type 21 was isolated from Staff No.35.
7	13	1	10	3		*		*	
8	15	6	22	22	*			*	Patient No.109 acquired type 22 in Week 21 but was dismissed before Staff No.6 was found to have acquired type 22 in Week 22.

Number.	Diagram No.	Serial No. of Staff.	Type acquired.	Week of acquisition	GROUPS				REMARKS
					Patients		Staff		
					1	2	A	B	
9	15	24	22	9	*			*	Patient No.47 acquired type 22 in Week 9 and may have infected Staff No.24. The converse is equally possible.
10	18	15	23	7	*			*	Patients No.23, 28, 49 and 51 carried type 23 in Week 7 and No. 21, 23, 28 and 37 carried type 23 in Week 6.
11	18	17	23	9	*			*	Patients No. 46, 51, 59, 69, 70 and 71 carried type 23 in Week 9 and No.51 and 54 carried type 23 in Week 8.
12	19	1	6	10	*		*		Patients No. 17, 45, 55, 67, 71 and 73 carried type 6 in Week 10 and No.17, 43, 45, 47, 55, 67 and 71 carried type 6 in Week 9. Staff No.14 was a constant carrier of type 6.
13	19	11	6	4	*		*		Patients No.13 and 17 carried type 6 in Week 4 and No. 12 and 13 carried type 6 in Week 3. Staff No.14 was a constant carrier of type 6.
14	19	13	6	9	*		*		Patients No. 17, 43, 45, 47, 55, 67 and 71 carried type 6 in Week 9 and No. 17, 26, 31, 43, 45, 52 and 55 carried type 6 in Week 8. Staff No.14 was a constant carrier of type 6.
15	19	18	6	8	*		*		Patients No. 17, 26, 31, 43, 45, 52 and 55 carried type 6 in Week 8 and No. 17, 43, 45 and 52 carried type 6 in Week 7. Staff No.14 was a constant carrier of type 6.

Number.	Diagram No.	Serial No. of Staff.	Type acquired.	Week of acquisition.	GROUPS				REMARKS
					Patients		Staff		
					1	2	A	B	
16	20	9	29	10		*		*	
17	20	12	14	3		*		*	
18	20	18	3	5		*		*	
19	20	24	35	15		*	*		Staff No.24 may have acquired type 35 from Staff No.28 who carried type 35 in Week 13 only.
20	20	28	35	13		*		*	
21	20	31	37	13		*		*	
22	20	33	14	16	*			*	Patient No.94 carried type 14 in Week 16 and had previously carried it in Week 13.

SUMMARY OF FINDINGS

C). Acquisition of Streptococcus pyogenes by ward patients. (Diagram 21).

Summary of results of tracing the source of the acquired streptococcus to a patient or to a member of staff.

Group 1. Source traced to the patient in the adjacent cot.

Group 2. Source traced to a patient in the same division but not in the adjacent cot.

Group 3. Source traced to a patient in another division of the ward.

Group 4. Source could not be traced to any of the patients in the ward.

Group A. Source could be traced to a member of staff.

Group B. Source unlikely to be a member of staff but not impossible.

Group C. Members of staff not swabbed in this week.

"+" Small number of organisms acquired.

"++" Many organisms acquired.

Number.	Week of acquisition.	Serial No. of Patient.	No. of S. pyog. acquired.	GROUPS							REMARKS
				Patients				Staff			
				1	2	3	4	A	B	C	
1	2	5	+				*		*		Staff No.10 carried S.pyog.in Weeks 1 and 3 though not in Week 2. No other patients carried S.pyog.
2	5	9	+				*	*			Five members of staff carried S.pyog.this week, 3 of whom carried it for the first time. Staff No.13 carried S.pyog.++ in Weeks 3,4,6 and 7. No other patients carried S.pyog.
3	5	10	+				*	*			
4	5	17	++				*	*			

Number.	Week of Acquisition.	Serial No. of Patient.	No. of S. pyog.acquired.	GROUPS							REMARKS
				Patients				Staff			
				1	2	3	4	A	B	C	
5	11	38	+			*			*		These 3 patients were in the ward but in different divisions to Patient No.42 when she developed scarlet fever in Week 10. Six members of staff carried S.pyog.this week, 4 of whom carried it for the first time.
6	11	58	+			*			*		
7	11	67	+			*			*		
8	11	55	+	*					*		In Week 10, Patient No.55 was in the adjacent cot to Patient No.42 when she developed scarlet fever. The staff situation was as stated for Patients No.38, 58 and 67.
9	12	49	+		*					*	In Week 11, Patient No.49 was in the same division as Patient No.55 who carried S.pyog.
10	12	53	+		*					*	In Week 11, Patient No.53 was in another division to Patient No.55 who carried S.pyog. In Week 12,however, Patient No. 53 was in the same division as Patient No.87, who carried S.pyog. on admission, three days prior to Patient No.53 acquiring S.pyog.
11	12	64	+		*					*	In Week 10, Patient No.64 was in the same division as Patient No.42; in Weeks 10 and 11 in the same division as Patient No.55 who acquired S. pyog. in Week 11. In Week 12 Patient No.64 was in the same division as Patient No.87 who carried S.pyog. on admission three days prior to Patient No.64 acquiring S.pyog.

Number.	Week of Aquisition.	Serial No. of Patient.	No. of S. pyog. acquired.	GROUPS							REMARKS
				Patients				Staff			
				1	2	3	4	A	B	C	
12	12	68	+		*					*	In Week 11, Patient No.68 was in the adjacent cot to carrier Patient No.67. Both were transferred to another division where, three days later, Patient No.68 was found to have acquired S.pyog. There were other carriers in this division, however.
13	13	84	+			*			*		There were numerous carriers in the other divisions. Seven members of staff carried S. pyog. this week, four of whom carried it for the first time.
14	13	88	++		*				*		Patient No.88 was in the same division as three carriers of S.pyog, patients No.53,64 and 93. Seven members of staff carried S.pyog. this week, four for the first time.
15	14	85	+		*				*		Patient No.85 was in same division as Patient No.84, in Week 13, when No.84 carried S.pyog. Four of the staff, carried S. pyog. this week.
16	14	86	++	*					*		Patient No.86 was in the adjacent cot to Patient No.84 when he carried S.pyog. in Week 13. Four of the staff carried S.pyog. this week.
17	14	92	+	*					*		Patient No.92 was in the same division as were 4 carriers of S.pyog., No.53,64,88 and 93 in Week 13. No.93 was in the adjacent cot. Four of the staff carried S.pyog. this week.

Number.	Week of Acquisition	Serial No. of Patient.	No. of S. pyog. acquired.	GROUPS							REMARKS
				Patients				Staff			
				1	2	3	4	A	B	C	
18	16	90	+	*					*		In Week 15, Patient No.90 was in the adjacent cot to carrier Patient No.85. Patient No.84 was in the same division although not a carrier in Week 15. Two members of staff carried S.pyog.and another,No. 14, acquired S.pyog.++ this week.
19	16	91	+		*				*		In Week 15, Patient No.91 was in the same division as carrier Patient No.85 and Patient No.84 who occasionally carried S.pyog. Two members of staff carried S. pyog.and another, No.14 acquired S.pyog.++ this week.
20	19	102	+		*				*		These 2 patients were in the same division as Patient No. 90 who carried S.pyog.on several occasions including Week 19. Four members of staff carried S.pyog.this week.
21	19	104	+		*				*		
22	21	101	+		*				*		Patient No.101 was in same division as Patients No.111 and 112 who carried S.pyog.in Week 20. Other carriers present in another division. Three members of staff carried S.pyog.this week (No.36 carried S.pyog.++).
23	22	113	+	*					*		Patient No.113 was in the adjacent cot to No.98 who carried S.pyog.++ in Week 22. Five members of staff carried S. pyog.(two of them had S.pyog. ++).

Number.	Week of Acquisition.	Serial No. of Patient.	No. of S. pyog. acquired.	GROUPS Patients 1	2	3	4	Staff A	B	C	REMARKS
24	26	114	+		*					*	Patient No.114 was in the same division as Patient No.84 who occasionally carried S.pyog. and did so in Week 26. (Patient No.114 acquired S. pyog. after 6 weeks in the ward and he did so when moved to Division A with Patient No.84).
25	27	128	++	*						*	Patient No.128 was in the adjacent cot to Patient No. 114 who acquired S.pyog. in Week 26.

- and the following seven other instances. (a) Four patients who acquired S.pyog.++ but had carried S.pyog.+ previously. (b) Three patients who were found to be carrying S.pyog. again after five or more weeks without it.

26	8	41	++				*		*		Improbable that infection came from any other patient. Staff No.13 however carried S.pyog. ++ in Week 7 and yet another member of staff carried S. pyog.+
27	10	42 (scarlet fever)	++				*		*		No source amongst the patients is probable. Staff No.13 still in ward but no longer a carrier Three members of staff carried S.pyog.
28	18	98	++				*		*		This patient had carried S. pyog each week since admission. Four members of staff carried S.pyog.

Number.	Week of Acquisition.	Serial No. of Patient.	No. of S. pyog. acquired.	GROUPS							REMARKS
				Patients				Staff			
				1	2	3	4	A	B	C	
29	23	124	++				*		*		This patient had carried S. pyog.in Week 22. Only two members of staff carried S. pyog. Not infected from Patient No.121 who lost S. pyog.when transferred to Division D.
30	12	9	+		*					*	In Week 11, these patients were in Division B where Patient No.55 carried S.pyog.
31	12	45	+		*					*	
32	23	84	+		*				*		This patient had carried S. pyog.on two occasions before but the last one was 7 weeks previously. In Week 22, Patient No.84 was in the same division as Patient No.121 who carried S. pyog. Other carriers were present in the other divisions. Only two members of staff carried S.pyog.

SUMMARY OF FINDINGS

D). Acquisition of Streptococcus pyogenes by the members of staff. (Diagram 22).

Summary of results of tracing the source of the acquired streptococcus to a patient or to a member of staff.

Group 1. A patient was the most likely source.

Group 2. The possibility of a patient being the source of infection could not be excluded.

Group 3. Source could not be traced to a patient.

Group A. Source could be traced to a member of staff.

Group B. A member of staff was unlikely to be the source but could not be excluded.

Number.	Week of Acquisition.	Serial No. of member of Staff.	No. of S. pyog. acquired.	GROUPS					REMARKS
				Patients			Staff		
				1	2	3	A	B	
1	3	13	++			*		*	Unlikely to be infected by another member of staff, only one other carrier at this time. No patients carried S.pyog. in Week 3.
2	4	5	+			*	*		No patients carried S.pyog. this week. Staff No.13 still carrying S.pyog.++.
3	4	6	+			*	*		
4	5	9	+			*	*		Three patients simultaneously acquired S.pyog. Staff No.13 and one other member of staff carried S.pyog. this week.
5	5	12	+			*	*		
6	5	18	+			*	*		
7	6	8	+		*		*		Six patients carried S.pyog. this week, none for the first time. Staff No.13 still carried S.pyog.++
8	6	21	+		*		*		

Number.	Week of acquisition.	Serial No. of member of Staff.	No. of S. pyog. acquired.	GROUPS					REMARKS
				Patients			Staff		
				1	2	3	A	B	
9	9	20	+			*		*	Only one patient carried S. pyog. this week. Staff No.13 by this week carried only S. pyog.+. There was one other carrier amongst the members of staff.
10	10	1	+	*				*	In Week 10, Patient No.42 developed scarlet fever and other two patients carried S. pyog. One other member of staff carried S.pyog.
11	10	14	+	*				*	
12	11	3	++	*				*	In Week 10, Patient No.42 developed scarlet fever. In Week 11, 4 patients acquired S.pyog. Two other members of staff were carriers of S.pyog.
13	11	25	+	*				*	
14	11	28	+	*				*	
15	13	4	+	*				*	In Week 13, 7 patients carried S.pyog. two for the first time. Three other members of staff carried S.pyog.but one, No.6., had not carried S.pyog. since Week 4 and another, No.8, since Week 6.
16	13	17	+	*				*	
17	13	24	+	*				*	
18	17	26	+		*			*	One patient carried S.pyog. Two other members of the staff carried S.pyog.
19	18	11	+	*			*		Three of the patients carried S.pyog., one of whom had S. pyog.++. Two other members of staff carried S.pyog.,one of them, newly arrived on the staff, carried S.pyog.++.

- and the following five other instances (a) Three members of staff who acquired S.pyog.++ but had carried S.pyog.+ previously. (b) Two members of staff who carried S.pyog. again after five or

more weeks without it.

Number.	Week of acquisition.	Serial No. of member of Staff.	No. of S. pyog. acquired.	GROUPS					REMARKS
				Patients			Staff		
				1	2	3	A	B	
20	11	9	++	*				*	In Week 10, Patient No.42 developed scarlet fever and in Week 11 other four patients acquired S.pyog. Two other members of staff carried S. pyog.
21	13	28	++	*				*	In Week 13 seven patients carried S.pyog. Six other members of staff carried S. pyog. this week; 5 of them for the first time.
22	16	14	++	*				*	Five of the patients carried S.pyog. in Week 16. Two other members of staff carried S.pyog.
23	13	6	+	*				*	In Week 13, seven patients carried S. pyog.
24	13	8	+	*				*	In this week, seven members of staff carried S.pyog., six for the first time.

6. Diagrams for Section 2, Chapter 2.

These diagrams were used in the tracing of the possible sources of pneumococci acquired by patients or members of staff. Each type of pneumococcus is dealt with in a separate diagram.

Diagram	Type of Pneumococcus.	Diagram	Type of Pneumococcus.
1	1	11	21
2	2	12	7
3	11	13	10
4	18	14	19
5	9	15	22
6	16	16	33
7	24	17	4
8	42	18	23
9	17	19	6
10	15	20	Types acquired by staff and never by patients.

and Diagram 21 - Similar diagram for Streptococcus pyogenes - patients findings only.

Diagram 22 - The incidence of Streptococcus pyogenes amongst the members of staff.

Note: Diagrams 1 - 19 show, for each week, the position in the ward of any patient who, at any time, carried the type of pnuemococcus in question. The ward divisions are lettered and the cots numbered as shown in Section 1, figure 1b.

Each week, the serial number of any patient concerned is inserted adjacent to the number of the cot occupied. The serial number is enclosed in a square if, in that particular week, the type was isolated from the patient. Changes in the position of the patient in the ward are indicated. The findings for the members of staff are also summarised in the diagrams.

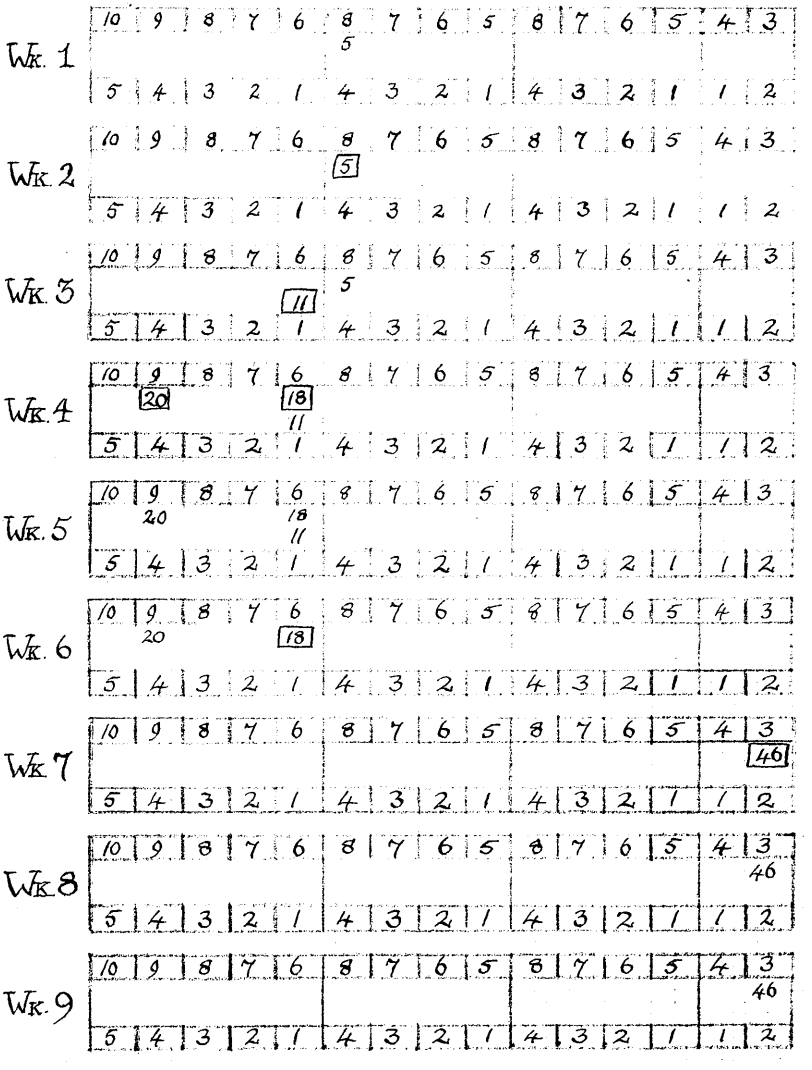
In diagram 21, the organism involved is Streptococcus pyogenes. If many organisms were carried at any one swabbing, this is indicated by "++".

Diagrams 20 and 22 are self explanatory.

* * * * *

DIAGRAM 1

PNEUMOCOCCUS TYPE 1



Patients acquiring

Type 1

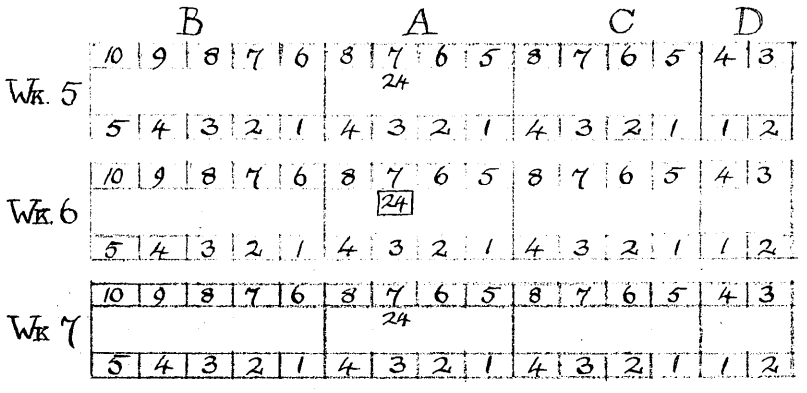
No. 5 in Week 2.

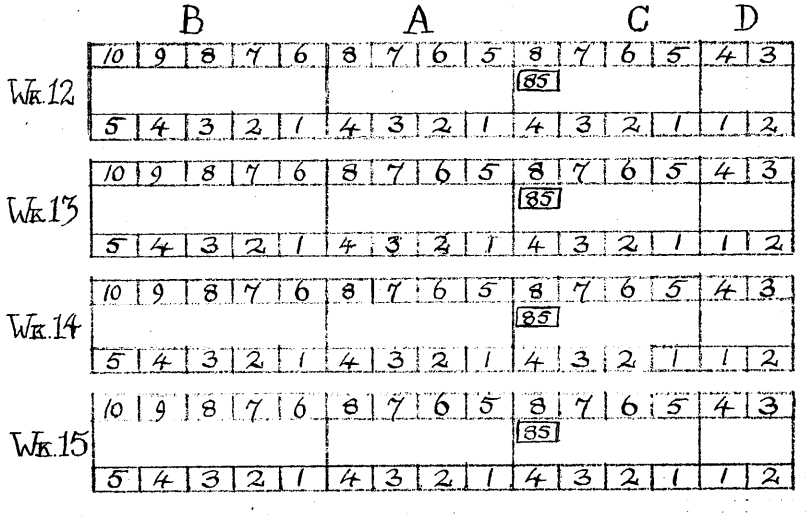
Isolations from Staff

None.

DIAGRAM 2

PNEUMOCOCCUS TYPE 2





Patients acquiring Type 2

No. 24 in Week 6.

Staff acquiring Type 2.

No. 33 in Week 15.

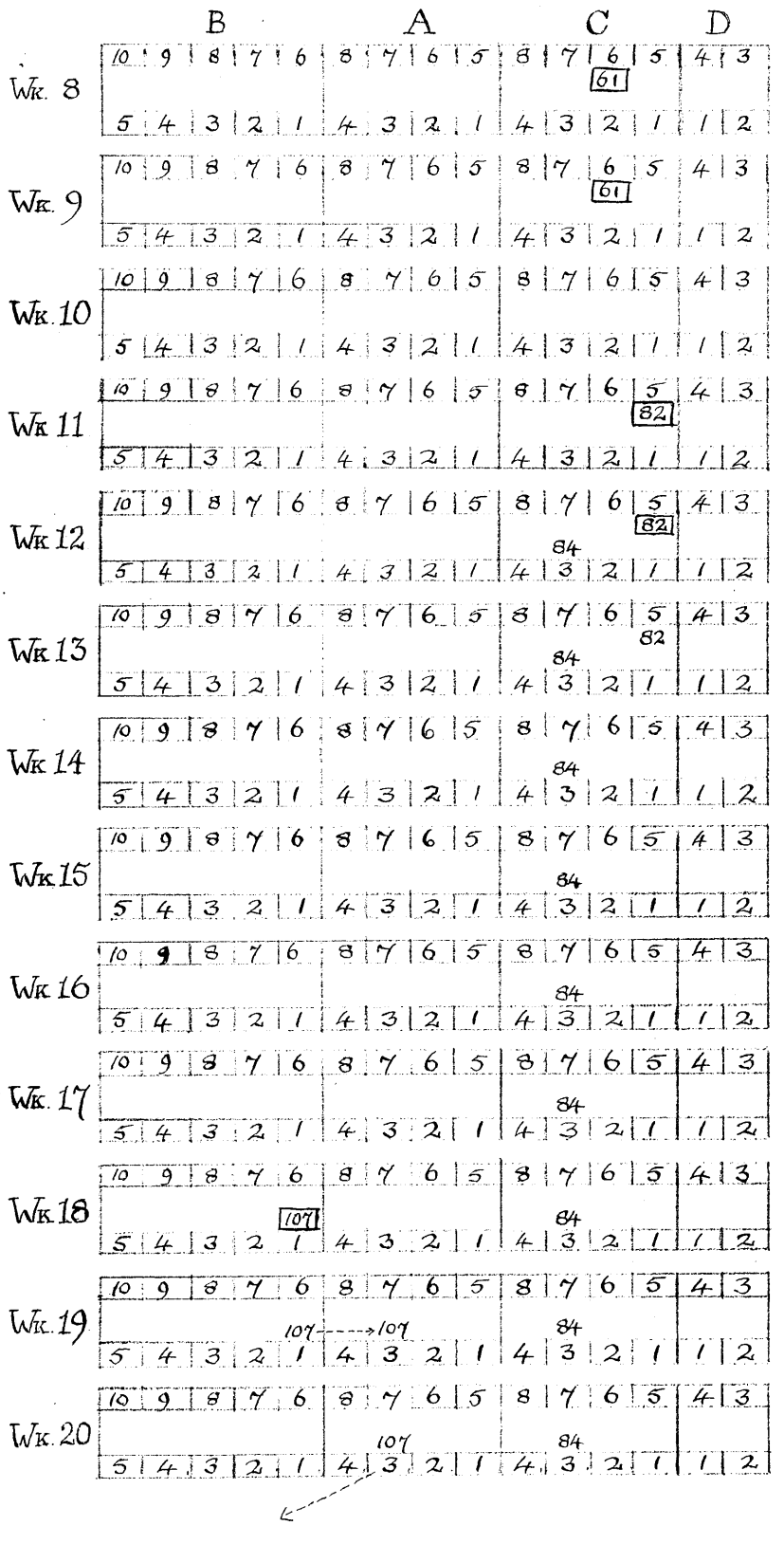
Other Isolations from staff.

No. 9 in Weeks 1 and 9.

No. 33 in Weeks 17 and 23.

DIAGRAM 3

PNEUMOCOCCUS TYPE 11



Patients acquiring Type 11.

No. 84 in Week 22.

Staff acquiring Type 11

No. 23 in Week 9.

Other isolations from staff.

None.

DIAGRAM 3

(CON'D)

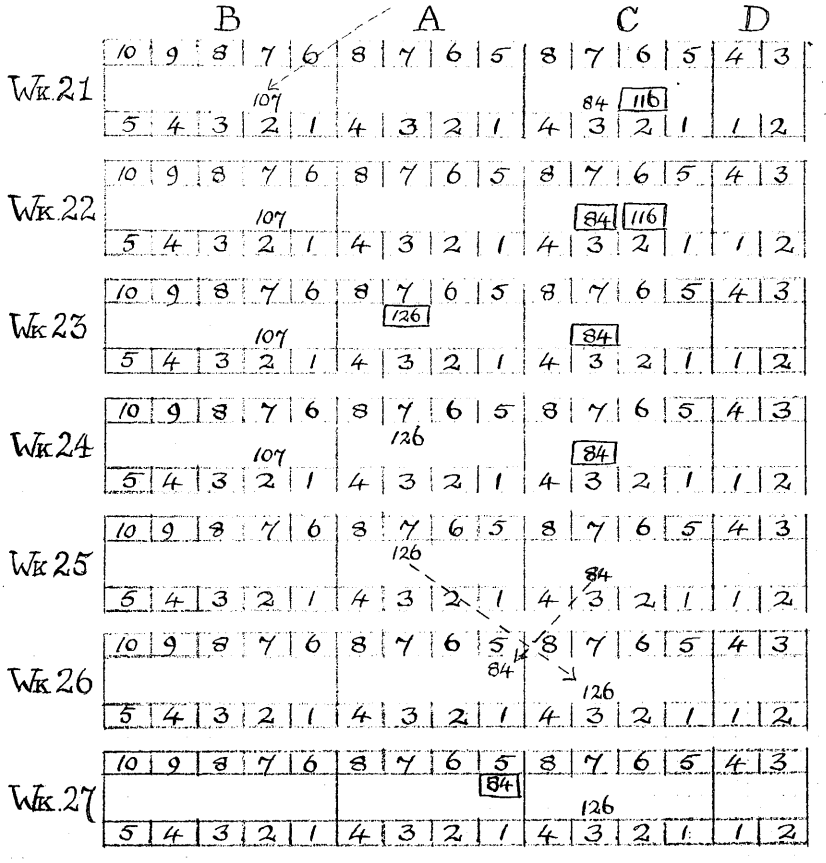
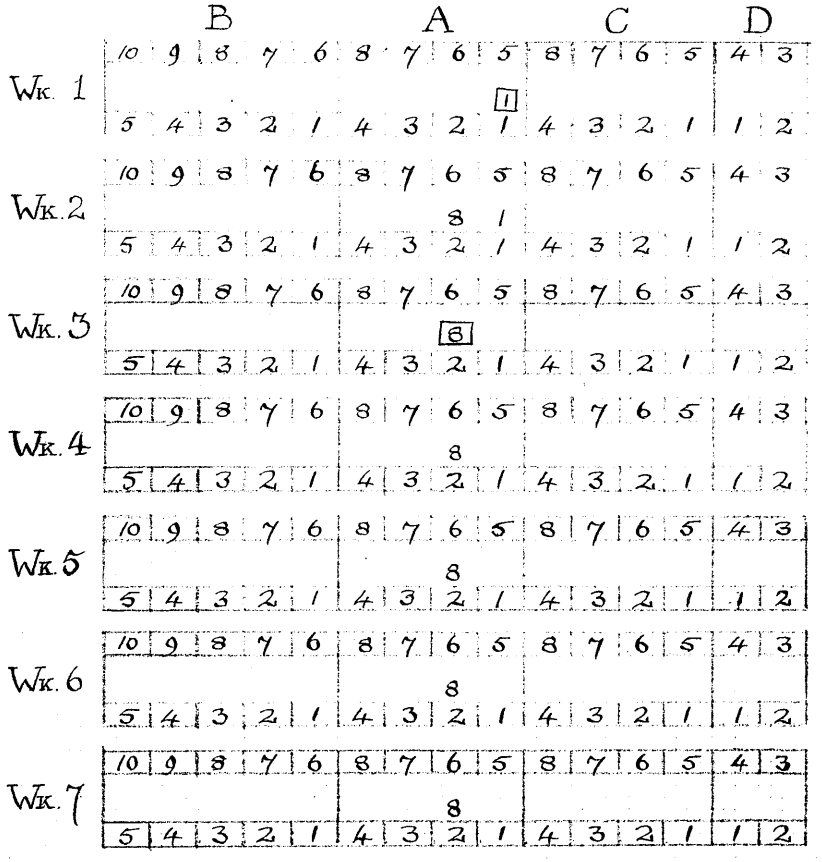
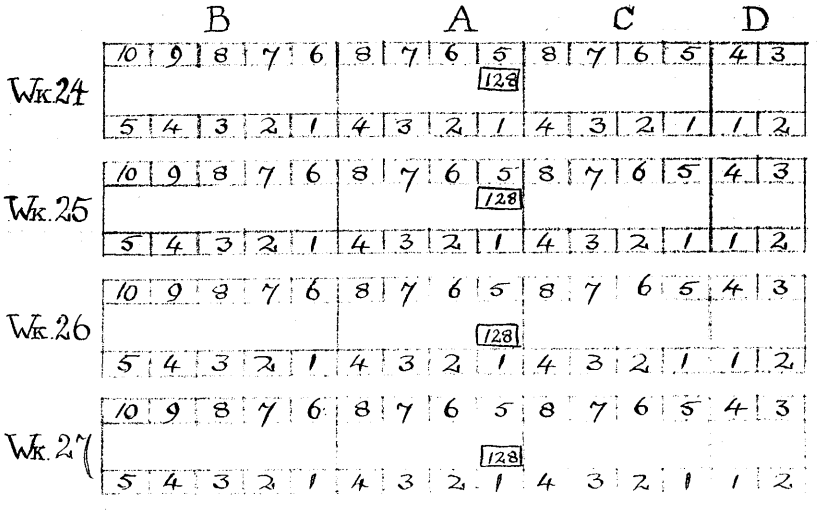


DIAGRAM 4

PNEUMOCOCCUS TYPE 18





Patients acquiring Type 18

No. 8 in Week 3.

Staff acquiring Type 18

No.11 in Week 9.

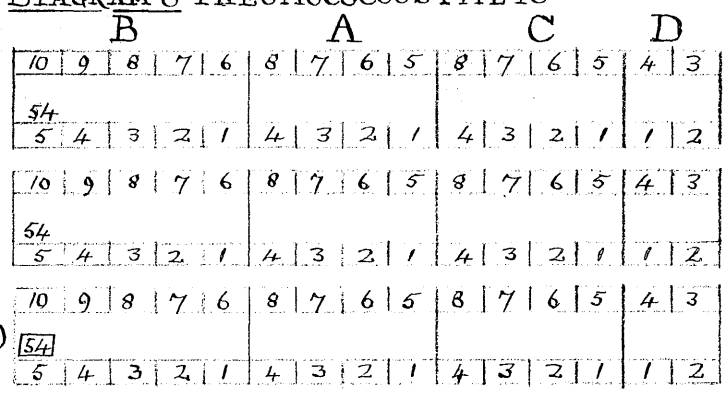
Other Isolations from Staff

None

Diagram 5 Pneumococcus type 9

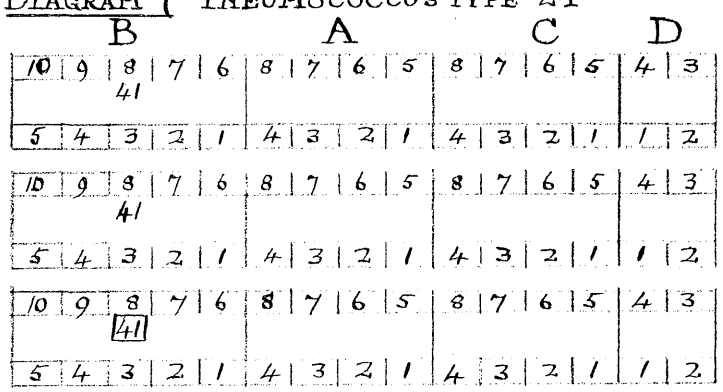
Type 9 was only isolated on this one occasion in Week 8. Not carried by the staff.

Diagram 6 Pneumococcus type 16



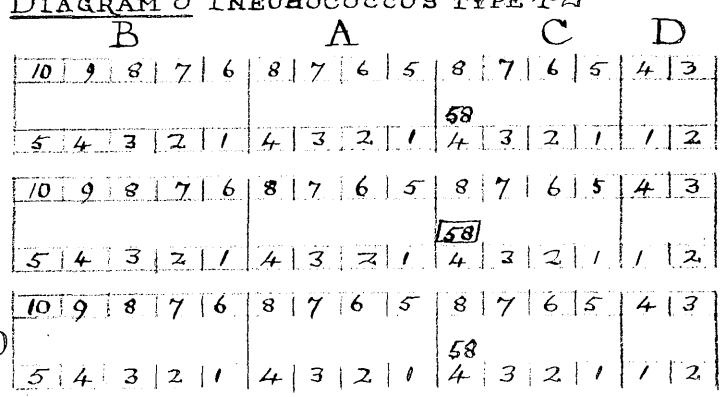
Type 16 was only isolated on this one occasion in Week 10.
Not carried by the staff.

Diagram 7 Pneumococcus type 24



Type 24 was only isolated on this one occasion in Week 9.
Not carried by the staff.

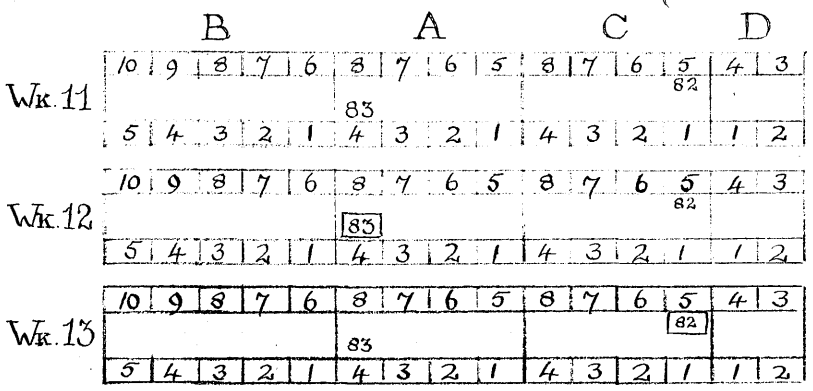
Diagram 8 Pneumococcus type 42



Type 42 was only isolated on this one occasion in Week 9.
Not carried by the staff.

DIAGRAM 9

PNEUMOCOCCUS TYPE 17



Patients acquiring Type 17.

No. 82 in Week 13

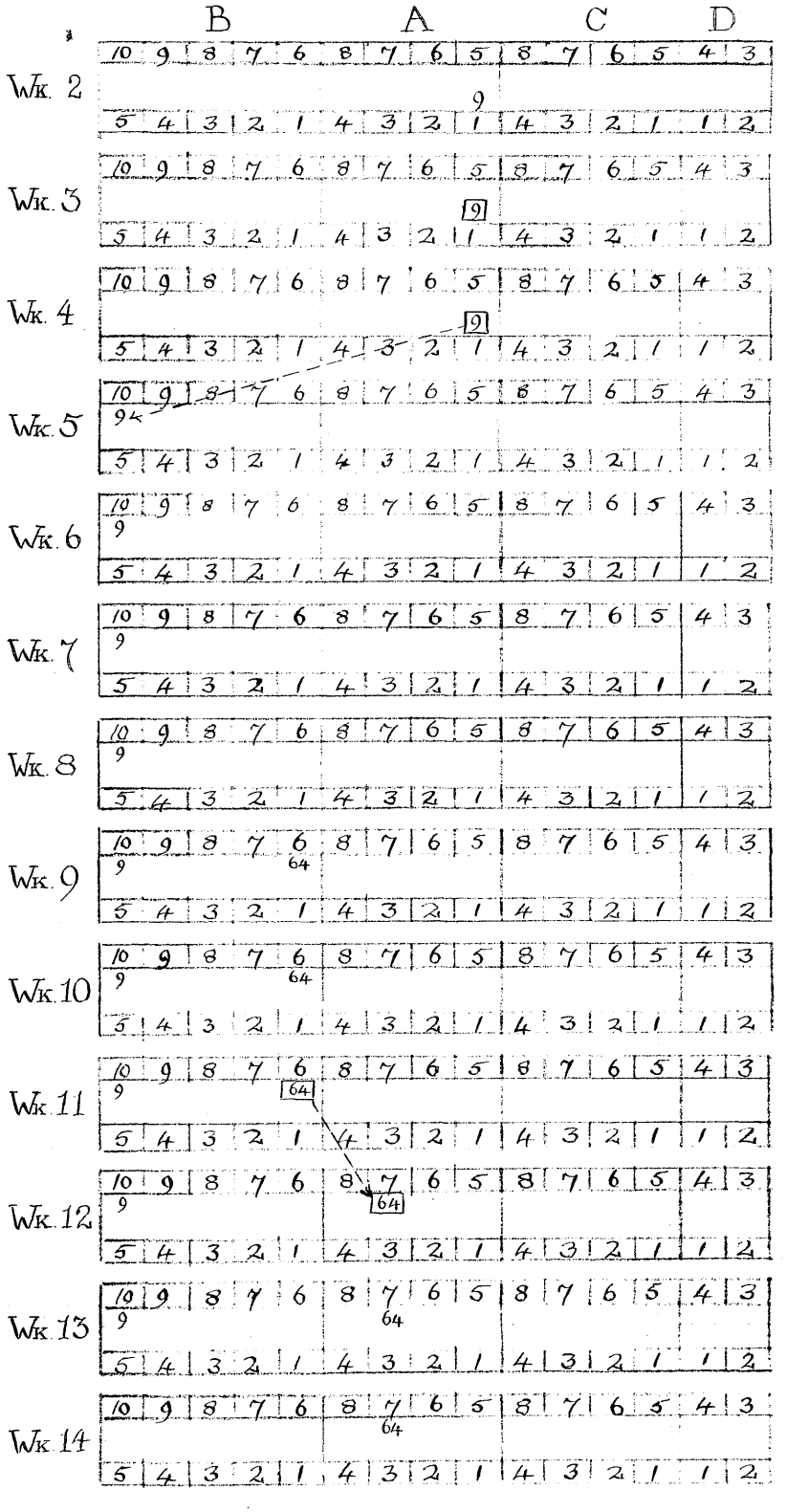
No. 83 in Week 12

Isolations from staff

None.

Diagram 10

Pneumococcus type 15



Patients acquiring Type 15

No. 9 in Week 3.

No.64 in Week 11.

No.100 in Week 17.

Staff acquiring Type 15

No.17 in Week 5.

No.14 in Week 15.

Other isolations from staff

None.

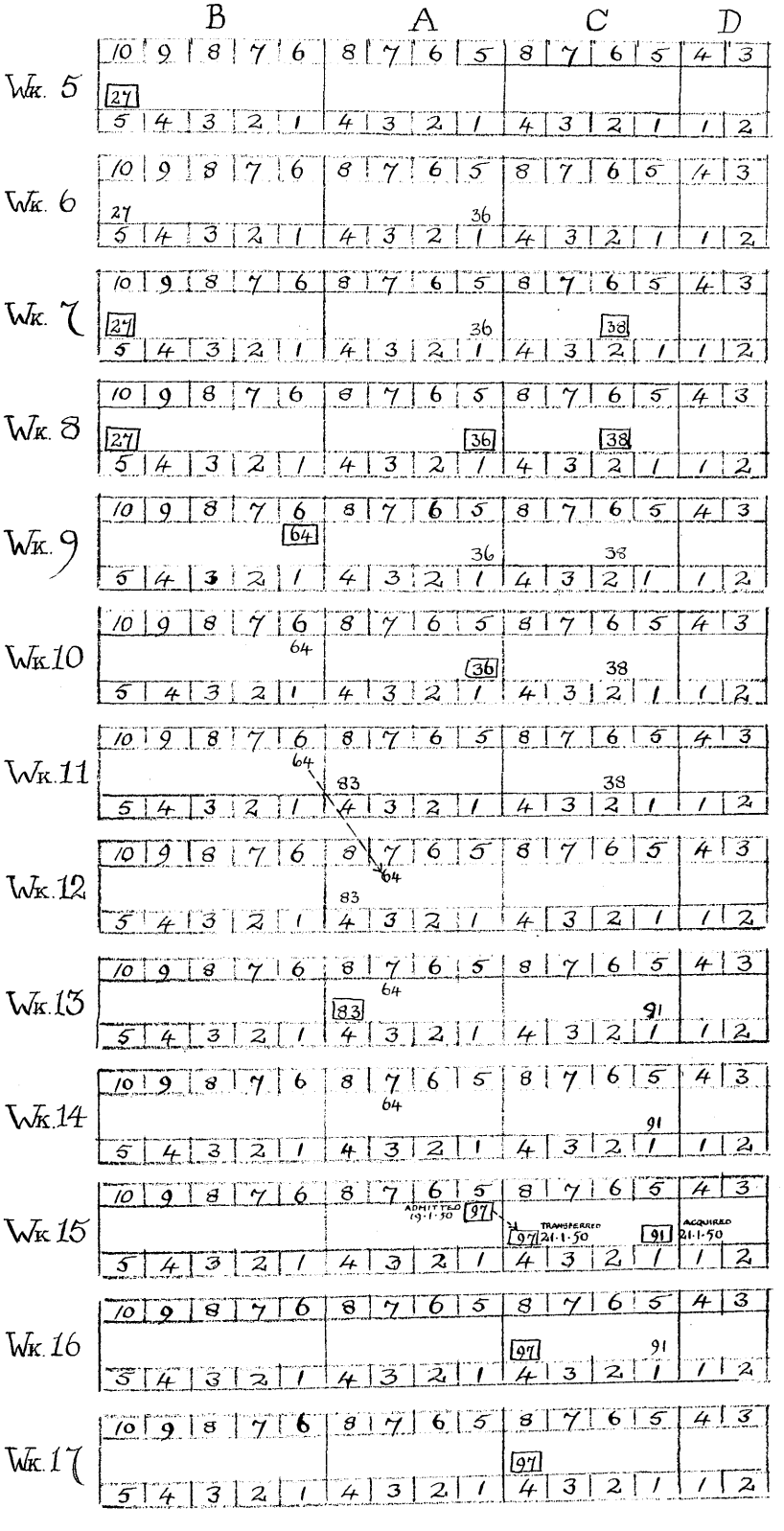
DIAGRAM 10
(CON'D)

	B					A					C					D	
Wk. 15	10	9	8	7	6	8	7	6	5		8	7	6	5		4	3
	5	4	3	2	1	4	3	2	1		4	3	2	1		1	2
Wk. 16	10	9	8	7	6	8	7	6	5		8	7	6	5		4	3
	5	4	3	2	1	4	3	2	1		4	3	2	1		1	2
Wk. 17	10	9	8	7	6	8	7	6	5		8	7	6	5		4	3
	5	4	3	2	1	4	3	2	1		4	3	2	1		1	2
Wk. 18	10	9	8	7	6	8	7	6	5		8	7	6	5		4	3
	5	4	3	2	1	4	3	2	1		4	3	2	1		1	2
Wk. 19	10	9	8	7	6	8	7	6	5		8	7	6	5		4	3
	5	4	3	2	1	4	3	2	1		4	3	2	1		1	2
Wk. 20	10	9	8	7	6	8	7	6	5		8	7	6	5		4	3
	5	4	3	2	1	4	3	2	1		4	3	2	1		1	2
Wk. 21	10	9	8	7	6	8	7	6	5		8	7	6	5		4	3
	5	4	3	2	1	4	3	2	1		4	3	2	1		1	2

100

DIAGRAM 11

PNEUMOCOCCUS TYPE 21



Patients acquiring Type 21

No. 36 in Week 8

No. 83 in Week 13

No. 91 in Week 15

Staff acquiring Type 21

No. 35 in Week 21

Other isolations from staff

None.

DIAGRAM 11
(CON'D)

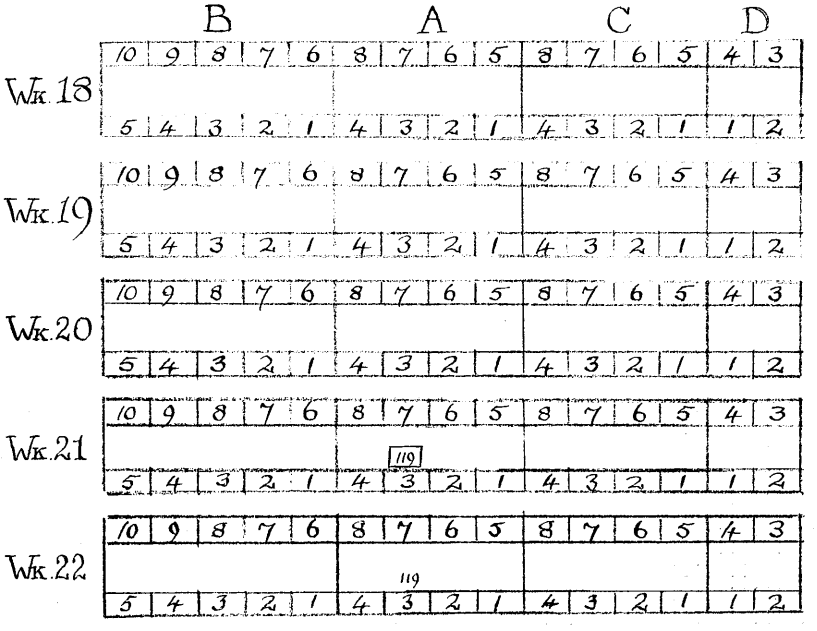
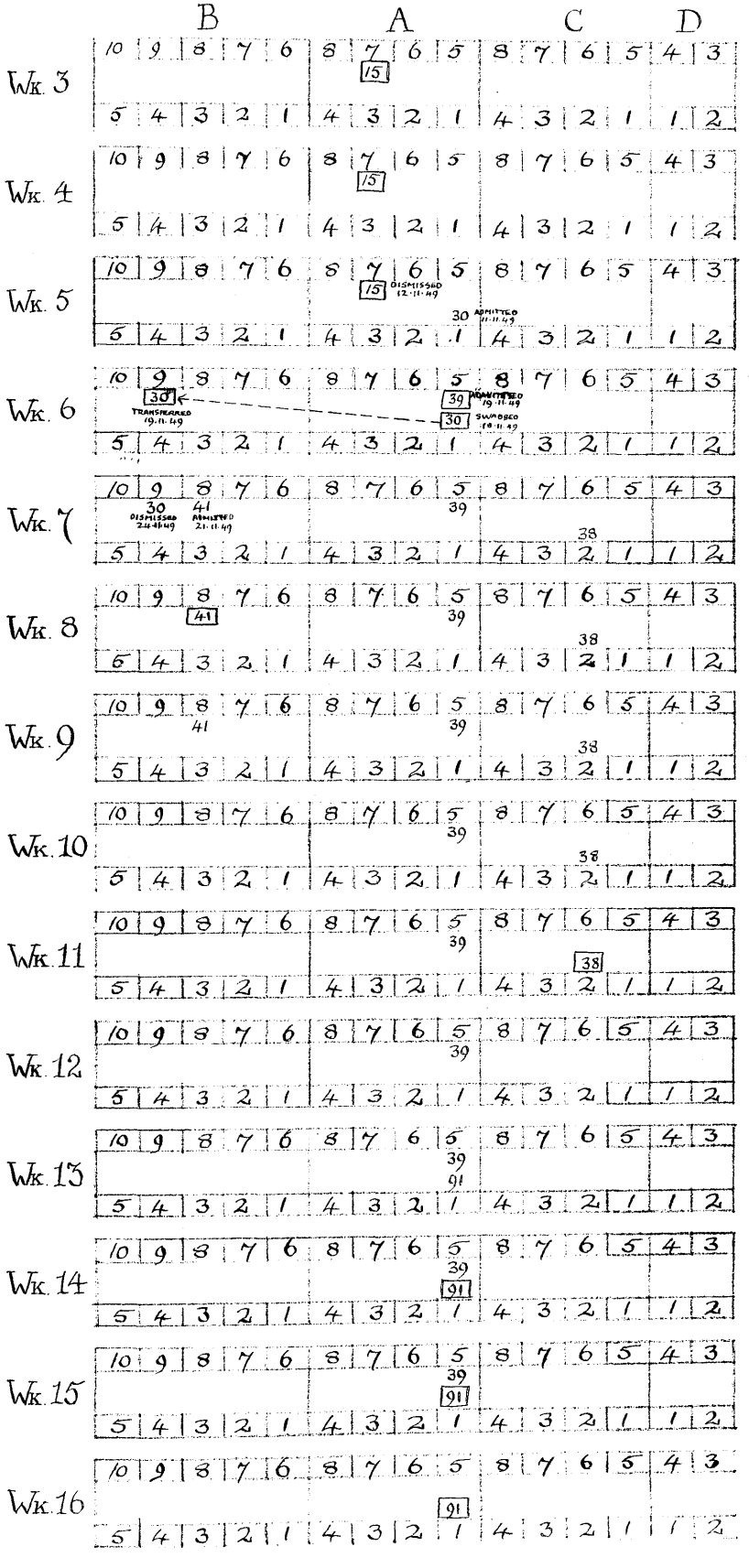


DIAGRAM 12

PNEUMOCOCCUS TYPE 7



Patients acquiring Type 7

No.30 in Week 6

No.41 in Week 8

No.38 in Week 11

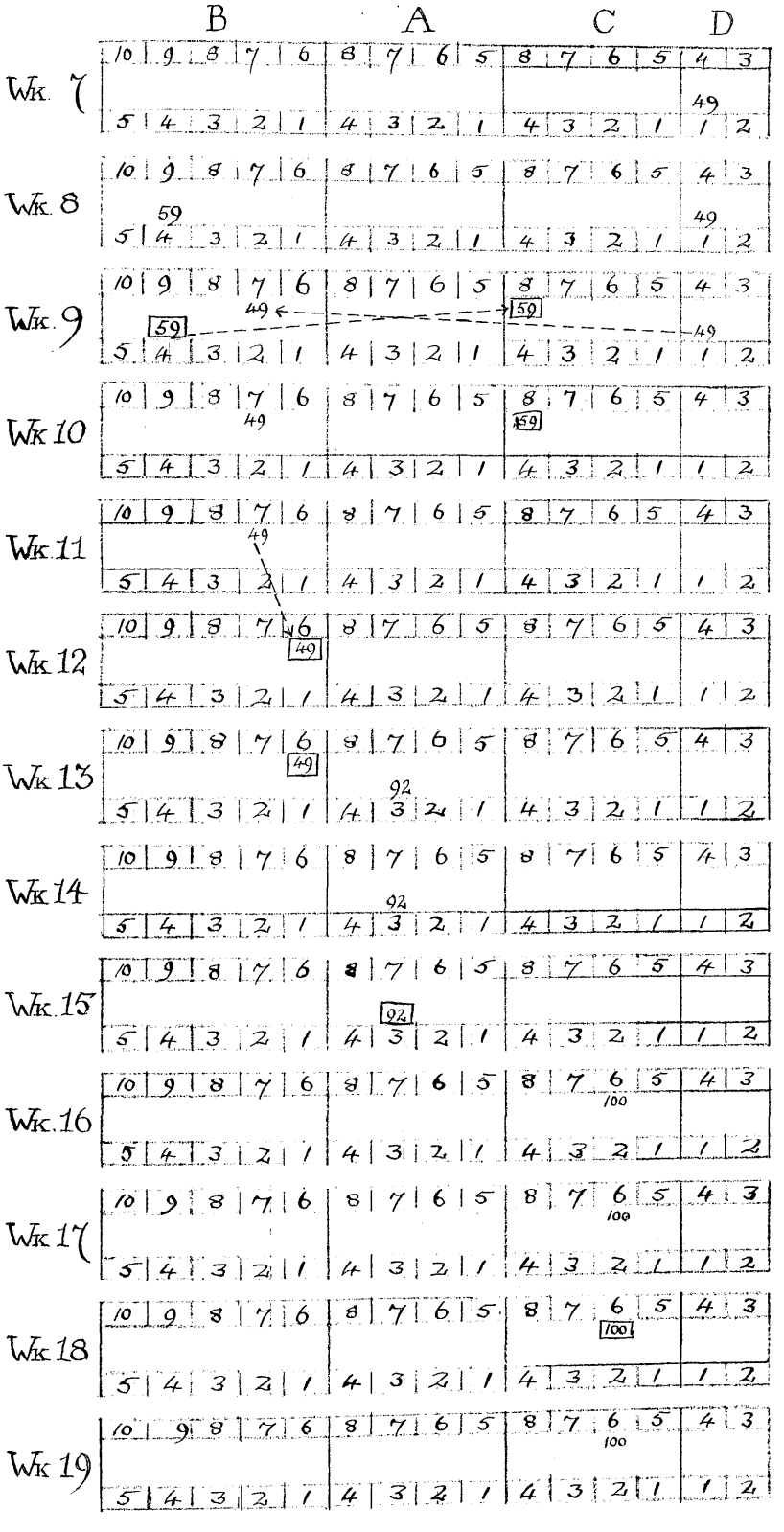
No.91 in Week 14

Isolations from Staff

None

DIAGRAM 13

PNEUMOCOCCUS TYPE 10



Patients acquiring Type 10

No.49 in Week 12

No.59 in Week 9

No.92 in Week 15

No.100 in Week 18

Staff acquiring Type 10

No. 1 in Week 3

Other isolations from staff

No.29 in Week 11

No.34 in Week 15

DIAGRAM 13
(CON'D)

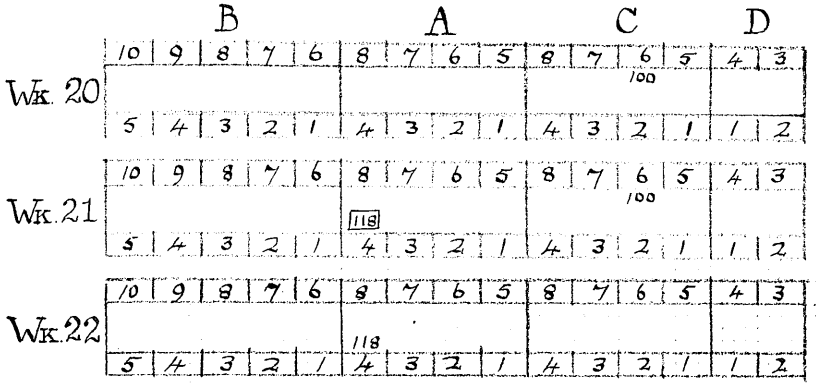
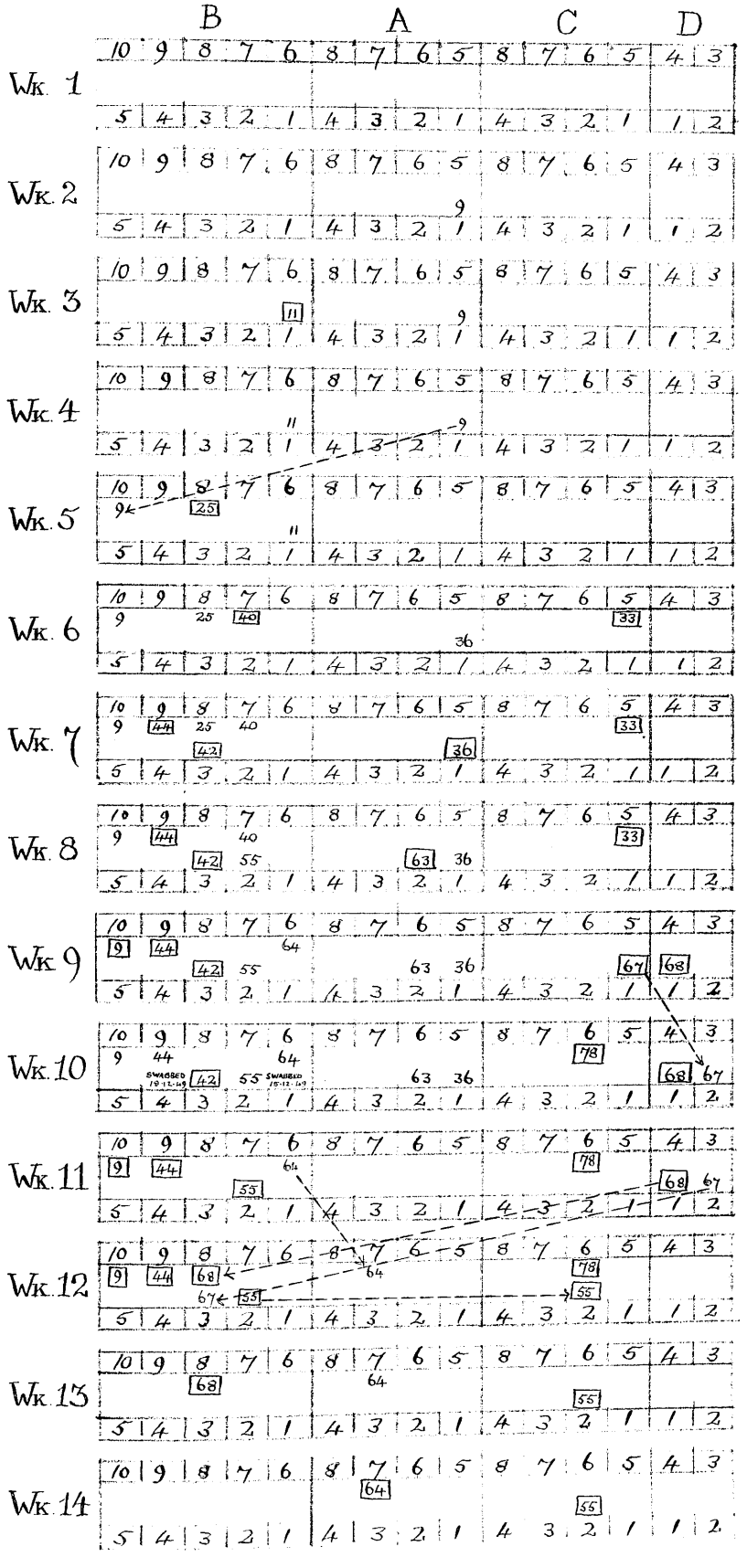


DIAGRAM 14

PNEUMOCOCCUS TYPE 19



Patients acquiring Type 19

No. 9 in Week 9

No.36 in Week 7

No.55 in Week 11

No.64 in Week 14

Isolations from Staff

No.33 in Week 14

DIAGRAM 14

(CON'D)

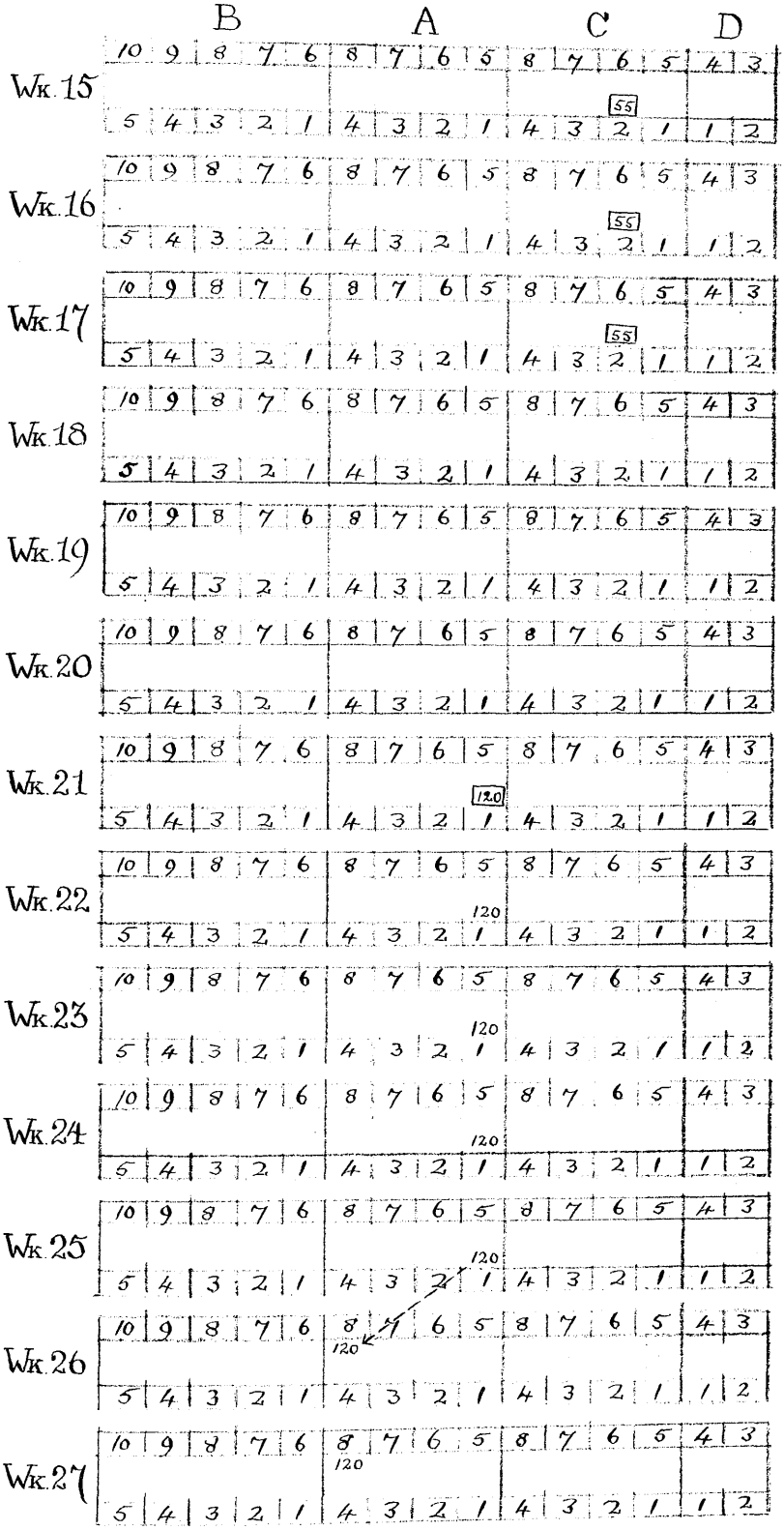
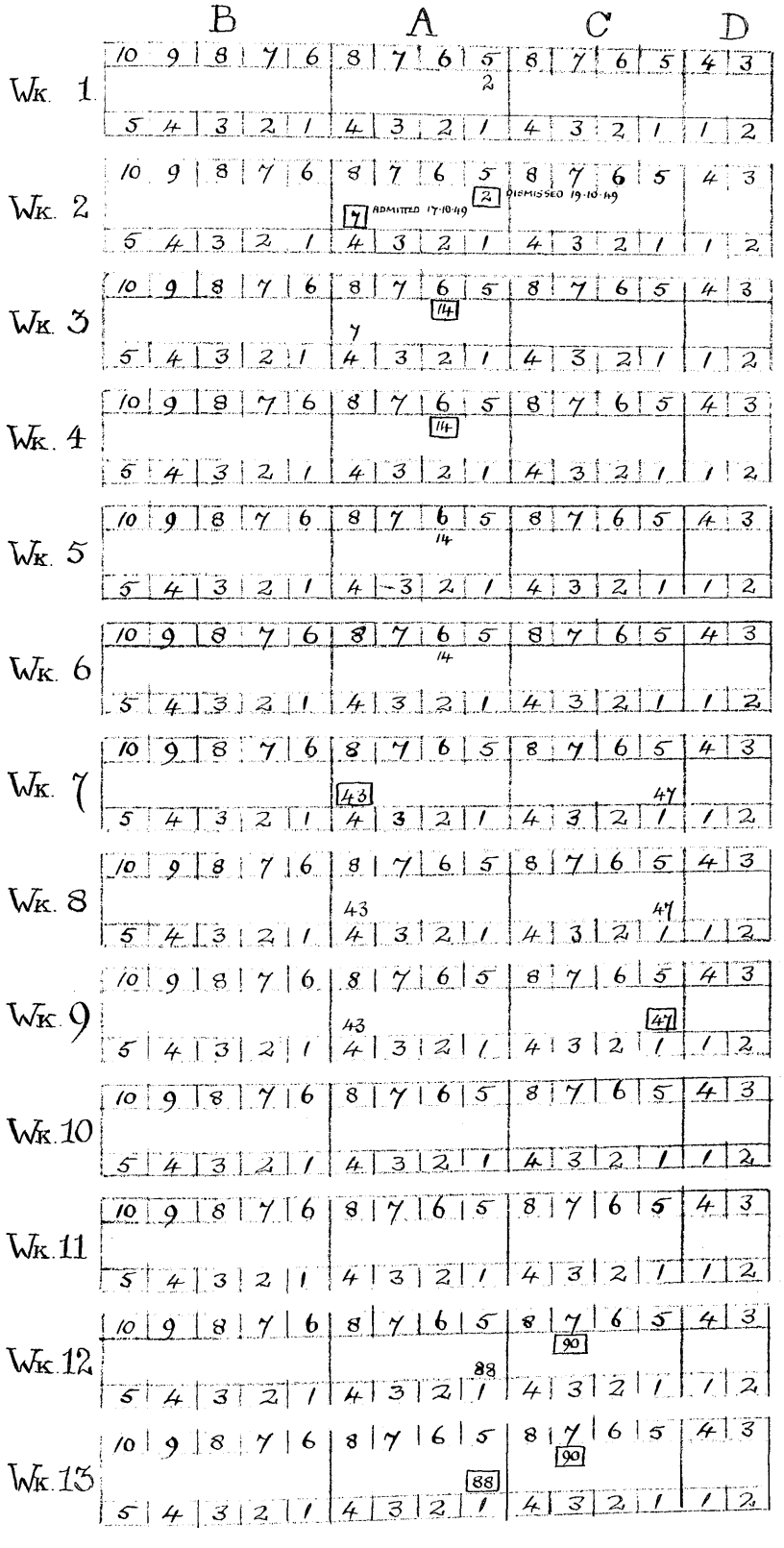


DIAGRAM 15

PNEUMOCOCCUS TYPE 22



Patients acquiring Type 22

No. 2 in Week 2

No. 47 in Week 9

No. 88 in Week 13

No. 109 in Week 21

Staff acquiring Type 22

No. 6 in Week 22

No. 24 in Week 9

Other isolations from staff.

None

DIAGRAM 15
(CON'D)

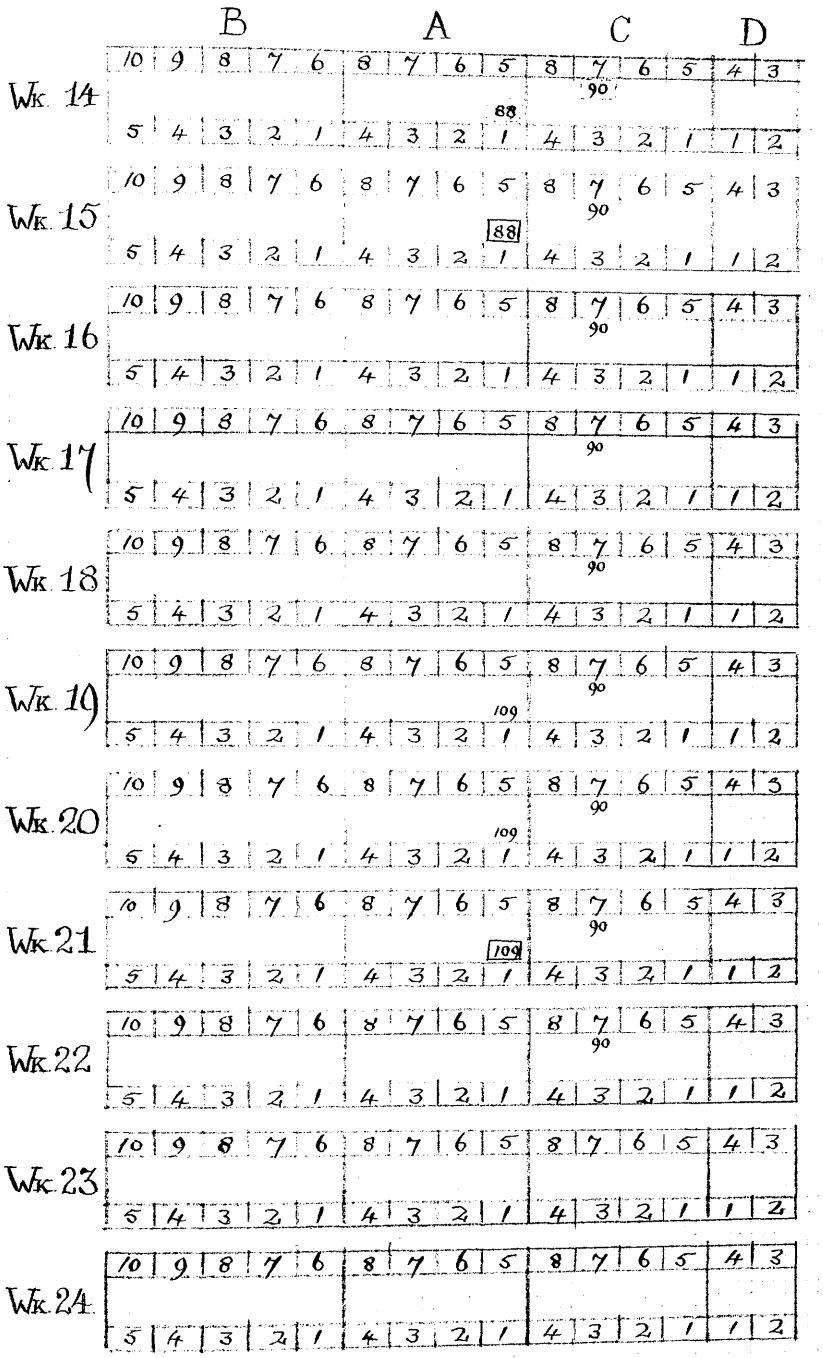
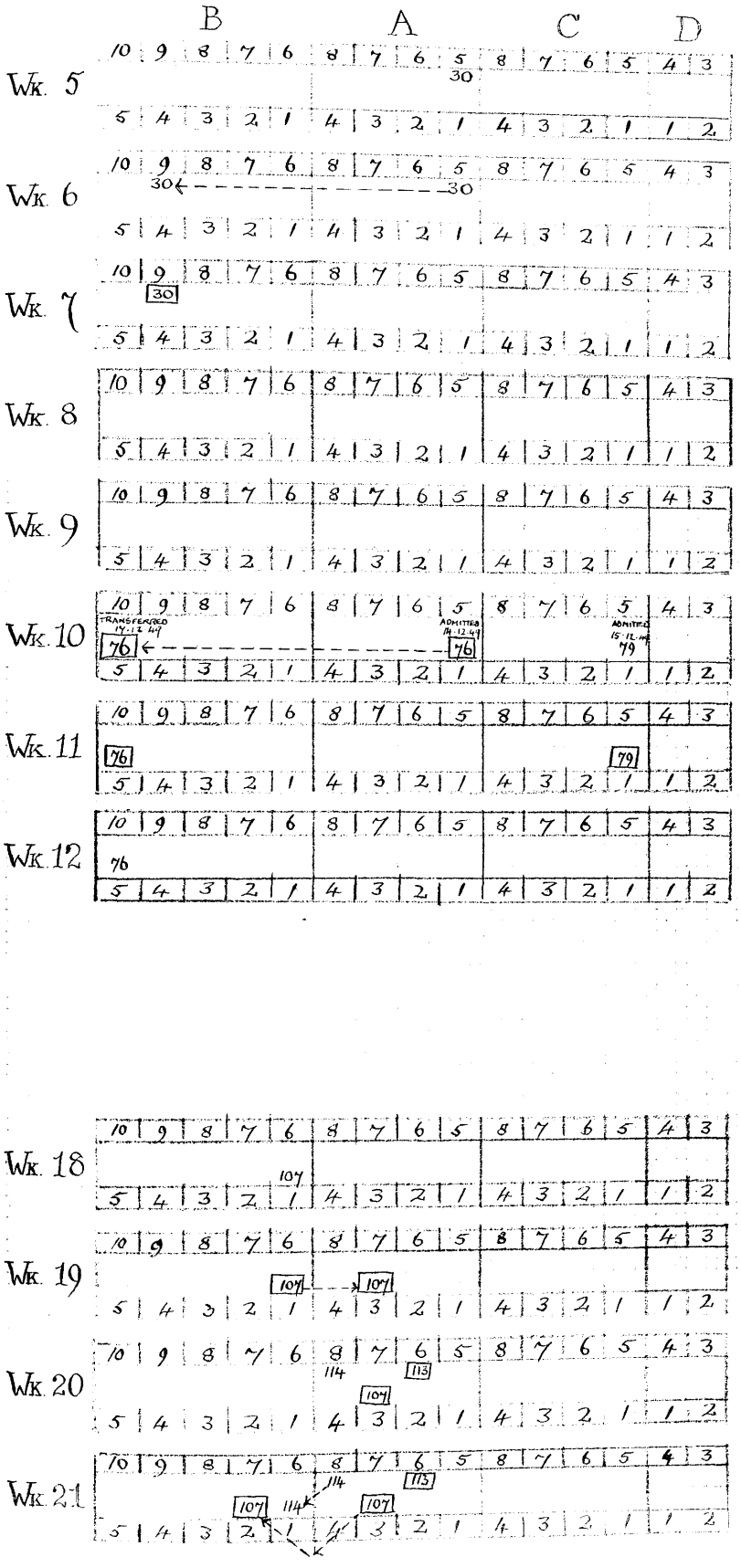


Diagram 16

Pneumococcus type 33



Patients acquiring Type 33

No. 30 in Week 7

No. 79 in Week 11

No. 107 in Week 19

No. 114 in Week 22

Isolations from Staff

None

DIAGRAM 16
(CON'D)

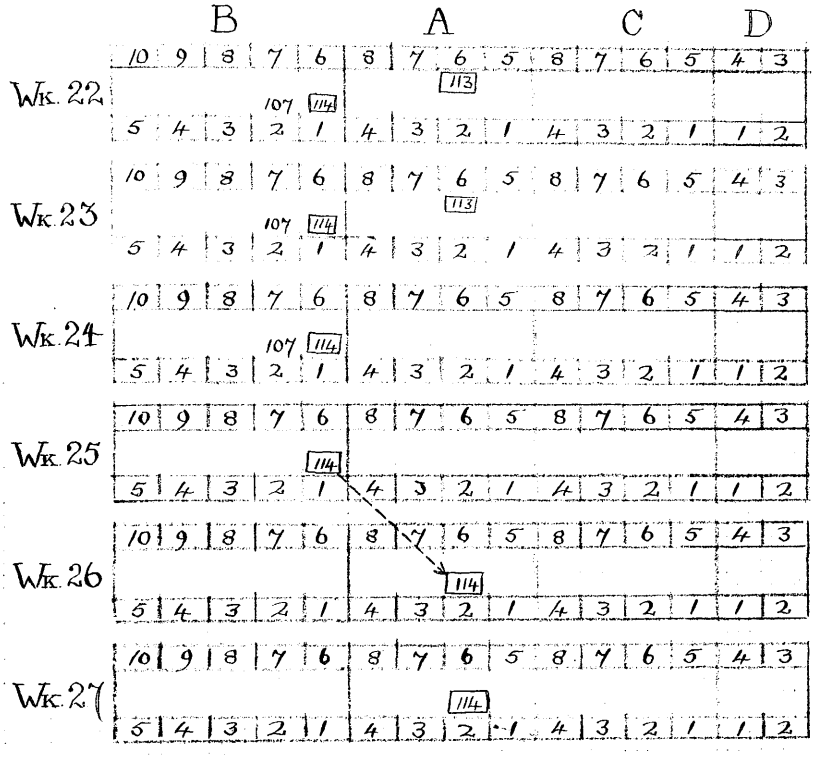
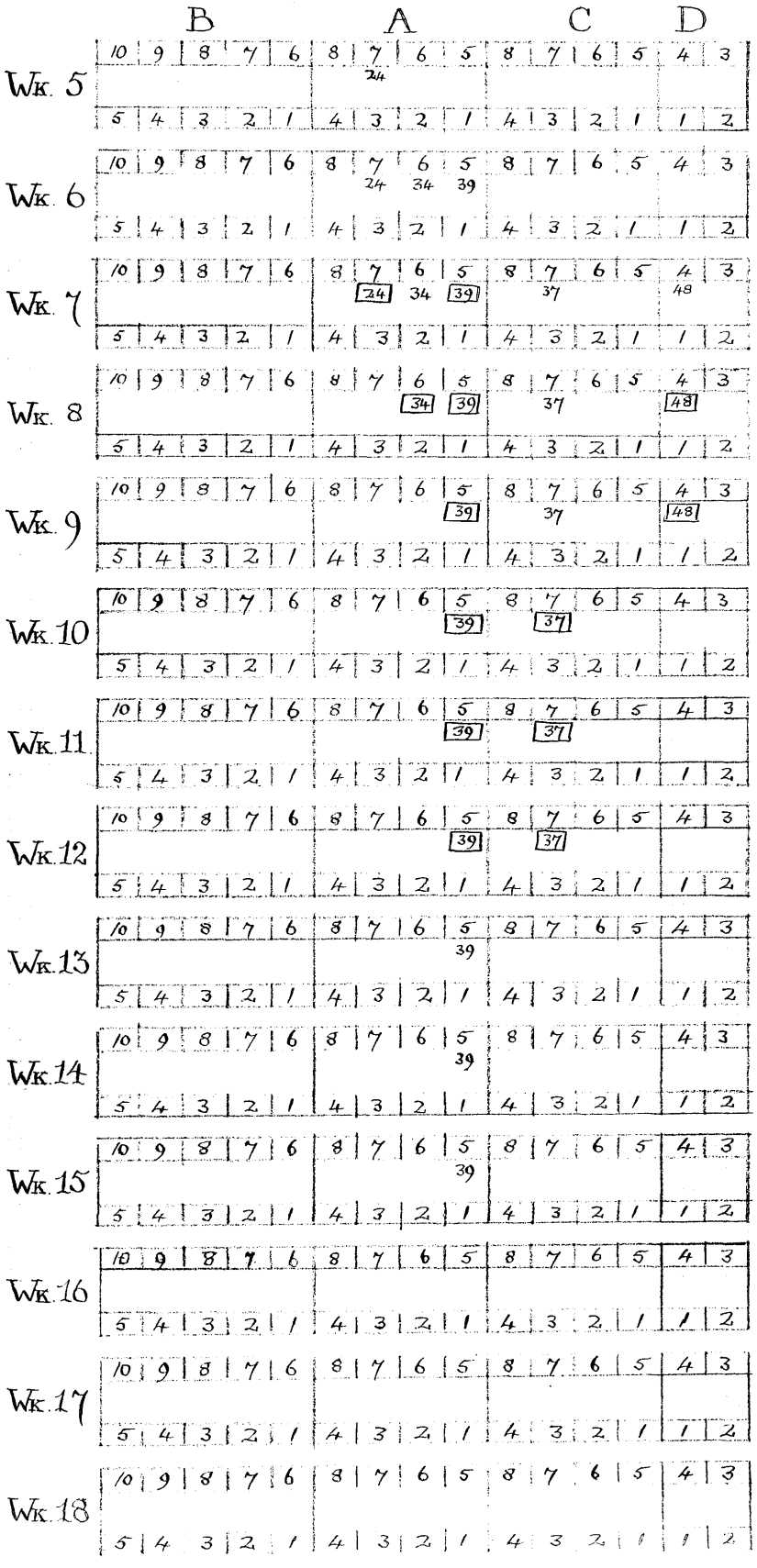


DIAGRAM 17

PNEUMOCOCCUS TYPE 4



Patients acquiring Type 4.

No. 24 in Week 7

No. 34 in Week 8

No. 37 in Week 10

No. 39 in Week 7

No. 48 in Week 8

No.110 in Week 21

Isolations from Staff

None

DIAGRAM 17

(CON'D)

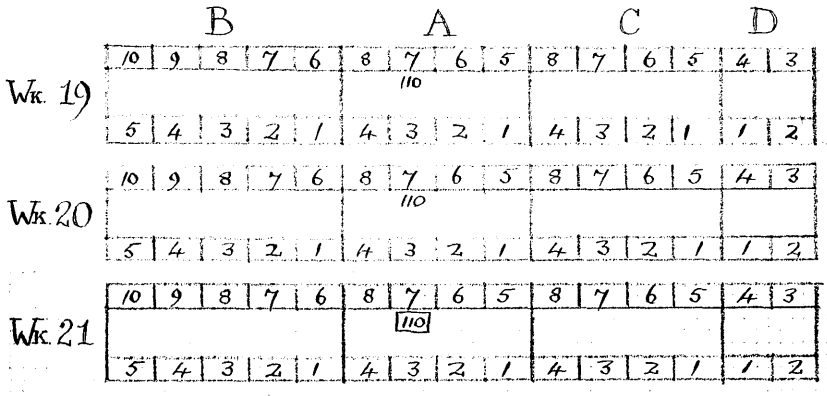
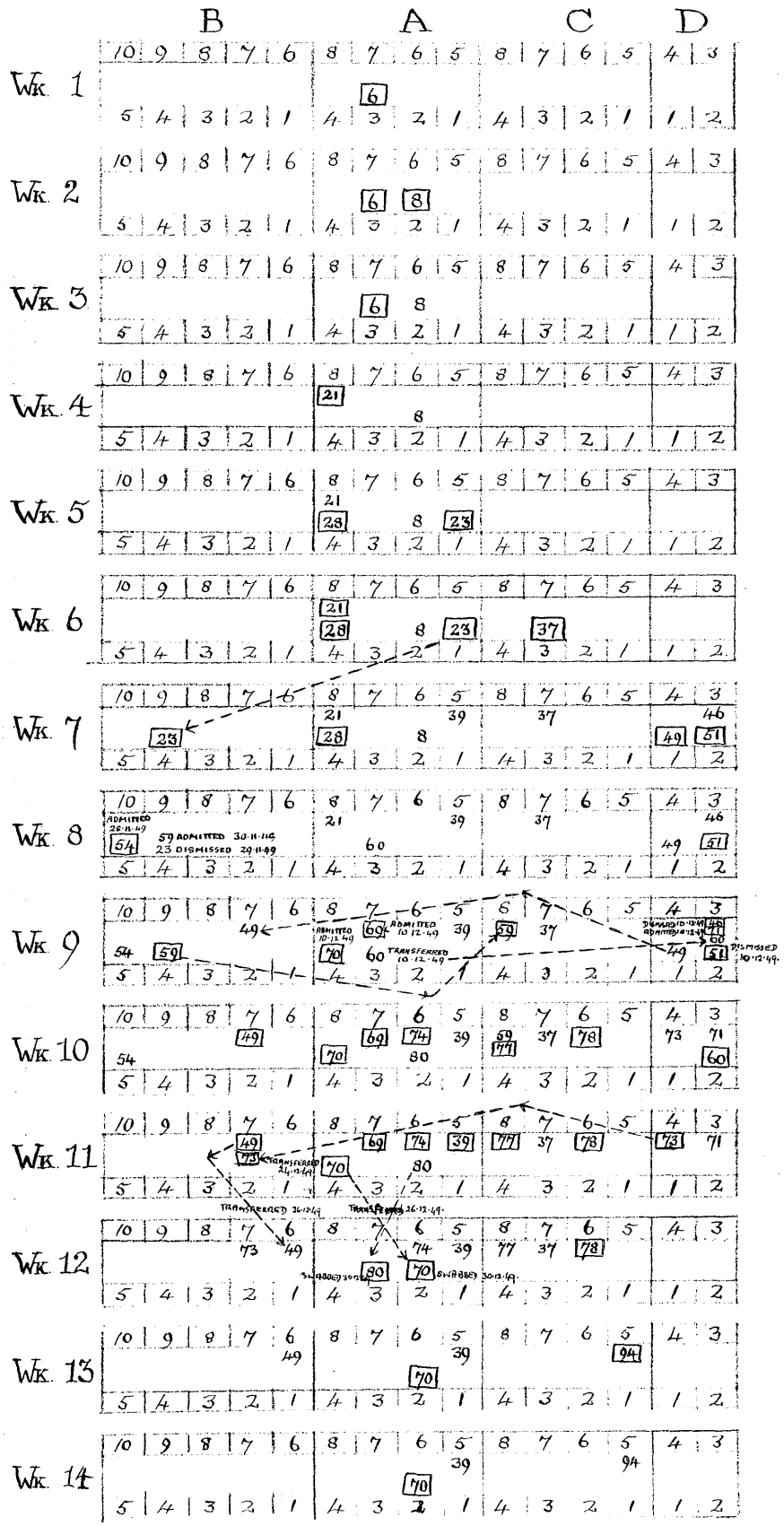


DIAGRAM 18

PNEUMOCOCCUS TYPE 23



Patients acquiring Type 23

No. 46 in Week 9

No. 59 in Week 9

No. 60 in Week 10

No. 39 in Week 11

No. 73 in Week 11

No. 80 in Week 12

No.104 in Week 17

Staff acquiring Type 23

No. 15 in Week 7

No. 17 in Week 9

Other Isolations from staff

No. 17 in Week 11

DIAGRAM 18
(CON'D.)

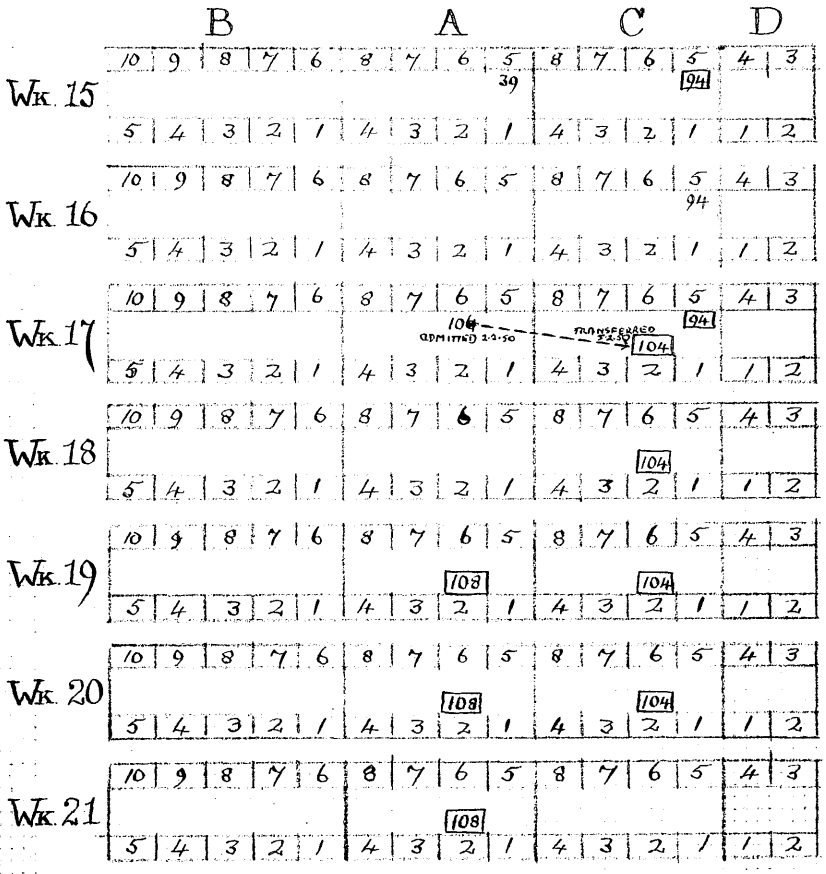
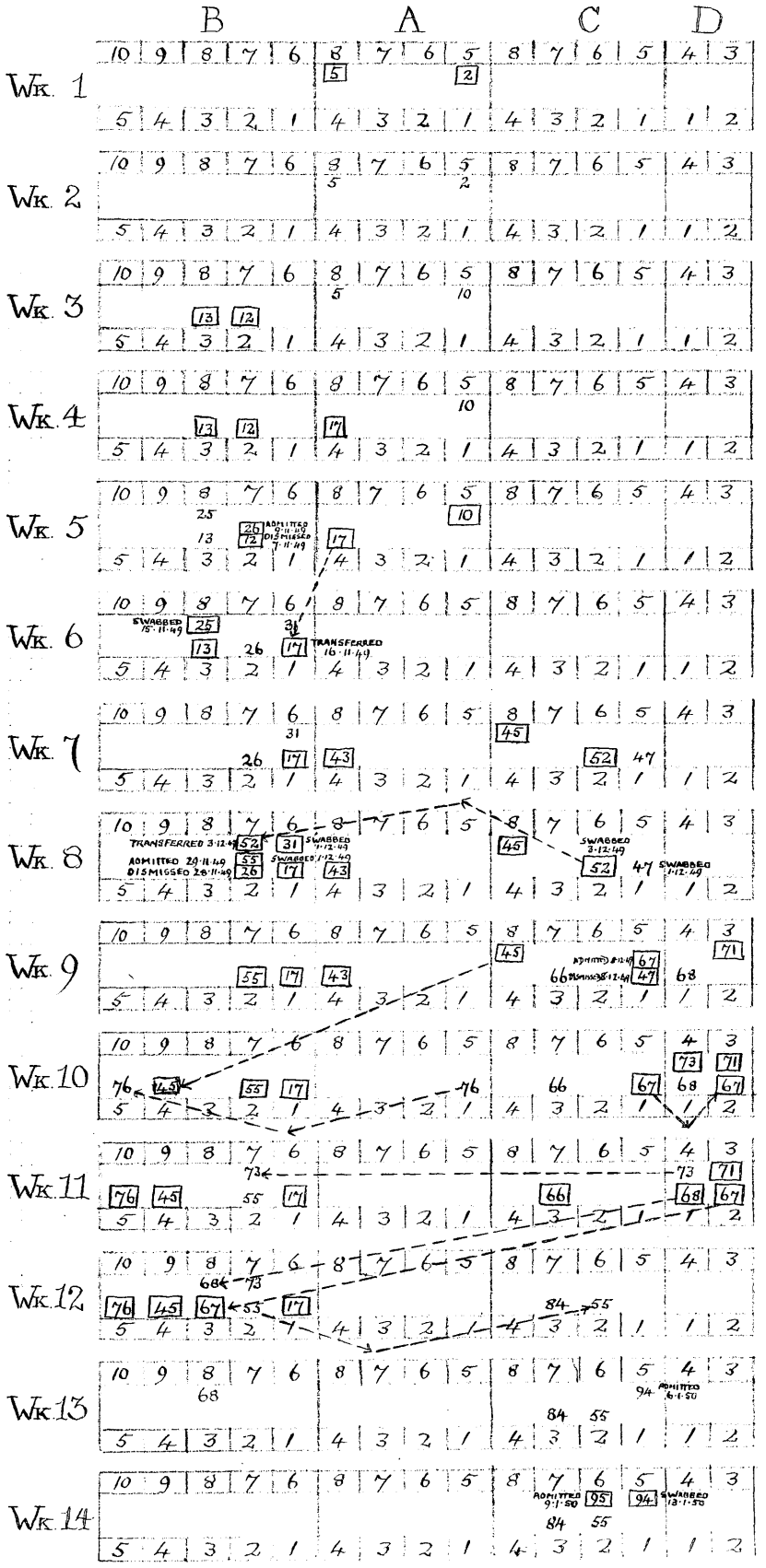


DIAGRAM 19

PNEUMOCOCCUS TYPE 6



Patients acquiring Type 6

No. 10 in Week 5

No. 25 in Week 6

No. 31 in Week 8

No. 47 in Week 9

No. 66 in Week 11

No. 68 in Week 11

No. 76 in Week 11

No. 84 in Week 15

No. 94 in Week 14

No. 98 in Week 17

No. 99 in Week 17

No.106 in Week 19

Staff acquiring Type 6

No. 1 in Week 10

No. 11 in Week 4

No. 13 in Week 9

No. 18 in Week 8

Other Isolations from staff

No. 14 in Wks.2-13

No. 24 in Week 7

DIAGRAM 19
(CON'D)

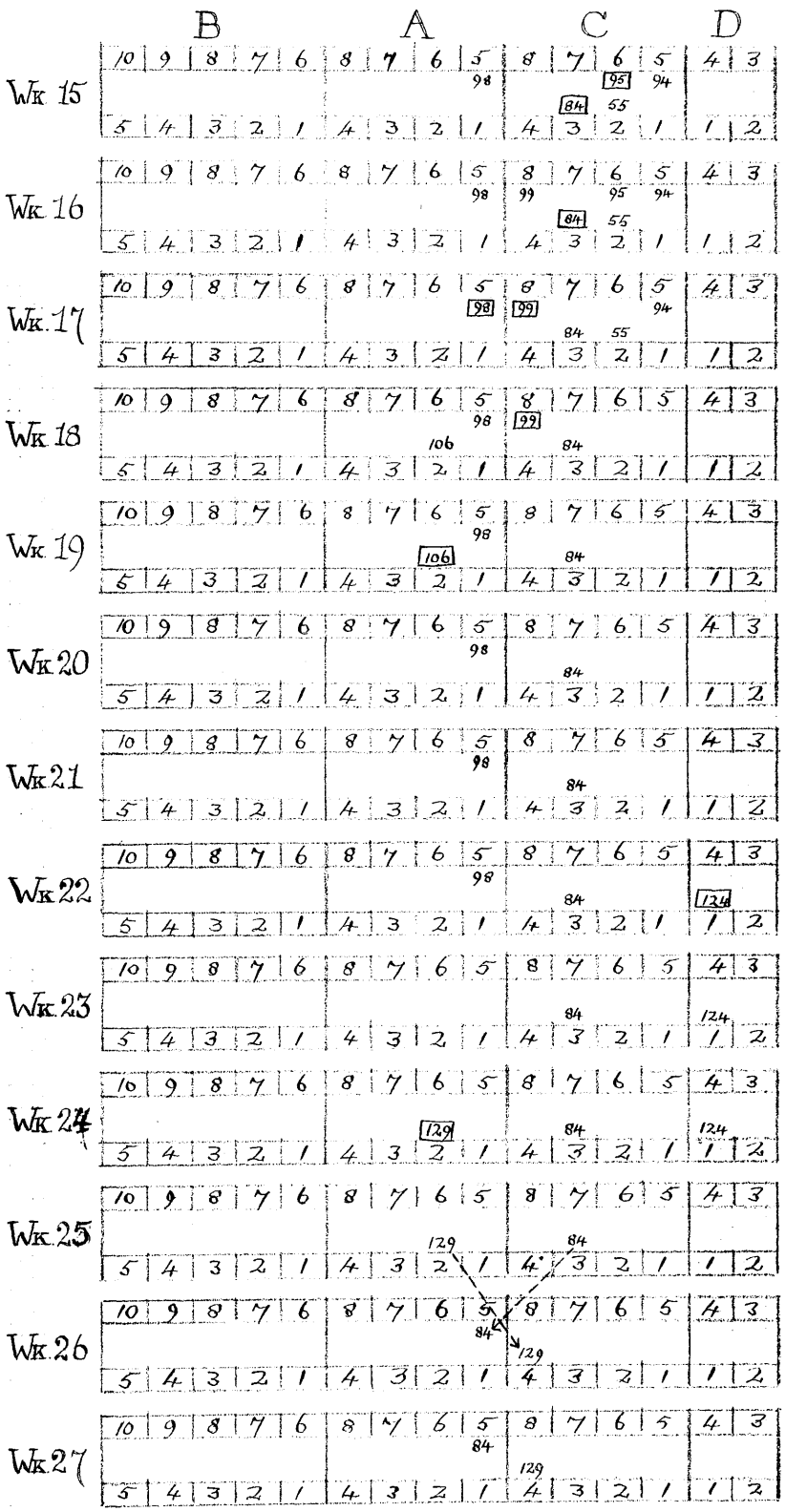


Diagram 20.

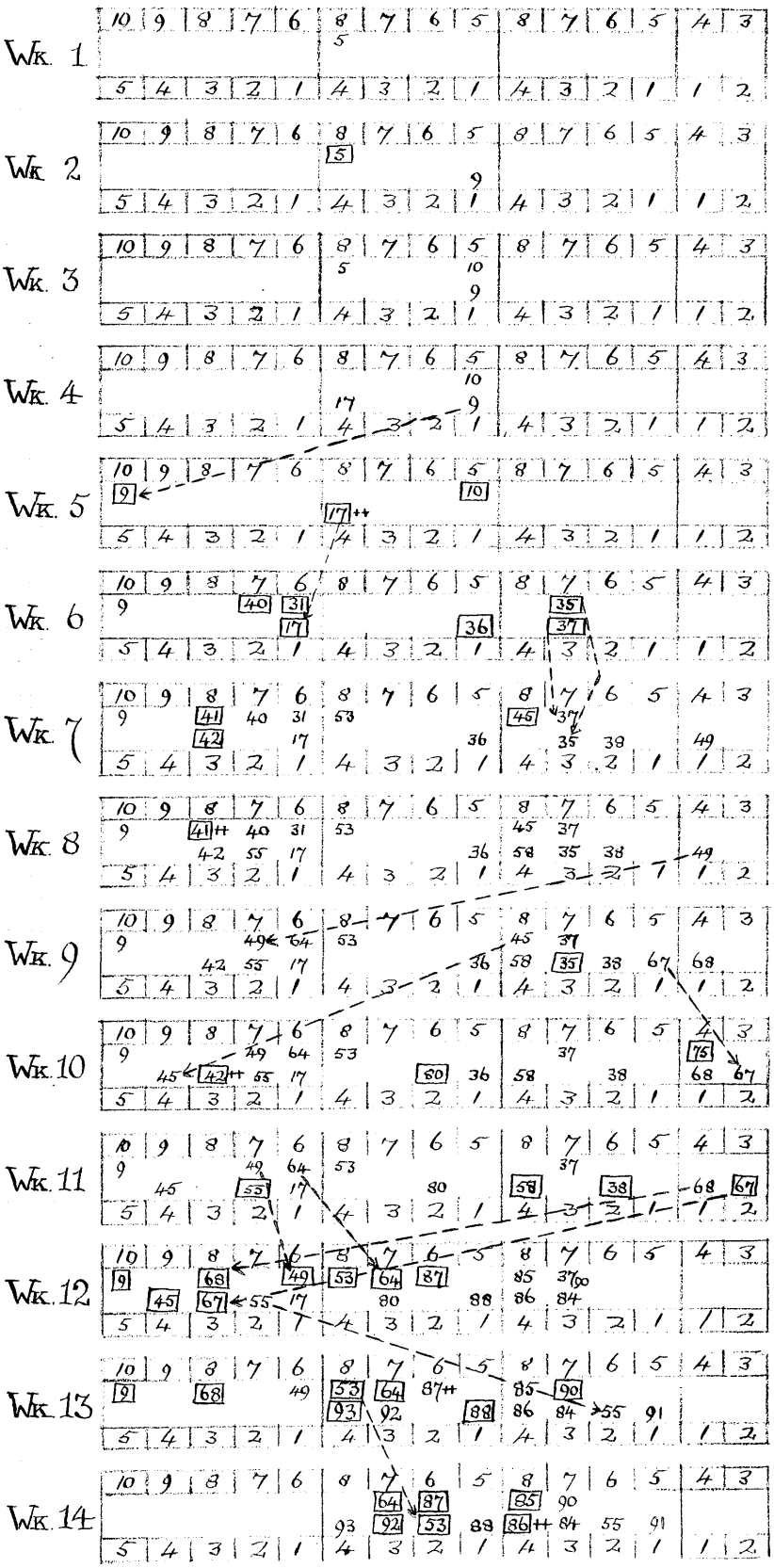
Types acquired by members of staff but never by patients.

Type	Serial No. of Staff.	Week when acquired.	Remarks
3	18	5	Never isolated from patients.
29	9	10	Isolated from patient No.86 in Weeks 12 and 15
14	12	3	Not isolated from any patient until Week 13
14	33	16	Patient No.94 carried Type 14 in Weeks 13 and 16.
35	24	15	Never isolated from patients.
35	28	13	Never isolated from patients.
37	31	13	Never isolated from patients.

In none of the above instances did another member of staff carry the type in question.

DIAGRAM 21

STREPTOCOCCUS PYOGENES



Patients acquiring S.pyog.

No.5	in Wk.2
No.9,10,17	in Wk.5
No.41	in Wk.8
No.42	in Wk.10
No.38,55,58, 67	in Wk.11
No.9,45,49,53, 64,68	in Wk.12
No.84,88	in Wk.13
No.85,86, 92	in Wk.14
No.90,91	in Wk.16
No.98	in Wk.18
No.102,104	in Wk.19
No.101	in Wk.21
No.113	in Wk.22
No.84,124	in Wk.23
No.114	in Wk.26
No.128	in Wk.27

DIAGRAM 21

(CON'D.)

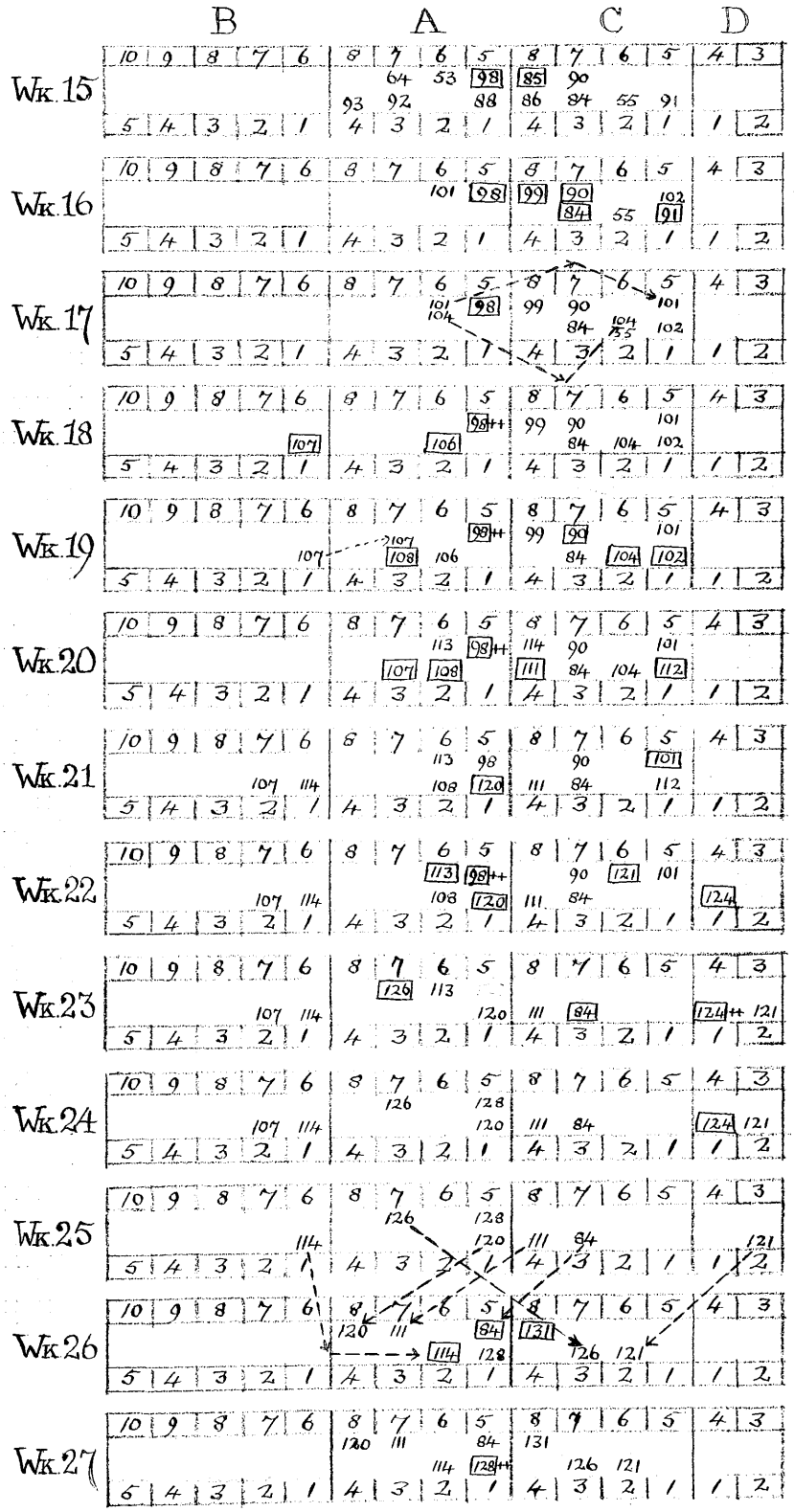
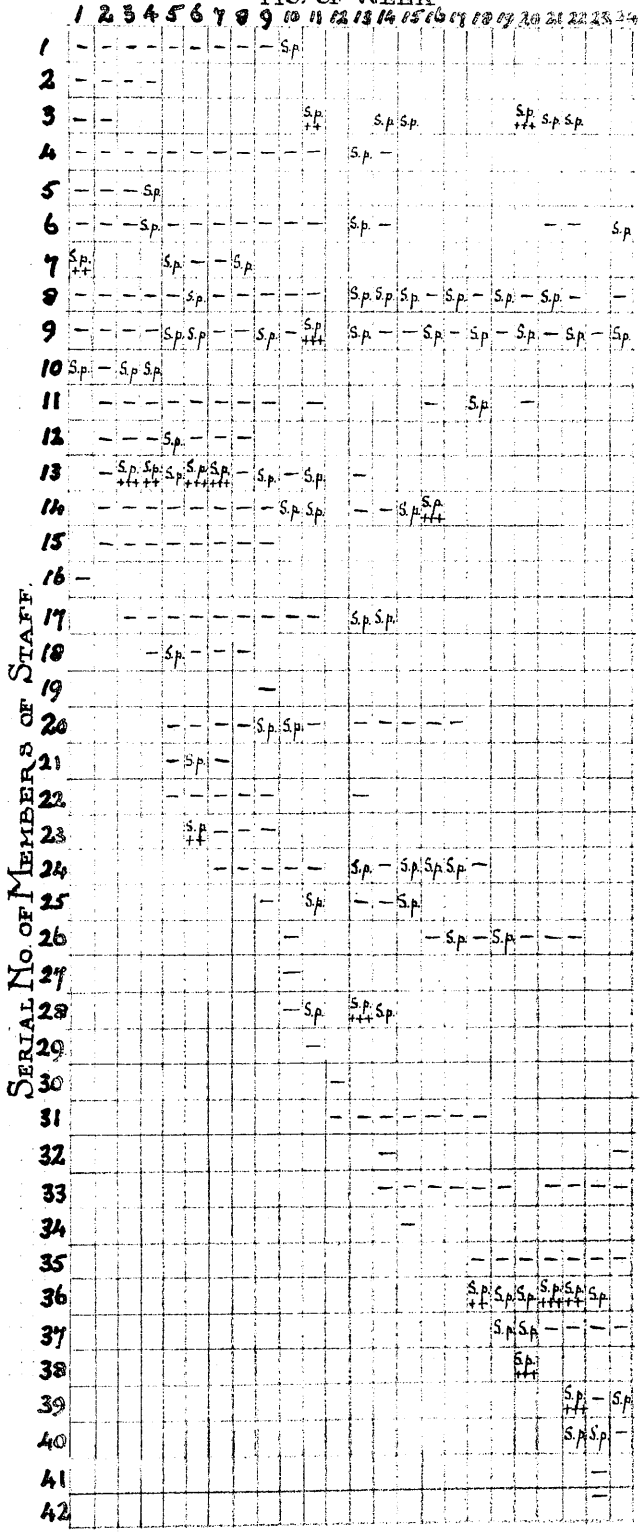


Diagram 22

The incidence of Streptococcus pyogenes amongst members of Staff



Members of Staff acquiring S.pyog.

No.13	in Wk. 3
No. 5,6	in Wk. 4
No. 9,12,18	in Wk. 5
No. 8, 21	in Wk. 6
No.20	in Wk. 9
No. 1,14	in Wk.10
No. 3,9,25,28	in Wk.11
No. 4,6,8,17,24,28	in Wk.13
No.14	in Wk.16
No.26	in Wk.17
No.11	in Wk.18