milk is inadequate. Finally, in houses and hospitals each mother should have her own room. If this can be afforded for the inmates of prisons it should be possible to afford it for the mother of babies. In her own room she would be free not only from the contagion of bacterial disease but also the contagion of desire to bottle-feed which at times spreads from mother to mother in a big ward.

Probably the most important cause of failure to breast-feed among those who desire to do so is the temporary inhibition of lactation which occurs shortly after the lying-in period when the mother returns to her household duties. This is interpreted as a final disappearance of the milk, with the result that she resorts to bottle-feeding. This could be overcome to a large extent by allowing the mother to be out of bed for a few days in the hospital before returning home, and by warning her that the disappearance of the breast-milk will probably last only for a day or two and that it can be restored with perseverance. I shall quote one more example to illustrate the faults of the mechanization of breast-feeding. Some time ago I was summoned to the house of young parents to see a three-months-old infant. The father was a lecturer in science, the mother a recent graduate of a university. The bookcase in unstained oak, the bare off-white walls, and the weekly political journals denoted their type and tastes. She was an earnest woman, breast-feeding her infant and anxious to continue. The complaint was that the child had failed to gain weight for a month and was but little over its birth weight. It was much troubled by frequent green stools and restlessness. I suggested that the child had no disease, but was receiving too little nourishment. This was denied by the mother, who produced figures and charts to show that test weighing had been used for every feed since birth, and that the infant had received the exact physiological amounts of breast milk—so many ounces a day for each pound of its body weight. She quoted authorities at me to prove her point. I persuaded her to discard the test weighing and to feed the infant five or six times a day until he appeared satisfied. In ten days he gained a pound, and there was no more trouble and no more test weighing. This over-mechanization is again seen in the frequent appeal to know whether a baby should be fed at four-hourly or three-hourly intervals. I would submit that the reply to this should be that the mother should cultivate an instinctive recognition of her own baby's needs, that she should come to know when the baby is satisfied, and that she should then choose the times of feeding most suitable to herself and her child. Only in difficulties should professional advice be sought.

Conclusion

It appears that the incidence of breast-feeding is declining. The causes of this are our increasing indifference to the value of breast-feeding and a mismanagement of the mother in the early stages of lactation. The correction of the fault lies mainly in the hands of those who tend and advise the mother in and shortly after her lying-in. Big hospital wards and crowded child welfare centres do not provide an atmosphere which encourages breast-feeding. Better education of doctors and maternity nurses in the care of infants may help to correct the fault; and that the importance of this education is not sufficiently recognized is suggested by the recent authoritative order of the Ministry of Health that panel practitioners attending their refreshing postgraduate courses are not to receive instruction in diseases of infancy or childhood.

THE SPREAD OF STREPTOCOCCAL DISEASE

BY

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Studies in the epidemiology of streptococcal disease are appearing with increasing frequency, and during the past ten years certain new principles have evolved concerning the prevention of these infections. Clinical practice has, however, been slow to apply these important principles. The obstetricians, stimulated by public anxiety concerning puerperal sepsis, have made valuable contributions to these studies, and the story of their success has recently been told by Professor Miles H. Phillips (1938); but the rest of the profession would appear to lag behind both in practice and in teaching. This is unfortunate, because in some quarters the obstetrician's particular interest in puerperal sepsis has given a specialist bias to a problem of general concern. For puerperal sepsis accounts for a small part only of the total morbidity and mortality attributable to *Streptococcus pyogenes*, and it seems not improbable that the practice of obstetrics is, in fact, less affected by the droplet spread of streptococci than are the other branches of clinical medicine and surgery. From the public health point of view it matters little through which portal the streptococcus or its toxins invade the body, and it is not always realized that what is written about the prevention of puerperal fever applies also to all disease spread by droplet contagion. But since puerperal fever has established itself as the classic type of grave streptococcal disease *sine eruptione*, it might be well for us to read again Phillips's historical note and add "*et alia*" after each mention of puerperal fever; for what applies to the classic type is equally applicable to scarlet fever, erysipelas, cellulitis, most wound infections and septicaemia, the bulk of nasopharyngeal and otic suppuration, many pneumonias and, probably, acute rheumatism and acute nephritis.

The crux of the whole matter is a paradox: an isolated case of puerperal fever is in itself evidence of an epidemic. It implies a widespread prevalence of streptococcal disease, and a slight rise in maternal mortality is paralleled by a much greater toll from other streptococcal accidents. If puerperal fever is preventable so are these other accidents, and information as to how they can be controlled is rapidly accumulating. Let us review some of the more recently established facts.

Bacteriology of Streptococcal Infections

Old nomenclatures are a hindrance. Okell's (1932) Milroy Lectures provide a good text. We are now concerned almost entirely with *Streptococcus pyogenes*, for this is the group most commonly pathogenic to man. *Str. viridans* and non-haemolytic organisms, apart from an occasional strain of *Str. pyogenes* which fails to give β haemolysis, are, except when they invade an unusual location such as a previously damaged endocardium or urinary tract, of little significance as disease producers and must be looked upon as part of the normal bacterial flora of man. The part played by anaerobic streptococci is not yet measured, but on clinical grounds it would appear to be of minor importance. The classification of haemolytic streptococci—and for our present purpose it is safe to say that we are almost entirely con-

cerned with those giving β haemolysis on horse-blood-agar plates—has been simplified by the introduction of serological typing. No longer does definition of a given organism depend on its morphological and cultural characters, which now become of academic interest only. We can forget such names as "*Streptococcus longus*," "*Str. brevis*," and "*Str. mucosus*." We can also forget *Str. epidemicus*, *Str. scarlatinae*, *Str. erysipclatis*, *Str. anginosus*, etc., and with them discard the "specificity" hypothesis which sought to associate a well-defined clinical entity with a particular and specific kind of organism. We now know that in any given epidemic a single strain of *Str. pyogenes* may produce angina, scarlet fever, erysipelas, cellulitis, lymphangitis, adenitis, otitis, sinusitis, meningitis, puerperal fever, septicaemia, etc. What Okell (1932) calls the "unitarian" hypothesis is now well tried, and must be accepted if we are to understand the very elements of streptococcal epidemiology.

The bulk of, but not all, haemolytic streptococci pathogenic to man are identifiable by a specific carbohydrate substance, and fall into Group A of Mrs. Lancefield (1928). This group is further subdivided into about thirty different "Griffith types" (Griffith, 1934), each characterized by a specific "M" substance upon which their identification by agglutination with absorbed sera depends. It is this typing technique which has made epidemiological studies and the proof of the unitarian hypothesis possible. A note of warning must be introduced here. These techniques are available in very few laboratories, and it is at present unfair to expect the majority of bacteriologists to undertake Griffith typing.

Epidemiology

The most striking epidemics of streptococcal infection are milk-borne. They are of comparatively common occurrence and are so impressive that they are believed by some to be the main source of spread. This is undoubtedly untrue, and in the epidemics about to be described no evidence of a food vehicle was found. Epidemiology is an oecological study, and the behaviour of an epidemic is influenced by its environment. For this reason one should hesitate to draw conclusions from isolated observations in time or space, and should be careful to pool as many experiences as possible before making deductions. The examples about to be described should be added to the pool, but as preliminary investigations they are of value because they deal with three distinct types of community. (1) *The semi-isolated community*: In this case a boarding school for boys in which contact with the outside world is limited and presumably the opportunity for introduction of new infection considerably restricted thereby. (2) *The general hospital*, in which the bacteriological environment of the inmates is particularly complex, since individuals living therein inevitably make contact with more diseased persons than they would meet with in their homes or elsewhere. (3) *The family*, which is probably the simplest unit of all and as such might well be the most satisfactory material for the study of a problem in clinical epidemiology.

Streptococcal Infection in a Boys' School

In Fig. 1 are given in graphic form the bacteriological findings from all upper-air-passage infections requiring more than twenty-four hours' exclusion from school in a community of about 320 boys during the years 1931 to 1934. The graph also includes the years 1929 and 1930,

but during this period bacteriological observations were incomplete. It will be seen that during the six years dealt with six different types of streptococcus became established and gave rise to the spread of disease. The main incidence of disease was in the Lent terms. In 1931 there suddenly appeared a new type of organism which gave rise to sixty-three cases. Nine other positive streptococcal cultures were obtained: one belonged to the type of which the school had had previous experience, and eight were unidentifiable. They may not have been Group A organisms. *Str. pyogenes* was not recovered from forty-one cases, and it was clear that streptococcus Type 18 gave rise to the bulk (60 per cent.) of the cases encountered in the school at this time. In the following term (1931, II) one case only of Type 18 infection was

FIG. 1.—Serological types (Griffith) of *Streptococcus pyogenes* producing nasopharyngeal infections in a semi-isolated community (boys' public school, population 300 to 320).

found, although about the same number of non-coccal infections occurred in the school. Streptococcal fever had almost disappeared. In the third term of 1931 a new type of organism, Type 13, was first detected, and continued to produce disease for five consecutive terms. In the last two terms (1932, III; and 1933, I) a number of carriers of this organism were discovered, and were detected occasionally until the Lent term of 1934. (Their presence is indicated by a small circle "o" in Fig. 1.) Type 13, however, seemed to have either lost its power to become epidemic or the school was effectively immunized against it.

During the Lent term of 1932 another type of organism, which had at that time not been included in the Griffith classification and which is indicated in Fig. 1 as Type "X," made its appearance and gave rise to the bulk of the disease affecting the community. It appeared again at the end of the year, since when it has not been encountered. Type 1, a well-recognized scarlet fever strain, was first recovered from the throat of a new boy who showed no signs of disease, and it was not until fifteen months later that clinical cases of Type 1 infection

began to occur, and gave rise to an epidemic of forty cases of nasopharyngitis.

Although during the two subsequent terms four carriers of Type 1 were encountered, no further epidemic spread of this organism followed. At the beginning of, the second term of 1933 a wave of non-streptococcal colds occurred. A month later, on June 11, in brilliant summer weather, a single case of scarlet fever appeared. Streptococci were not discovered in the throat, and the portal of entry appeared to be a trivial scratch on the boy's foot. It is interesting to record that a streptococcus giving a clinical picture very suggestive of Type 1 caused an epidemic in a neighbouring village during October and November, 1933, and there was some evidence that the infection had been carried by a school servant and had been kept going through the medium of one or two apparently sporadic cases of sore throat seen during the summer months. At the beginning of 1934 Type 6 gave rise to a small epidemic, and was detected in the school during two terms, after which it completely disappeared from the community; during 1935 and 1936 there was no epidemic spread of streptococcal disease, this organism having apparently been replaced by pneumococci.

It is tempting to speculate further upon the immunological implications arising out of Fig. 1. It was quite certain that infection with, let us say, Type 18 gave no immunity against infection with Type 13; in fact, there were some boys who reacted to all six types of infection. Other boys, although equally exposed, reacted to none. It is also possible that as the result of so many contacts with streptococci some form of group immunity had begun to result by 1933 and was effective in 1934 and 1935, when the health of the school was unusually good, but this improvement was more closely associated with a deliberate attempt to improve the hygienic conditions within the school by reducing, in September, 1933, the number of the community from 340 to 230 boys, and thereby attaining much more effective spacing in dormitories, classrooms, and playrooms.

The six epidemics described showed certain features which have now been seen so frequently that they may be considered almost characteristic of the epidemiological picture produced by the droplet spread of streptococci. Two of the epidemic periods have been described in greater detail elsewhere (Bradley, 1932a, 1932b), and can be compared with the observations of other workers (Glover and Griffith, 1931). Furthermore, one may conclude that streptococcal disease, unless milk-borne, spreads slowly and does not give rise to steeple-like epidemics or overwhelm communities with dramatic suddenness. The maximum incidence in the Lent term has already been referred to. Three or four weeks of a term often elapsed before cases occurred with sufficient frequency to cause anxiety of epidemic spread, and another month had passed before the epidemic had reached its peak, after which the decline in case incidence was gradual. The spread of infection was intensified by the simultaneous presence or previous occurrence of other catarrhal disease such as "influenza," non-coccal colds, or measles, the last-mentioned having a marked effect in elevating the passage of streptococci. The complications of influenza and measles were attributable to the streptococcal types producing coincident infection in the sick-rooms. On one occasion thirteen boys, with negative throat swabs at the time of admission to a sick-room for a non-coccal pyrexial nasopharyngitis, were brought into contact in the same ward with a boy suffering from primary streptococcal

fever. Five of the thirteen, occupying beds adjacent to that of the streptococcal case, contracted secondary streptococcal disease; and a sixth, occupying a bed on the other side of the ward, also became involved (Bradley, 1932b). This is one of many similar examples in which ignorance allowed grave disease to spread among already sick boys. A more enlightened physician would have prevented the introduction of a streptococcal case into a non-streptococcal ward, and so have avoided a fatality. At present such a mistake can be excused because it requires extensive clinical experience to distinguish between streptococcal and non-streptococcal nasopharyngitis without bacteriological evidence. There are, however, no practical difficulties, cost excluded, against the immediate application of simple bacteriological techniques to this problem. Failing such aid, and even in epidemic times, every boy with nasopharyngitis of unexplained aetiology must be looked upon as a danger to the community and nursed on the barrier system until it is proved that he is not suffering from an infection not already involving his fellows.

It will be seen how during each term one, and occasionally two, types of *Str. pyogenes* established themselves and gave rise to epidemic disease, so that towards the middle of the term the flora of the school had become fairly constant, and the bulk of infection encountered was attributable to one Griffith type. Some months later another type would appear and apparently overgrow its predecessor. The latter would be recovered occasionally only, was not the obvious cause of new disease, and was apparently residing in an immune carrier. Strangely enough, these carriers did not start off new epidemics—possibly because, in a closed community, they were living in an immunized population. If this suggestion is tenable it is strong evidence that a person immune to one Griffith type is not protected against invasion by another. He will, however, be partially or completely protected against the rash-producing Dick toxin, which is produced in varying amounts by most, if not all, types of *Str. pyogenes*. The incidence of scarlet fever is therefore fortuitous and is no measure of the extent of streptococcal parasitism.

It may be assumed that there were six emergency periods when new infection was introduced to the school, and it is probable that had intelligent action been taken when first these infections were detected much of the sickness in the school could have been averted.

Spread of Streptococci in a General Hospital

These studies have been attempted in a provincial hospital. They are not comprehensive, since it has at times been difficult to obtain information regarding the occurrence of P.U.O. or nasopharyngitis, and in certain wards the investigations have, for one reason or another, been partial or inadequate. It is, however, probable that Fig. 2 is significant for about 120 hospital beds, mainly in the medical wards. During the twelve months beginning November, 1936, six types of *Str. pyogenes* gave rise to multiple cases of infection contracted during the patients' stay in hospital. Fifty such infections were observed, in seventeen of which scarlet fever, due to four Griffith types, was diagnosed. Two deaths were directly attributable to these infections; two other infected patients died prematurely. The problem is therefore one of considerable importance. The obstetrical, and ear, nose, and throat wards were not included in the investigation, and scant attention was given to the surgical wards. Yet it is in the last that ward infections with *Str. pyogenes* are known to do most damage. The hospital under

observation is well spaced and well equipped; the hygienic conditions are above the average. Fig. 2 attempts to show the probable direction of spread, but it is obvious that some intermediaries between the homologous cases were not detected, and that the lines of spread indicated in

Yet the tendency was to relate the scarlet fever to a common source of infection, although, in fact, four distinct sources were responsible, one of which could be traced to a child admitted to the hospital with otitis media from a home from which two children had been removed to

isolation with scarlet fever during the preceding fortnight. In three of the epidemics there was evidence that nurses had acted as intermediaries in the transmission, but there was nothing to suggest that nurses had been more active than other people in introducing the infection into the hospital in the first instance. Furthermore, where the spread was sufficient in amount to allow for deductions the evidence pointed rather to inter-bed spread. One of the most striking features of these infections was that they ended as suddenly as they began, although no special action was taken to control them. In two cases the spread came to an end immediately after dissemination to other wards had been observed.

Fig. 3 represents the picture from September, 1937, to January, 1938. During this period four and possibly five more infections were introduced.

We then tried to identify the source of the organisms, a fair picture of streptococcal flora of the hospital having been established during the previous months. Two cases of Type 25 infection followed the introduction of this type in a patient suffering from quinsy; a case of cellulitis gave rise to fifteen secondary cases of

Type 6 infection. The routes of spread of Types 22 and 6 have been extracted from Fig. 3 and represented separately in Figs. 4 and 5. The Type 11 infection was apparently introduced by a patient with suppurating cervical glands; two of the secondary cases also had suppurating of the glands of the neck. It is not improbable that two Type

FIG. 2.

Scarlet fever
Scarlet, sine eruptione
Cold or sore throat
9 1 6=Griffith type of *Streptococcus pyogenes*

the diagram are in some instances guesswork. In the absence of an investigation of this type it would have appeared to a casual observer that scarlet fever was endemic in the hospital. Occasionally cases of scarlet fever were grouped, but more commonly it was impossible to associate them with other cases of this exanthem.

FIG. 3.

Scarlet fever
Scarlet, sine eruptione
Cold or sore throat
9 1 6=Griffith type of *Streptococcus pyogenes*

FIG. 4.

Scarlet fever
Scarlet, sine eruptione
Cold or sore throat
9 1 6=Griffith type of *Streptococcus pyogenes*

28 cases followed the introduction of this organism into the babies' ward by a case with otitis media due to the same type of streptococcus. A bacteriological "cross-section" of the hospital at this time, shall we say at the middle of December, would have shown an extremely complicated picture, since six types of infection were present, and it would have been impossible to draw conclusions from such an investigation. Other workers who have attempted to unravel this problem by serological methods have been disappointed by their results, not improbably because they have attacked the problem at a time when droplet infections of all sorts were particularly rampant. From Figs. 2 and 3 it is reasonable to conclude that the introduction of much of this disease to the hospital could have been avoided by the proper isolation of known cases of streptococcal disease, and that a considerable amount of dissemination could have been controlled had there been proper exclusion of nurses and doctors suffering from acute nasopharyngeal infections. At no time was there any need to postulate a chronic carrier, all spread being accounted for satisfactorily on the assumption that ambu-

FIG. 5.

— Scarlet fever
 - - - Scarlet, sine eruptione
 . Cold or sore throat
 x Griffith type of *Streptococcus pyogenes*

lant cases of or convalescents from acute nasopharyngitis were responsible. Again, in these hospital studies the slow tempo of streptococcal droplet spread is demonstrated. Three only of the fifty-one attacks of nasopharyngitis or P.U.O. represented in Fig. 3 were associated with erythematous rashes, and in none of these three cases was a diagnosis of scarlet fever made, although, of course, all the fifty-one cases (most of them being trivial in character) were potentially as dangerous to the community as fifty-one cases of scarlet fever.

In this connexion attention is drawn to Appendix A of Sir Arthur MacNalty's report on the state of the public health (1936), as it is probable that patients discharged from infected wards of a general hospital during epidemic times do considerable damage in their homes. Actually only three returned cases came to my knowledge. It will be appreciated that each small wave of infection was a separate problem, and that efficient prophylaxis at the very beginning of each wave would have made a considerable difference in the morbidity from these troubles.

Family Epidemics

There is considerable clinical evidence that the inhabitants of a small rural village will behave in much the same way as a semi-isolated community. In a larger population—for example, a county town—perhaps two or three types of streptococci will spread, while in a large city almost all the Griffith types may be encountered at different times during a period of twelve months. Thus the cultures obtained from fifty-six streptococcal infections in New York City during the first four months of 1936 fell into sixteen of the Griffith types (Bradley, 1937), and I am informed by Drs. Coburn and Pauli that during 1937 all the Griffith types were found to be producing disease in New York City. When, however, individual families are studied it is observed that the disease produced during a family epidemic results from one type of infection only, and many examples of this have been recorded elsewhere (Bradley, 1937). For the purposes of the present discussion one or two examples will serve to illustrate this point.

Case 1.—On February 20, 1936, a girl aged 12 contracted a pyrexial sore throat which lasted a week. On February 27 her brother, 15 months old, became ill, and later developed bilateral otorrhoea, and was under observation for mastoiditis. On February 28 another brother was taken ill with a cold, and ran a fever for three days. On March 3 the first patient, a known rheumatic subject, was admitted to hospital, where she developed an attack of carditis and polyarthritides which ended fatally. Type 6 streptococci were found in profusion in the throats of these three patients. Their mother had suffered a trivial sore throat on February 29, but throat cultures were free from haemolytic organisms by March 3.

Case 2.—In another family a girl aged 4 was removed to an isolation hospital on February 1, 1936, with a diagnosis of diphtheria, but was sent home on February 14, some doubt having been cast on the diagnosis. Two days later one brother developed a severe pyrexial cold with otitis, and after two more days another brother had a bad cold. The mother and a fourth child made no complaint of ill-health, but all five members of the family gave a profuse growth of Type 22 *Str. pyogenes*, and one boy developed an attack of rheumatism five weeks later, while still under observation.

Case 3.—On March 2, 1937, Gwendoline, daughter of a farm labourer in an English village, went to bed with a bad cold and felt ill until March 8. On March 7, however, she got up because her mother was delivered of a baby, the eighth in the family. She helped nurse her mother, who on March 10 became suddenly ill with puerperal fever associated with a scarlatiniform rash, from which she eventually died. Fourteen days after the onset of the mother's illness the father had "influenza," and Evelyn, aged 4, contracted a severe cold two days later, while still five days later, on April 1, Gladys, aged 17, had a cold, and on April 7 Fred, aged 14, contracted "influenza." On April 6 Gwendoline was troubled with pains in her lower abdomen, which persisted for three days. She was vaguely unwell until April 9, when her illness declared itself as typical rheumatic fever. Type 15/17 streptococci were recovered from the throats of four of these patients, but were absent from the mother's throat. A pure culture of this organism was, however, obtained from the pus in a pelvic abscess subsequently operated upon.

The mention of rheumatic fever in each of these histories results from the fact that the starting-point of an inquiry into these family epidemics was the study of that disease. A study of otitis media or scarlet fever or cervical adenitis might have called attention equally well to these family epidemics, and a similar state of affairs will be discovered in almost every family from which a case of streptococcal disease is admitted to hospital. Without doubt disregard for these apparently trivial colds and sore throats has defeated the existing legislation for the control of puerperal and scarlet fever, erysipelas, etc.

Discussion

This paper is mainly concerned with the epidemiological information forthcoming from the application of Griffith typing. Suggestions for the control of droplet spread are not made, for they depend to some extent on certain clinical aspects of the problem which will be discussed in a subsequent paper. Certain conclusions may, however, be drawn from the data presented:

1. The unitarian hypothesis outlined by Okell must be adopted.

2. With a few exceptions streptococci pathogenic to man fall into one group (Mrs. Lancefield, Group A), identifiable by a precipitin reaction with a carbohydrate "C" substance extracted with HCl. An organism not giving Group A "C" substance is in all probability, but not conclusively, not pathogenic to man. These human pathogens are divisible into about thirty Griffith types, which breed true in epidemics and are probably distinct and immutable antigens. They are identified by agglutination with absorbed sera, the latter reactions probably depending on a type-specific "M" substance. There is evidence that the clinical picture produced by some of these types varies in certain details from that produced by others.

3. The rash of scarlet fever must be looked upon as entirely fortuitous in its occurrence and as of little value in the assessment of streptococcal parasitism. The occurrence of rash depends on the coincidence of two variable factors: (a) a Dick-positive patient infected by (b) an organism with a high Dick-toxin content. The idea that scarlet fever is sometimes highly contagious and sometimes hardly contagious at all requires revision. The reason for this idea is now obvious.

4. Scarlet fever is, however, a most useful indicator of the existence of streptococcal disease in a community. Erysipelas, most puerperal fever, cellulitis, the bulk of otitis, and certain types of tonsillitis are similarly useful clinical indicators.

5. The aggregate of these indicators probably represents not more than one-half of the morbidity due to *Str. pyogenes* infections.

6. Present knowledge suggests that the great bulk of streptococcal disease (even when apparently sporadic and unassociated with a high incidence of *pyogenes* morbidity) is zymotic in character and associated with a raised carrier rate of streptococci, the enhanced parasitism being due to one type or, at most, a few types of organism in any one community at any given time.

7. Apart from occasional milk-borne spread, a striking phenomenon easily recognized in most instances, the bulk of streptococcal disease is transmitted by droplet. This common type of droplet spread has certain peculiarities. (a) It is a community disease. Spread within home, school, or other institution accounts for the greater part of the contagion. Casual contacts in street, vehicles, and shops, or from visiting relatives in the case of hospital patients, are relatively of minor importance. (b) Its tempo is comparatively slow, probably because transmission is mainly through immune carriers or subclinical cases. Often five and sometimes ten days elapse between the detection of even the most trivial infection in home or hospital ward. (c) The bulk of vector immune carriers are infected for a short period of time only. These transitory carriers must be distinguished from, and are probably of much greater importance than, persons suffering from chronically infected foci.

8. *Streptococcus pyogenes* is almost ubiquitous in temperate zones, and to many it presents an enormous and unassailable problem. These difficulties are simplified by the knowledge that a number of distinct serological types of organism are at work, each acting as a separate unit. Preventive measures might succeed in controlling some, if not all, of these units.

9. In practice, investigation and prophylaxis will start with the detection of one of the many clinical indicators mentioned in conclusion No. 4. If adequate steps are taken at the time of detection of a new Griffith type in any family or community there is a reasonable chance of aborting epidemic spread.

10. Legislation exists for the quarantine and isolation of three only of these clinical indicators. The existing rules are notoriously ineffective, and new regulations with corresponding legislation must be evolved. Isolation and other hospitals admitting acute streptococcal disease are, under present conditions and because of cross-infection (Allison and Brown, 1937; MacNalty, 1936) a potential source for the dissemination of new infections throughout a community.

11. Knowledge of immunological problems relating to *Str. pyogenes* is very incomplete, but it is probable that little or no cross-immunity or group immunity is conferred by infection with any one Griffith type, although homologous immunity probably persists for a considerable time after infection. Immunity to Dick toxin does not imply immunity to streptococcal infection and invasion.

12. A type-specific epidemic tends to run its course through a community and to die out spontaneously. Persistence of an epidemic or the occurrence of relapses should suggest the presence of another type of infection.

13. Control over streptococcal disease will not be obtained until it is realized that *Str. pyogenes* is one of the more common causes of colds, sore throat, and "flu." On the other hand, *Str. pyogenes* tends to spread more rapidly in a community already involved in an epidemic nasopharyngitis due to some other cause—for example, measles. The occurrence of streptococcal complications in measles and similar non-coccal infections is almost without exception part of a separate epidemiological problem. The presence of the complicating organism depends on a zymotic infection coincident with, yet independent of, the primary measles or other nasopharyngeal infection. The bulk of recent evidence contradicts the idea that organisms producing complications were already present in the patient before the attack of measles, etc.

14. Control does not necessarily depend upon complicated bacteriological techniques. Clinical methods are of greater fundamental value and must not be ignored.

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