Streptococcal school outbreaks: a method of investigation and control

By JOAN M. BOISSARD and R. M. FRY*

Public Health Laboratory Service, Cambridge

Effective control of contagious epidemic disease lies in the recognition and neutralization of the source of infection or of the principal means of transmission, or in protection of the population at risk, or in some combination of these three. Where infections of the upper respiratory tract are concerned, that is, where the principal means of transmission is by contaminated air, purification is not generally possible apart from special environments. Control of spread of infection must therefore reside in dealing effectively either with the source or with the population at risk. This is the problem confronting the investigator into outbreaks of streptococcal disease in schools and training establishments—communities, often residential, notoriously prone to such infection and to its serious complication, rheumatic fever.

With the exception of immunization against the Dick toxin streptococcal infections have not proved amenable to artificial forms of protection other than by mass treatment with sulphonamides or penicillin. It has been claimed that penicillin in therapeutic doses for '10 or more days' for all at risk, regardless of their carrier state, is a certain method of terminating an outbreak (Seal, 1955). In considering this procedure, in so far as it halts an epidemic, it seems reasonable to ask why it should succeed when treatment of infected persons only does not necessarily do so. There can only be one answer, that the methods of investigation do not disclose all the important sources of infection, and failure to include them all in the treatment group spells failure in control. But is every patient or carrier of equal importance as a source of infection? If not—and we think not—can the important, in this sense, be distinguished from the unimportant? It is here that the aim of the investigation must be defined with precision and a distinction drawn between the detection of an infected person for his own sake and his detection as a disperser for the sake of the community. The distinction is fundamental for it needs to be understood that the kind of inquiry we have in mind is aimed at recognizing only those carriers who are also dispersers.

In the spread of streptococcal infections from the upper respiratory tract it is axiomatic that the infecting organisms must be expelled in some way from the original host in order to reach and infect a new host. Apart from streptococcal infections of the skin and ear, which are obvious sources of environmental contamination, the two common sites of infection in the host are the tonsil and the nose. Nasal infections are often ignored, or at least not looked for, though it is

* Present address: Department of Pathology, University of Cambridge.

obvious that infected nasal discharges can easily, and will inevitably leave the host and be dispersed in his surroundings via the hands or the handkerchief or by post-nasal discharge contaminating the saliva.

We first became interested in this problem while investigating epidemics of diphtheria associated with evacuees also infected with a variety of streptococcal types (Boissard & Fry, 1941). Nasal infections, ear infections and infected skin lesions of all kinds quickly impressed us with their importance as sources of infection, and we concluded that wherever infected secretions or discharges could be shed into the environment they were potent disseminators of disease. To these we soon added a fourth. Observations on cross-infections in the isolation hospitals arising from children with coughs led to an investigation of the saliva from acute and convalescent patients with either scarlet fever or diphtheria or both. The patients were mostly very young, very sick children, and the saliva specimen was in fact a mouth swab—taken all round the mouth and under the tongue but avoiding the tonsils and back of the throat. We found that in almost half the patients tested in a period of 4 months we were able to isolate group A streptococci from the mouth swab in the first week of illness. Thereafter, although streptococci could be grown from a tonsillar swab—often for very many weeks—none could be recovered from cough plates or mouth swabs in patients whose infection was confined to the tonsil. Hamburger (1944), working with Army recruits and collecting samples of saliva, was able to isolate streptococci from a much larger proportion of his samples, 80 %, for a much longer period, up to 3 weeks. It is probable that our mouth swab discloses only very heavily contaminated salivas, and that, in the absence of a nasal infection, contamination of such a degree is a feature of only the early stages of a tonsillar infection and may be taken as indicative of it. Thus, in assessing the capacity of an infected person to act as a disperser, it seemed to us that a salivary swab, in combination with a nasal swab, might well be substituted for the customary throat swab in the clearance from infectivity of convalescents and contacts. Ear and skin lesions apart, only persons with streptococci in the nose or saliva need consideration as sources of infection.

These are the general considerations which gradually led us to the use of nose and mouth swabs, at first, in 1946, in persistent tonsillar carriers after an acute infection for clearance from infectivity, and later, in 1959, for mass investigations to disclose significant sources of infection. The advantages of mouth over throat swabs are that they are quick and easy to take, no spatulas are required and the number likely to be positive in the mouth alone—as compared with the throat alone—is small, usually less than 5 % of the population swabbed. Moreover, by disclosing only those carriers who are also dispersers, all the positive results are significant.

In 1937 sulphanilamide powder, applied locally, was used to treat a streptococcal infection in multiple sinuses which had been discharging for 3 years. A permanent cure was effected in 3 weeks (Purdie & Fry, 1937). We found local applications of sulphanilamide powder equally effective in streptococcal impetigo. These results encouraged us to try the effect of intranasal sulphanilamide powder, by insufflation on two longstanding nasal carriers of 11 and 5 months duration. This treat-

ment was completely successful and was successfully repeated on twenty-six patients, all in hospital, who had been carrying group A streptococci in the nose for periods varying from 4 to 21 weeks (Boissard & Fry, 1942). These results are detailed with the follow-up histories of these patients in table II and fig. 4 of the paper referred to. The details of treatment can be modified to suit particular circumstances, but the basic course consists of insufflation of both nostrils twice a day, morning and evening for a week. In schools which disperse for the weekend it is better to carry insufflations on for two 5-day weeks. There is no need to exclude children from school while being treated. At the end of the period of insufflation there should be an interval of one clear week before carriers are swabbed for clearance. The treatment can be repeated if necessary, although in the majority one course of insufflation, properly given, is adequate.

RESULTS

Since 1941 we have dealt with several dozen school outbreaks of streptococcal infection. The seven described below and summarized in Table 1 have been chosen to illustrate points we think are of interest and importance.

Table 1. *A summary of the main points of the illustrative outbreaks*

Out-break	School	Year	Term	Approx. no. in school	Type of investi-gation	Swabs taken*	Epidemic type
1	L.C.	1943	Summer	300	Limited	N & T	8/25
2	L.S.	1964	Autumn	300	Limited	N & T	4/28
3	L.S.	1954	Spring	300	Mass†	N & T	3
4	L.S.	1955	Spring	300	Mass†	N & T	18
5	K.C.S.	1962	Spring	150	Mass	N & M	28
6	L.H.S.	1965	Spring	66	Mass	N & M	3
7	F.S.SW.	1965	Spring	340	Mass	N & M	6

* N = nose, T = throat, M = mouth.
† School swabbed in two parts.

The first two, widely separated in time, have in common the importance of a chronic disperser as a source of infection. The history in such outbreaks is often so suggestive that it is possible to undertake a limited investigation with a good prospect of success.

Outbreak 1

This occurred in a boarding school of about 300 boys, and covered one school year. It began with cases of scarlet fever and sore throat in the autumn term of 1942. In the spring term of 1943 there were no cases of scarlet fever and very few sore throats, but in the summer term, up to the time of our first visit to the school, there had been three cases of scarlet fever and seven of acute tonsillitis. Enquiry for a boy with a chronic cough or catarrh at once produced the name of one boy, whose personal history had a remarkable bearing on the incidence of infection in the school. Eighteen months earlier he had had scarlet fever, which left him with a persistent rhinorrhoea, still present when he first entered the school in September

1942. During the spring term of 1943 he was kept at home for treatment, and he rejoined the school for the summer term with his rhinorrhoea as bad as ever. Cultures from this boy, from the most recent case of scarlet fever and from five of the seven cases of acute sore throat all showed infection with group A streptococci of type 8/25. The disperser's infection was confined to his nose. He was treated with sulphanilamide insufflation and cured. Enquiry a year later confirmed that no further cases had occurred, and the boy had remained free from symptoms.

An account of this outbreak has been published (Boissard & Fry, 1944), but the details are summarized here because the original publication is somewhat inaccessible.

Outbreak 2

This outbreak, summarized in Table 2, occurred in a boarding school of over 300 boys in the autumn term of 1964. The school re-assembled on 17 September, and all the new boys had routine nose and throat swabs examined. Five were found positive for group A streptococci in the throat alone. No further action was taken.

Table 2. *The results of bacteriological examination in outbreak 2*

Date	Name	Disease*	Culture from N	Culture from T	Type	Form	House	Dormitory	Remarks
17. ix.	A.M.P.	.	−	+	4/28	V_2B	W	1	New boy
6. x.	J.K.G.B.	Tons	−	+ + +	4/28	V_2A	S	.	.
15. x.	D.J.R.	Tons.	−	+ + +	4/28	VI	W	1	.
16. x.	Q.B.P.	Sinus.	−	+ + +	4/28	V_1B	W	2	.
20. x.	N.C.	Tons.	+	+ + +	4/28	IV A	W	1	.
20. x.	E.G.	Tons.	−	+ + +	4/28	V_2C	W	1	.
21. x.	Mrs S.	Tons.	−	+ + + +	4/28	Cleaner	W	.	Worked in dormitories 1 and 2
23. x.	A.M.P.	.	+	−	4/28	V_2B	W	1	The only positive found on swabbing dormitories 1 and 2

* Tons, = tonsillitis; Sinus. = sinusitis.

The first case of acute tonsillitis occurred on 6 October, the second on 15 October and the third on 16 October. Cases 4 and 5 occurred on 20 October by which date it was known that the earlier cases were all type 4/28. Moreover, cases 2 to 5 were all in one house (W), three in dormitory 1 and one in dormitory 2 on the same top floor across a passage. This drew attention to a new boy, A.M.P., swabbed on admission to the school on 17 September and at that time found to have type 4/28 in his throat. He was in dormitory 1 of W house where three of the four infections had occurred, and he could have had class-room contact with case 1.

An initial limited investigation was advised, confined to the thirty-two in dormitories 1 and 2 in W house, and boys with colds or catarrh anywhere in the school. A total of forty-one boys was swabbed on 23 October and a single positive was found, the new boy A.M.P., now positive in the nose alone with the same type 4/28. On the same day swabs were received from Mrs S., a cleaner whose

duties were confined to the top floor of W house, who had developed an acute tonsillitis on 21 October, and was also found to be infected with type 4/28. A.M.P. gave a history of severe acute tonsillitis at his preparatory school the previous spring for which he was kept in bed for a week, a story which added to the possibility that he was a chronic nasal carrier from that time and that the admission swabs
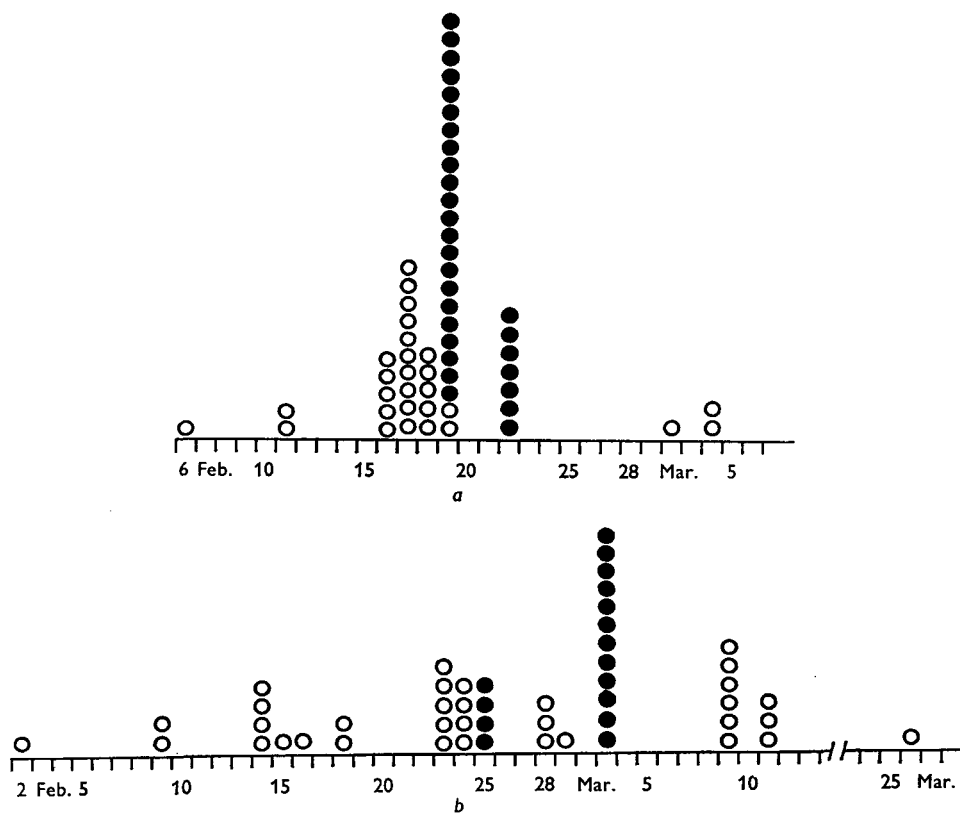

Fig. 1. (a) Outbreak 3, (b) outbreak 4. ○ = cases of acute streptococcal infection; ● = dispersers found at mass swabbing.

were perhaps mislabelled. He and the other boy with a positive nasal swab were treated with sulphanilamide insufflation and cleared of nasal infection. No more infections with this type occurred in the school for the rest of the term.

Outbreaks 1 and 2 are strikingly similar; both appear to have been started by a single individual with a chronic infection following an acute attack. In both recognition and treatment of the disperser brought the outbreak to an abrupt end.

Outbreaks 3 and 4

These two outbreaks occurred in the same boarding school in the spring terms of 1954 and 1955. In each year the first case of tonsillitis occurred in the first week of February, followed by a build-up of cases in the next 2–3 weeks as shown by the open circles in Fig. 1 a, b.

By the time investigations could be undertaken it was clear that mass swabbing

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alone offered hope of control. For administrative reasons the swabbing was done in two parts, the junior school first in each year followed by the senior school 3 days later in 1954, and 6 days later in 1955. Nose and throat swabs were taken each time, and the numbers of nasal carriers are shown by the black circles in Fig. 1. All the nasal carriers were treated with sulphanilamide insufflation for a week without exclusion from school.

In 1954 where the interval between the two swabbings was short, and where the first investigation gave the larger number of dispersers, the outcome was reasonably satisfactory. Only three more cases of infection occurred, all within 10 days of the second swabbing, and there were no more infections in that or the next term.

In 1955, when the interval between the swabbings was longer, and when the number of dispersers eliminated by the first swabbing was small, the effect of the intervention was less decisive. Four infections occurred between the two swabbings and nine more within 10 days of the second swabbing. These are probably linked, more or less directly, with the dispersers found at the second swabbing, but there is another possibility. On each occasion the number of carriers found positive in the throat alone exceeded the nasal positives found at the same time. Some of these would almost certainly have shown positive mouth swabs, had these been taken, and such temporary dispersers could have given rise to some of the post-swabbing infections. That no chronic dispersers were responsible is strongly suggested by the fact that, apart from a single case near the end of term, there were no further cases after 10 days, and there were no further cases in the next term.

If mass swabbing is considered necessary, the whole community should be swabbed at the same time, and this outbreak illustrates the drawback of dividing the swabbing. Any division is likely to be an artificial one from the epidemiological point of view, as here, where division into lower and upper school bears no relation to the division into houses and dormitories.

Outbreaks 5, 6 and 7

These three outbreaks, one in 1962 and two in 1965, have several points in common. They all occurred in the spring term, and were all investigated by mass swabbing of the whole school on one day, using nose and mouth swabs instead of nose and throat swabs. The schools however differered greatly in size, and the epidemic type of streptococcus was different in each. All dispersers, whether nose or mouth positives, were treated with sulphanilamide insufflation as before, and penicillin lozenges to suck for two days were advised for those whose mouth swabs were positive.

Outbreak 5 was in a preparatory school of 149 boys, seventy-five of whom were boarders. Fig. 2a shows that there were nine infections up to the day of the mass swabbing, the last two of which appeared on that day. Five dispersers were found and treated without exclusion from school. All the acute cases and all but one of the dispersers were boarders. No further cases due to type 28 occurred that term or in the next.

Outbreak 6 was in a boarding school of sixty-six boys. The school re-assembled

after Christmas on 12 January; the first case of tonsillitis occurred on 21 January and the second on 25 January. On the same day a swab was taken from a boy with a 10-day history of a discharging ear, shown in Fig. 2*b* in black, for 15 January, the day the discharge started. By 27 January it was known that all the strains belonged to type 3, and as the likely source of infection had been operating since 15 January, mass swabbing was advised. For administrative reasons this was
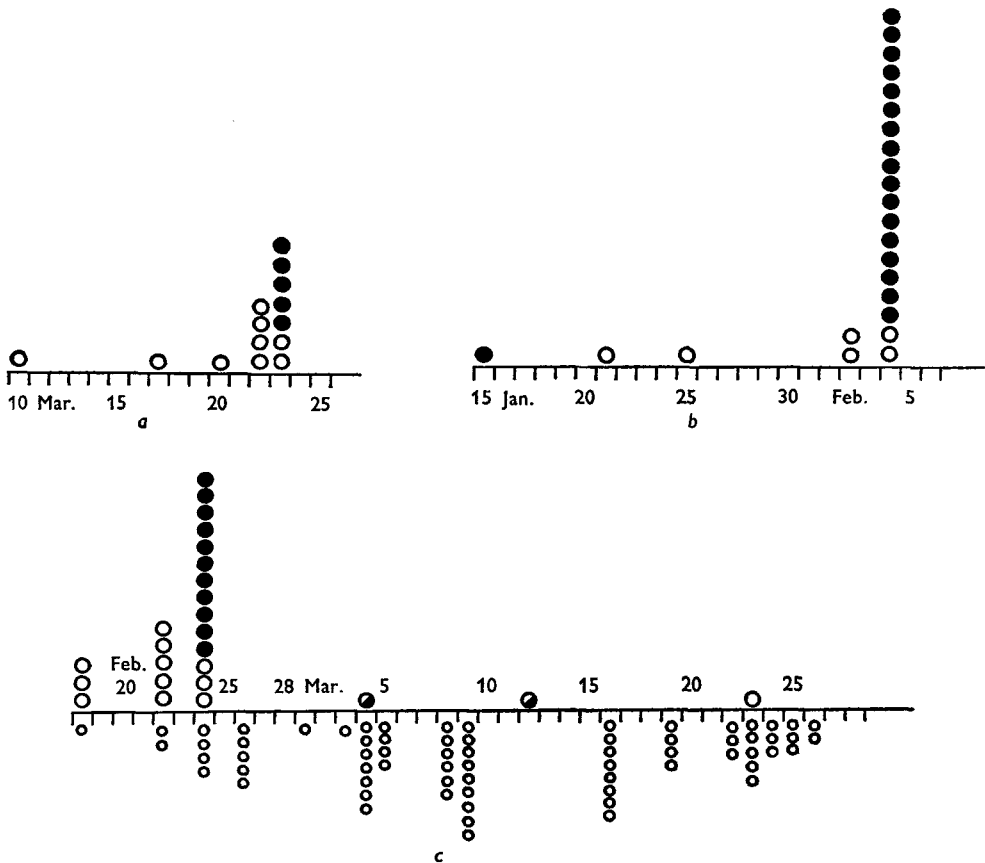

Fig. 2. (*a*) Outbreak 5; (*b*) outbreak 6; (*c*) outbreak 7. Acute infections and dispersers shown as in Fig. 1. ○ (below the line) = cases of non-streptococcal respiratory illness. ◐ see text, page 228.

postponed until 4 February, by which time four more cases had occurred, two on 2 February, and two on 4 February. Seventeen dispersers were found on 4 February. They were treated, and no new cases occurred. About the middle of March, however, there were fourteen cases of upper respiratory disease from whom no bacterial pathogens were isolated, except for one boy with a throat culture positive for type 3 streptococci. It appears highly probable that his symptoms were due to whatever was causing the illness in his fellows at the time and that he was a symptomless throat carrier who would not have been recognized at the mass swabbing, when throat swabs were not taken.

15-2

Outbreak 7 was in a boarding school of 340 children. This, like the previous outbreak, was complicated by a non-streptococcal upper respiratory illness, this time concurrent with the streptococcal infections, and the number of such cases was so large that we have shown them in Fig. 2c by the small open circles below the zero line. In the figure it is seen that there were eleven cases of acute streptococcal tonsillitis between 18 and 24 February, the day of the mass swabbing. On that day nose and mouth swabs were taken from 324 children and eleven dispersers were found. These were treated. Meanwhile, many cases of sore throat with negative swabs were occurring, but two children, one on 4 March and one on 12 March showed a scanty growth of type 6 streptococci in cultures from their throats. These two are shown in Fig. 2c above the line, as half-black circles, to indicate that they were probably cases of non-streptococcal sore throat who happened to be undetected throat carriers. The single case shown on 23 March, however, was a heavy nasal positive and must be considered a new infection. The possible source of this new infection is of some interest. A child who developed an acute infection on 22 February, was treated with penicillin and sent home for a period of convalescence. She returned to school without being re-swabbed, and on 15 March was found to be a heavy nasal carrier. She was then given sulphanilamide insufflation *for the first time*, combined with a second course of penicillin for a suspected infection of the antrum, and was cured. This child may represent the early stage of a chronic disperser of the type found in outbreaks 1 and 2.

No further cases occurred during that term or the next.

DISCUSSION

We have in these seven examples illustrations of two fundamentally different types of outbreak. In one, which we call the limited epidemic, virtually all persons infected from the primary source develop symptoms of acute infection and are as a result removed or treated. The symptomless disperser does not seem to arise. An example of this is seen in outbreak 2, where complete investigation of the disperser's surroundings showed no symptomless infections. The same is probably true of outbreak 1. In both these the elimination of the initiator brought the infections to an end.

This is in contrast to the second type, the unlimited outbreak, in which, whether the initiator is recognizable or not, factors which we believe to be associated with the organisms rapidly give rise to many symptomless dispersers so that the epidemic becomes self-propagating. Removal of the *initiator* alone is unlikely to end the outbreak. This type is shown in all its details in outbreak 6. In such an outbreak we think that only mass swabbing offers a good prospect of control, but the control still depends on the neutralization of the human dispersers. The environmental contamination appears to be self-limiting, and although a few cases may arise after the neutralization of the dispersers (see Fig. 1a, b) we would not expect to see any beyond about 10 days. It is infections arising beyond this period that suggest that an undetected disperser is at large. This has been discussed under outbreak 7, and is shown in Fig. 2c where, because of the large concurrent

outbreak of non-streptococcal upper respiratory infection, the bacteriological control could hardly have been more extensive.

We have here been considering conditions in which early information about the history of the outbreak and the epidemic type of the infecting organism is available. But it must be made clear that the method of control does not depend on this information, though its availability may on occasions make it possible to reduce the scale of the investigation with safety. Control depends on mass swabbing to recognize dispersers, and where epidemic conditions exist almost all the dispersers found will be of the epidemic type. Though a few persons dispersing strains of non-epidemic type may be included in the treatment group the number is likely to be very small, and treatment can do them no harm.

It is of importance to determine the type of the epidemic strain at some stage in order to establish that control has been effected. Twice we have seen one outbreak succeed another almost without an interval, and only the knowledge of the streptococcal types enabled us to show with certainty that the second epidemic was not a recrudescence due to failure to control the first.

In treating nasal infections, with or without an associated positive culture from the mouth, we have used a well-tried method because we have found that it succeeds. We do not believe that this is the only method, and would welcome an equally effective alternative to sulphanilamide powder, if only for use when we meet a sulphonamide-resistant strain of streptococcus. But we have not yet failed to clear up a nasal infection, and this may be because of the very high concentration achieved by local application of a sulphonamide with a relatively high solubility (1/170 in water for sulphanilamide). It is reasonable to suppose that organisms that are resistant to the concentrations reached in the body fluids during oral treatment (1/20,000–1/50,000 approx. in the blood) may yet respond to the much higher concentrations achieved by local application of sulphanilamide.

The numbers of dispersers positive in the mouth alone is relatively small, and though they are undoubtedly of importance as sources of infection while the saliva is heavily infected this degree of infection diminishes very rapidly with the passage of time alone. In most of our work we have included them in the nasal insufflation group without additional therapy because it was the simplest thing to do administratively, because treatment could do them no harm and might do some good by preventing a nasal or a middle ear infection from arising and because at the end of their week of treatment they would in any case be nearing the end of the period when they would be recognized, by our methods, as dispersers. We have in certain circumstances, such as children sitting a public examination, advocated penicillin lozenges for 2 days, but this is entirely in the interests of the contacts. We doubt its value as a therapeutic measure in effecting the cure of a tonsillar infection. An efficient alternative capable of making the saliva non-infective would be welcomed.

We have, throughout this account, advocated chemotherapeutic measures of control because we know of no other, except strict exclusion from school of all dispersers. Since nasal carriers may remain positive for weeks, months or even years this is clearly an unsuitable method. In general terms, however, we state

emphatically that, in our opinion, control can be achieved by finding all dispersers as we have defined them and by dealing with them by any means available that will prevent them from acting as dispersers.

SUMMARY

A method of controlling outbreaks of streptococcal infection of the upper respiratory tract in communities such as boarding schools is described.

It is suggested that, in a search for carriers, nose and mouth swabs should be taken in place of the customary nose and throat swabs. These swabs will detect those who are dispersing streptococci into the environment, as distinct from the carriers who are not dispersers. It is essential that infections of the skin and ears should also be looked for.

It is recommended that all dispersers, whether nose or mouth positive, should receive sulphanilamide insufflation in both nostrils twice daily for a week, or longer if necessary, without exclusion from school. In addition, penicillin lozenges for 2 days may be recommended for the mouth positives only.

The history is considered to be an important factor in deciding the extent of the initial investigation, and examples are given to illustrate this. When mass swabbing is considered necessary, it is highly desirable that the whole community should be swabbed at the same time, as early as possible in the outbreak. It is also desirable that bacteriological follow-up should be available to check that control has been effected.

REFERENCES

BOISSARD, J. M. & FRY, R. M. (1941). The association of haemolytic streptococcal infection of the nose with diphtheria. *Publ. Hlth, Lond.*, **54**, 105.

BOISSARD, J. M. & FRY, R. M. (1942). Chronic nasal diphtheria carriers; cure with sulphanilamide. *Lancet*, i, 610.

BOISSARD, J. M. & FRY, R. M. (1944). The detection of a streptococcal carrier. *Mon. Bull. Minist. Hlth*, **3**, 160.

HAMBURGER, M. (1944). Studies on the transmission of hemolytic streptococcus infections. II. Beta hemolytic streptococci in the saliva of persons with positive throat cultures. *J. infect. Dis.* **75**, 71.

PURDIE, A. W. & FRY, R. M. (1937). Chronic haemolytic streptococcal infection treated with *p*-aminobenzenesulphonamide. *Lancet*, ii, 18.

SEAL, J. R. (1955). Oral penicillin prophylaxis of streptococcal infections. *Am. J. publ. Hlth*, **45**, 662.