STREPTOCOCCAL INFECTIONS AMONG CHILDREN IN A RESIDENTIAL HOME

II. POTENTIAL SOURCES OF INFECTION FOR INDIVIDUALS

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In part I of this series (Holmes & Williams 1958*a*) we enumerated the streptococcal infections observed in our 30 months' survey in the Barkingside Village of Dr Barnardo's Homes and indicated broadly which groups of children suffered most frequently. The present paper attempts to answer the question: '*From what sources could individual children have been infected?*' *To give a complete answer* to this question one would need a detailed bacteriological survey of the whole population carried out at intervals of a very few days, and this we have not got. Nevertheless, the information that we have seems to justify an attempt at an incomplete answer to the question.

We can name all the children, and many of the staff, who were living in any particular cottage at any time, and for the majority of children between the ages of 3 and 11 years we also know the class in school or nursery school attended, and the names of the other children in that class. Children of 11 years and over attended schools outside the Village so that we know only about their cottage-mates and their Barkingside class-mates, and not about their numerous class-mates from outside the Village.

So far as infections are concerned, we have a bacteriological record of a very large proportion of the children and staff with clinically manifest streptococcal infections, in the acute stage and in convalescence. And for 159 of the 459 cases of known streptococcal infection of the respiratory tract or ear, which were secondary cases in cottage outbreaks of infection, we have a record of the carrier state of all the healthy children in the cottage. We know of healthy carriers among school class-mates only if, by chance, they were discovered in an investigation of a cottage outbreak.

Clearly there are large defects in our knowledge: our investigation of cottage outbreaks was often carried out after the few cases had occurred so that the records of healthy-carrier contacts may be too few; and we have no details of the numerous

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casual contacts in the Village and in the school who must account for a great part of the spread of infection. The deficiencies in our knowledge are particularly great for the first cases in cottage outbreaks.

Moreover, the fact that a child was exposed to a particular carrier is not, of course, evidence that he contracted his infection from that carrier. In some cases there is collateral evidence that makes the presumption reasonably strong, but more often the contacts indicated are those who seem to us the most likely source from which the infection was spread, and there is necessarily a considerable element of judgement involved when we specify one among many as being the most likely source. We have generally tabulated the 'heaviest' carriers to whom a particular child was exposed—regarding nasal carriers, heavy or light, as 'heavier' than simple throat carriers.

METHODS

Classification of known infected contacts

For each case of acute streptococcal sore throat, or presumed streptococcal otitis or cervical adenitis (the 'index' case), we noted the infected persons—cases of sore throat, etc., or healthy carriers—to whom the child was known, or reasonably assumed, to have been exposed in his cottage or in his school class, within the 7 days before the onset of his illness.

Number and 'danger' of infected contacts. In addition to recording the number of the contacts to whom the index case had been exposed, we were able in some cases to 'specify' one of these carriers as the most likely source of the infection. For example, four cases of sore throat due to *Type 2* developed in Ethel Bolton Cottage between 9 and 12 November. All the healthy children and staff were swabbed on 12 November and one child, J.R., was found to be a heavy nasal carrier, and had skin lesions also infected with *Type 2* streptococci; his brother and one other child were light nasal carriers. J.R. was transferred on 12 November to Gordon Cottage and 4 days later (16 November) himself developed a sore throat due to *Type 2*. Two more cases occurred in Gordon Cottage on 17 and 18 November, and when the healthy children there were swabbed on 19 November no carriers were found. It seems most probable that J.R. was responsible for the infection in all six cases. Such clear examples were not very numerous and we often had to 'specify' carriers on less evidence. But we were, in general, reluctant to include a carrier among the 'specified' sources merely because he was the only contact available for a single infection. We classified the 'specified' sources as α, β, or γ to denote respectively decreasing degrees of confidence in our specification. Although we have endeavoured to avoid bias in this assessment from preconceptions as to who would be most likely to spread streptococci, it is certain that we have been more apt to blame heavy rather than light carriers, and to give more weight to nasal than to throat carriers.

The time of discovery of the carriers. Although we have largely relied on bacteriological investigations carried out immediately prior to the day of sickening of the index case, we sometimes had only investigations made afterwards. In such cases, the carriers found could have been infected before the index case, and might

therefore have been the source of his infection; they might have been infected at the same time as the index from a common source; or they might have been infected from the index case. Sometimes there was general epidemiological evidence that the carrier had been infected earlier and had infected the index case, and in twelve instances this evidence seemed strong enough for us to specify the carrier as the source (see below, Table 3). Carriers discovered more than 7 days after the index case sickened were never included, and in most cases the carriers were discovered within 2 or 3 days after the onset of illness in the index case.

The stage of carriage. We distinguished 'incubating', 'convalescent', and 'healthy' carriers. Persons who became ill with a streptococcal infection were considered potentially 'incubating' carriers for up to 7 days (or more if demonstrated bacteriologically) before sickening, but in most cases in which such a carrier was suspected as the source the relevant period was not more than 2 or 3 days. When manifest disease developed, the patient was removed from the cottage to the hospital so that contacts were only exposed to sick persons in the incubation stage and just at the time the disease became manifest.

Convalescents were not assumed to be carriers for more than 7 days after their last positive swab. A convalescent from a sore throat who subsequently relapsed with another attack of sore throat or suffered otitis, etc., as a complication, was considered to be 'convalescent' in the interval between illnesses.

'Healthy' carriers were children who did not, at the material time, become ill with their streptococcal infection.

The abundance of streptococci carried. Carriers were put into one of three categories: heavy nasal carriers with numerous streptococci in a nasal culture; light nasal carriers, with smaller numbers of streptococci in a nasal culture; and throat carriers who had no streptococci in their nose. Nine children with heavily infected skin lesions were classed with the heavy nasal carriers, although some of them were only light nasal or throat carriers.

When a contact was known to be a light nasal or throat carrier before the onset of illness in the index case, but was found to be a heavy carrier soon afterwards, we have entered him in the tables as though the nasal cultures had already become 'heavy' at the time of exposure (see Table 4, below).

Cases with no known infected contacts

For many cases of infection we knew of no contacts infected with the same type of streptococcus either in the cottage, or, when the child went to one of the schools for which we had a record, at school. These instances were grouped separately when there had been cases of this streptococcal type in other cottages in the Village during the previous 4 weeks, or when no cases had occurred in the Village for at least 4 weeks.

Relapses, re-infections and 'suspect' infections

An illness was held to be a 'relapse' if the patient was known to have carried the infecting type of streptococcus continuously since a previous attack of streptococcal illness, and in this analysis the term 'relapse' included illnesses otherwise

regarded as complications, e.g. an otitis following a sore throat. (Such a relapse was not included at all if it occurred before discharge from hospital.) In the case of prolonged carriage, a single negative swab was disregarded if followed by one or more positive swabs.

Illnesses were regarded as 're-infections' only when a child failed to yield streptococci from two consecutive swabs taken at an interval of a week or more between the two illnesses; the two illnesses are entered independently in the tables.

When children were known to carry streptococci in their throats for some time before they became ill there was necessarily some doubt whether the illness was in fact due to streptococci; we have regarded as 'suspect' any instances of illness other than a relapse in children known to have carried streptococci for more than 7 days before sickening.

RESULTS

Contacts of children with streptococcal illnesses

Of the 459 illnesses studied, 48 (10·5 %) appeared to be relapses and 46 (10·0 %) were 'suspect' as just defined. We are, therefore, left with 365 cases suitable for analysis for source of infection (Table 1).

Table 1. *Sources of streptococcal infections in children and staff*

Possible source of infection	Streptococcal sore throat			'Streptococcal otitis'	Other streptococcal infections	Grand total
	Children	Staff	Total			
Number of cases	280	56	336	94	29	459
Percentage relapses	8·2	1·8	7·1	21·2	13·8	10·5
Percentage carrying infecting Type, 7 or more days before onset of illness	6·8	1·8	6·0	22·4	17·2	10·0
Number of cases analysed	238	54	292	53	20	365
Percentage exposed to one 'specified' carrier	11·4	5·6	10·3	3·8	10·0	9·3
Percentage exposed to several carriers, one 'specified'	25·2	7·4	21·9	20·7	30·0	22·2
Percentage exposed to one carrier, not 'specified'	4·2	3·7	4·1	3·8	5·0	4·1
Percentage exposed to several carriers, none 'specified'	27·7	61·0	33·9	28·2	25·0	32·7
Percentage considered infected in hospital	2·1	0	1·7	3·8	0	1·9
Percentage considered infected outside the Village	2·1	3·7	2·4	3·8	10·0	3·0
Percentage with no contacts known in cottage (or school), cases or carriers due to same streptococcal type in other cottages	19·3	9·3	17·5	18·9	15·0	17·5
Percentage with no contacts and no cases due to same type in the Village	8·0	9·3	8·2	17·0	5·0	9·3

Of the 238 children with sore throats, 11·4 % were exposed to a single carrier who was thought to be the source of their infection. A further 25·2 % were exposed to several carriers, one of whom was thought to be responsible. In a great many other cases (31·9 %) there were one or more infected contacts although none could be specified as the source. However, the difficulty often lay in selecting a particular source rather than in finding one, particularly for the last persons to be attacked in a cottage outbreak.

A number of children became ill within 7 days of entering the Village. When there

was no evidence of previous infection with the particular streptococcal type in the cottage, these (2·1 %) were presumed to have been infected before admission. The hospital seemed to be the source for the same proportion (2·1 %), because these children developed their illness within 7 days of discharge, and children or nurses infected with the same type of streptococcus were being nursed in the hospital at the time.

We are left, therefore, with 27·3 % (65) of the 238 patients with sore throats for whom we know of no immediate contacts who might have been the source of the infection; in two-thirds of these the particular streptococcal type was known to be causing illness elsewhere in the Village at the time.

Twenty-five (39 %) of the sixty-five infected children with no known contacts were in cottages not investigated bacteriologically at the time, and the absence of contacts may merely reflect the failure to look for them. However, in fifty-two cases the children in the cottages were examined within 7 days before or after the occurrence of the index case, and in 21 % of these no contact was found who might have infected the index case; this proportion is very close to the 27·3 % observed for the whole series. Similarly, the proportion of children for whom no contact was found was no higher among those whose school-mates were unknown to us (22·2 %) than in the whole series.

Otitis was more often a 'relapse' by our definition than was sore throat, for the simple reason that it commonly complicated a streptococcal sore throat; it also more commonly occurred in children who had been carrying the streptococcus for some time (the 'suspects'). Altogether 43·6 % of the cases of otitis fell into one or other of these two categories. We cannot, however, exclude a streptococcal cause for otitis simply on the fact of long carriage before the onset of the illness; we have observed children with streptococcal otorrhoea—in which the streptococcus seems very likely to have some causal role—who were known to have carried the streptococcus in the throat for at least a month previously.

There was a striking excess in the proportion of 'suspect' infections in the *nursery-age* children (Table 2) compared with the *school-age*—sufficient to raise a query as to the part the streptococci were really playing in the younger children's illnesses.

The *reception* and *permanent* groups did not differ appreciably, except in the proportion of relapses of sore throat; which for some unknown reason were more frequent in the *permanent* group.

'Specified' sources of infections

There were 205 cases of sore throat (indicated in Table 1, rows 5–8) for which we could indicate one or more known infected contacts; for 94 (39 % of the sore throats analysed) we felt able to specify a single individual, seventeen of whom were almost certainly (α), forty-six probably (β), and thirty-one possibly (γ) the source of the infection (Table 3). For only 23 (11 %) of the 205 infections were the 'sources' quoted not discovered until after the onset of illness.

Of the group classified as having α sources, four were infected from incubating, eleven from convalescent, and two from healthy carriers; all but two with abundant

Table 2. *Percentage of illnesses attributed to various sources at different ages and in reception or permanent cottages*

	Exposed to one or more known infected persons (or infected in hospital or outside the Village)	No infected contacts known	Relapses	Carrying infecting Type, 7 or more days before onset of illness ('suspect')	Total illnesses
Sore throats					
School-age, reception and permanent	63·8	27·0	6·8	2·4	207
Nursery and baby, reception and permanent	56·2	12·3	12·3	19·2	73
Reception, school-age	66·4	27·1	3·7	2·8	107
Permanent, school-age	61·0	27·0	10·0	2·0	100
Otitis					
School-age cottages	32·0	24·0	26·0	18·0	50
Nursery and baby cottages	41·5	17·1	14·6	26·8	41

Table 3. *Time of discovery of carriers suspected as sources of infection*

	Exposed to one or more carriers, one individual specified as the probable source				No one individual specified		
Time at which carrier discovered	α*	β	γ	Total	Exposed to several carriers but no evidence to incriminate one	Exposed to one carrier but no good incriminating evidence	Grand total
Before or on same day as sickening of index case	10	32	24	66	66	7	137
After sickening of index case	2	7	3	12	6	5	23
Light nasal or throat-only carrier(s) before, heavy carrier(s) after, sickening of index case	5 (3)	7 (4)	4 (0)	16 (7)	29	—	45 (7)
Grand total	17 (3)	46 (4)	31 (0)	94 (7)	99	12	205 (7)

* α, β, γ: see text.

Numbers in brackets indicate cases in which a single child changed his or her carrier status; otherwise the specified individual is the heavier carrier, discovered after the occurrence of the index case.

streptococci in their nasal cultures. The distribution was not very different for the forty-six infections with β sources (Table 4).

Among the ninety-four instances where a single individual source of infection could be indicated, incubating, convalescent, and healthy carriers were similar in number—twenty-nine, thirty-six and twenty-nine, respectively. Where no one individual could be specified as the source of an infection we have tabulated the state of the 'heaviest' carrier found; thus a case is entered under 'heavy nasal' if one of his contacts was such even if there were also several light nasal or throat carriers among them.

Heavy nasal carriers were among the contacts of no fewer than 128 (62 %) of the

Table 4. *Nature of the sources for 205 cases of sore throat with recognized infected contacts*

Nature of carrier specified as source		Exposed to one or more carriers, one individual specified as the probable or possible source					No one individual specified: Exposed to several carriers, but no evidence to incriminate one	No one individual specified: Exposed to one carrier, but no good incriminating evidence	Grand total
		α*	β	γ	Total				
Incubating	Heavy nasal	4†	11	1	16	29	5	0	37
	Light nasal	—	4	3	7		2	—	
	Throat only	—	5	1	6		—	1	
Convalescent	Heavy nasal	11	11	4	26	36	7	2	54
	Light nasal	—	3	4	7		6	—	
	Throat only	—	3	—	3		1	2	
Healthy	Heavy nasal	2	8	10	20	29	31‡	—	81
	Light nasal	—	1	3	4		12	2	
	Throat only	—	—	5	5		2	5	
Combination of incubating, convalescent, healthy	Heavy nasal	—	—	—	—		21	—	33
	Light nasal	—	—	—	—		5	—	
	Throat only	—	—	—	—		7	—	
Total	Heavy nasal	17	30	15	62		64	2	128
	Light nasal	—	8	10	18		25	2	45
	Throat only	—	8	6	14		10	8	32
Grand total		17	46	31	94		99	12	205

* For explanation of α, β, γ, see text.

† One child, responsible for two cases, was a light nasal carrier and had heavily infected skin lesions.

‡ One child, responsible for one case, was a light nasal carrier and had heavily infected skin lesions; one child responsible for one case, was a throat-only carrier and had heavily infected skin lesions.

Note. When a child was exposed to more than one carrier and none was 'specified', the source classified was the heaviest carrier found.

total 205 cases of sore throat with recognized contacts, and for no more than 32 (16%) could we find only throat carriers. We felt, however, that in eight of these cases the throat carriers—all either incubating or convalescent—were probably responsible for spreading infection (β in Table 4).

Only five of the specified throat carriers had their saliva examined for streptococci; three yielded no streptococci, one moderate, and one large numbers. The most that can be concluded from this is that some throat carriers appear to be dangerous without having infected saliva.

There were twenty-three convalescent carriers who were specified as the source of some other infections and who had been examined on their admission to hospital with a streptococcal infection; for these we can date reasonably accurately the acquisition of their infection. The mean interval between this date and the date of onset of the last secondary case attributed to them was 22 days; the longest interval was 40 days, and ten of the twenty-three intervals lay between 20 and 30 days inclusive. The greatest interval between the sickening of an incubating infector and the person infected from him was 6 days, and in most cases the interval was 2–4 days. It was not usually possible to date the acquisition of infection by the healthy carriers, but one such carrier was specified (though only at the γ level) as a source 278 days after she was first found infected.

Comparison of school and cottage as source

Sixty-nine of the 205 sore throats were in children who attended the school in the Village, so that we knew all their class-mates, although we had not as a rule any extensive bacteriological records for more than a few of them. In twenty-eight of the sixty-nine cases, a cottage-mate was specified as the source (α, β, or γ) or was the only known infected contact; in sixteen the same was true of a class-mate. In the remaining twenty-five cases carriers were present in both school and cottage; in six the school and cottage contact was the same child; in ten equally heavy carriers were present in both, and in five cases the cottage, and in four cases the school had the heavier carriers. Of the sixty-nine sore throats, therefore, at least 41 % could have been contracted in the cottage, and at least 23 % in the school; the remainder may have been caught in either.

In summary (Table 5) it seems likely that about 50 % of all the sore throats observed in children in the Village were due to infection contracted in the cottage. The proportion contracted in school is very difficult to estimate; it is certainly more than the 12 % of Table 5 because some of the infections in schoolchildren, whose school contacts were not known to us, must have been contracted in school and there must have been unrecognized healthy carriers among the known school-mates. The suspect infections, particularly common in the nursery children, were probably largely contracted in the cottage.

Table 5. *Summary of sources of infection* (*excluding relapses*)

	No. of sore throats due to infection probably contracted					
	In the cottage	In school or nursery school	Outside the Village; in hospital; or presumed from other cottages in the Village	No. with no source known	No. of 'suspect' infections	Total
Babies	6	—	0	0	0	6
Nursery-age children						
School known	10	3	5	3	8	29
School not known	—	—	—	—	—	—
Holidays	5	—	—	—	2	7
No school*	15	—	2	1	4	22
School-age children						
School known	41	28	19	7	2	97
School not known	32	—	12	4	1	49
Holidays	14	—	5	1	1	21
No school*	9	—	13	3	1	26
Total	132	31	56	19	19	257
Percentage of total	**51·3**	**12·1**	**21·8**	**7·4**	**7·4**	**100·0**

* These children were mostly newcomers to the Village who had not yet started to attend school.

Note. The cases with equally heavy contacts in school and cottage have been distributed equally between these two sources.

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A similar analysis was carried out on the source of cases of otitis and other respiratory infections and, though the numbers are small, the trends resembled those for the sore throats.

Re-infections

In nine cases we considered that illnesses might have been due to re-infection with a type of streptococcus that had produced illness in the same child earlier. Only three of these, however, conformed to our definition in having had two nose and throat cultures free of the streptococci, and of these one had had penicillin treatment for the first attack. The other two cases seemed unequivocal and are worth recording in detail. G.A. aged 4 years had a sore throat due to *Type 2* commencing on 30 June 1951; he was treated with 'Cremor-mezathine', but continued to carry the streptococci in his throat until 17 January 1952. Subsequent cultures taken on 4 February, 25 February and 6 March showed no haemolytic streptococci. He became ill with a sore throat on 4 April 1952 and *Type 2* was isolated from his throat on three separate occasions. The other child, C.B. aged 7, was admitted to hospital with pyrexia on 22 July 1952 and yielded a heavy growth of *Type 22* streptococci from both nose and throat (and is known not to have been a carrier 1 month previously). She was treated with sulphamezathine for 6 days and nose and throat swabs taken on 30 July, 5 August and 19 August yielded no haemolytic streptococci. On 24 August, 1 month after the first illness, she became ill again with an acute sore throat and had *Type 22* streptococci in nose and throat.

DISCUSSION

Since Hamburger and his colleagues (e.g. Hamburger, Green & Hamburger, 1945*a*, *b*) established the importance of the nasal carrier of streptococci as the carrier most likely to spread infection to others, there has been some discussion on the proportion of cases infected by nasal carriers. The analysis reported in this paper is a contribution to this discussion. In all the instances in which we felt fully confident in attributing a child's infection to a particular carrier, that carrier had numerous streptococci in his nose. But such confidence on the source of infection was only possible for 17 of 238 illnesses, and then because the particular carriers were highly infective and usually responsible for several secondary cases. We shall show again (Holmes & Williams, 1958*b*), that the heavy nasal carriers were undoubtedly more infective than other carriers.

In fourteen instances we considered it probable (eight) or possible (six) that an infection was contracted from a person who harboured streptococci in the throat only. It is, of course, possible that we failed to discover some other contact who was a nasal carrier, and responsible for the infection; or that the carrier whom we specified had been a nasal carrier for a short period, during which the infection was transmitted. Part of the reluctance to attribute spread to the throat carriers arises from the difficulty of demonstrating that they emit streptococci from the throat in any ordinary activities (e.g. Duguid, 1946; Rubbo & Benjamin, 1953); salivary carriers are better able to disperse the organisms but are not particularly

common except in the acute stages of an infection (Hamburger & Robertson, 1948; Hamburger & Green, 1946). Unfortunately, the saliva of only five of the throat carriers considered to have spread infection was examined, and in only one of the five did it yield large numbers of streptococci.

Nevertheless, about 70 % of all the persons developing streptococcal infection are known to have been in contact—in their cottage or school—with one or more carriers of streptococci of the same serotype, and for some 60 % one or more of these was a heavy nasal carrier.

We do not pretend that in all these cases the carrier we recognized was the source of infection in our index case. We have listed separately the ninety-four sore throats for which a definite source seemed to be discoverable and eighty of these could have been infected from a nasal carrier (Table 4). This might well be a biased sample, but nasal carriers could have been the source just as often in the cases for which we had less clear evidence.

In the recent accounts of streptococcal epidemics attempts have not often been made to specify the individuals responsible for the spread, and we know of no attempt at the sort of quantitative assessment given in Table 4. In several accounts of epidemics the importance of healthy carriers is stressed and it is therefore, perhaps, salutary to note that about 18 % of all the sore throats attributable to recognized contacts seem to have been contracted from persons who were in the incubation period of their illness. This, of course, would be regarded as commonplace in most infective diseases and indeed perhaps was also regarded as commonplace in streptococcal infections at the time when quarantine regulations were devised.

For this paper the analysis has been confined to the persons who were potential sources of infection. It is possible that the period for which a carrier may be dangerous can be extended if he contaminates fomites that can remain infected after the carrier has ceased to be dangerous. Such evidence as we have suggests that this is not common, but it will be more appropriately discussed in the subsequent section on the spread of infection in the cottages (Holmes & Williams, 1958*b*).

SUMMARY

1. For each case of streptococcal illness—sore throat, otitis, or other respiratory tract illness—observed in a 30-month study in a large children's home an attempt was made to enumerate the infected contacts from whom the infection might have been derived.

2. Of 459 illnesses, 10·5 % seemed to be relapses of a previous illness, and in 10·0 % the child was known to have been carrying streptococci for more than 7 days before sickening, so that the causal relation of the streptococcus is in doubt.

3. Of the 365 other illnesses, 31·5 % could be attributed with varying degrees of confidence to infection from one specified carrier, 36·8 % could have been derived from one or more recognized contacts although there was not sufficient evidence to specify one in particular. For 17·5 % no infected contacts were recognized, but infection with the particular streptococcus was known to be present in the

community; and for 9·3 % there were no contacts and the streptococcus was not known to be present in the Village.

4. Of the ninety-four illnesses for which the source could be specified as one particular carrier, 66 % were derived from heavy nasal, 19 % from light nasal, and 15 % from throat carriers. Persons incubating a streptococcal illness and healthy carriers were each responsible for 31 % of the illnesses and convalescent carriers for 38 %.

5. It seemed that at least 50 % of all illnesses could have been contracted from contacts within the cottage; the proportion due to school infection was much more difficult to estimate but was at least 12 %.

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