STREPTOCOCCAL INFECTIONS AMONG CHILDREN IN A RESIDENTIAL HOME

IV. OUTBREAKS OF INFECTION

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(With 1 Figure in the Text)

The previous papers in this series (Holmes & Williams, 1958*a–c*) recorded a 30-month study, among the 300–500 children resident in the Barkingside Village of Dr Barnardo's Homes, of the incidence of streptococcal infection, the potential sources from which individual children might have become infected, and the influence of various factors on their susceptibility to infection. This paper is concerned with the introduction of infection into, and the spread through, the cottages in which the children lived, and some similar analyses of the spread of infection into and through the Village as a whole.

COTTAGE OUTBREAKS

Of 367 cases of streptococcal illness among children and staff (excluding the 'suspect' infections and relapses defined by Holmes & Williams, 1958*b*), 169 were considered to be secondary to other cases in a total of 62 cottage 'outbreaks' (Table 1), and 128 were sporadic cases in the cottages; 20 were in fact part of recognized school outbreaks. There were 194 occasions when a streptococcus was thought to have been newly introduced into a cottage and produced one or more cases of illness. For 3 additional single-case introductions the streptococcus proved untypable or was lost and these introductions have been excluded from all analyses. Bacteriological investigations were made of the healthy contacts in the cottage on at least one occasion after 106 of the 194 introductions, the proportion being much higher for the introduction followed by secondary cases (58/62) than for those without (48/132). Two or more swabbings of the healthy children were made for 81 introductions, and for analyses of total bacteriological attack rates we have relied on the results from these 81 'well-swabbed' incidents.

Of the 194 new introductions of streptococci, 153 (79%) led to illness only among the children, 17 (9%) only among the staff, and 24 (12%) among both

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children and staff. The staff were much more often affected in *nursery-* or *baby-*cottage outbreaks (52 % of 56) than in *school-age* cottage outbreaks (9 % of 138). To a large extent this must have been due to the fact that the *nursery* cottages had an average of about ten nurses each compared with the average of about three for the *school-age* cottages; it probably also reflects the greater contact between staff and the younger children, and the fact that the staff in the *nursery* cottages were younger and had more recently arrived in the Village than those in the *school-age* cottages.

Table 1. *Cottage outbreaks of streptococcal infection*

	No. of introductions		No. of cases	
Single-case introductions	128	132	128	136
Two-case introductions without subsequent spread	4		8	
Introductions followed by one or more secondaries to give a total of:				
2 cases	26	62	52	231
3 cases	15		45	
4 cases	8		32	
5 cases	2		10	
6 cases	3		18	
7 cases	2		14	
8 cases	3		24	
12 cases	3		36	
Totals		194		367

Sore throats were by far the most common illness, making 237 (76 %) of the 310 illnesses among the children and 54 (95 %) of the 57 among the staff; 53 of the children's illness were otitis or otorrhoea and 20 were other respiratory tract illnesses. In most of the analyses all these streptococcal illnesses have been grouped together.

The small difference between the number of cases analysed in this paper and the number (365) analysed in Part II (Holmes & Williams, 1958*b*) arises from slight differences in the criteria for inclusion in the two series.

Source of cottage introductions

The following terms are used. The 'primary' is the first person to become ill in a particular episode and the first to be infected is the 'introducer'. The 'source' is the person (or place) from whom the introducer was infected. The primary case and the introducer often appeared to be the same person, but in some cases this must have been a false conclusion, due to the fact that we had insufficient bacteriological information about the healthy contacts of the primary case.

In 52 (27 %) of the 194 incidents the primary case appeared to have been infected from someone else within the cottage—a healthy carrier, a convalescent carrier transferred from another cottage, or an incubating carrier who was the second or third case to occur in the cottage (rows *a*, *b* and *c* of Table 2). In the remaining 142 incidents, the introducer and primary seemed to be the same person; but in 82 of these the cottage was not swabbed. For 22 (27 %) of these 82 introductions we could, nevertheless, indicate a likely extra-cottage source for the infection of the primary. We cannot tell how many would have been found to have intra-cottage

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sources had we examined the healthy contacts, but, of all the 103 cottages swabbed within a few days of the introduction, 43 (42 %) had their first case probably infected within the cottage. If this proportion were applicable to the last row of Table 2 we might have expected to find some 34 more healthy-carrier introductions—making 86 in all. This is probably an overestimate, since healthy-carrier introductions spread to secondary cases more often than others (see below), and were therefore more often examined bacteriologically.

Altogether, 58 of the 194 cottage introductions could be attributed to recognizable contacts with one or more known infected children. Another 14 were almost certainly new importations from outside the Village, and 40 of the remaining 122 introductions were of types that were not thought to have been present in the Village at the time. These are further discussed below under 'Village Epidemics' (p. 228).

Influence of mode of introduction on spread within cottage

Secondary cases of illness occurred more often when the introducer was a healthy or convalescent carrier than when the primary case appeared to be the introducer (rows *a* and *b* compared with *d* + *e* of Table 3). This may reflect the presence of the active disperser in the cottage over a longer period, in addition to possible spread from the primary case during the incubation period. Of the incubating carriers (row *c*) six later developed sore throat, one an otitis and one a sinusitis.

After allowing for the difference between intra-cottage and extra-cottage sources for the primary case, there was no difference in the frequency of secondary cases following introductions by nurses and children, nor following introductions by children who developed sore throat and those with otitis, etc.

Influence of carrier state

On the other hand, the carrier state of the primary case on admission to hospital had a notable effect. On the left side of Table 4 (based on incidents in which the primary case seemed to be the introducer) is shown the proportion of cottages with secondary cases within 7 days after the admission of the primary case to hospital ('early spread') against the carrier state of the primary case on admission to hospital. For those cottages with no 'early spread', the development of secondary cases after the return of the primary case from hospital ('convalescent spread') is similarly shown at the right of the Table.

Two differences emerge: patients with streptococci in their nose, especially those with large numbers, generated secondary cases more often than patients whose infection was localized to the throat; and patients with their tonsils removed were responsible for more 'convalescent spread' (but not more 'early spread') than those who retained their tonsils. The tonsillectomized children were more often nasal carriers than the non-tonsillectomized, but the standardized rates shown at the foot of the table suggest that, even when this is allowed for, nasal carriage and tonsil state (in convalescence only) both independently affect the incidence of secondary cases, although on the figures available the only trend to reach the 5 % significance level is that for different degrees of incubating carriage.

Table 2. *The source of infection of introducers*

Mode of introduction	Source of introducer's infection: From recognized contacts: At school	From recognized contacts: In hospital	From recognized contacts: Elsewhere	Introducer transferred from other known infected cottage	Outside Village	Not known: *Streptococcus* known to be in Village	Not known: *Streptococcus* not known to be in Village	Total: No.	Total: % of grand total
(*a*) By a healthy carrier	6	5 (1)*	0	3 (2)	3	16	6	39	*20·1*
(*b*) By a convalescent infected outside	0	2	0	3 (2)	0	0	0	5	*2·6*
(*c*) Introducer infected primary while incubating streptococcal illness	1	2	0	0	2	3	0	8	*4·1*
(*d*) Introducer thought to be primary case (cottage swabbed)	9	3 (1?)	3	3 (1)	5	30	7	60	*31·9*
(*e*) Introducer thought to be primary case (cottage not swabbed)	15 (1)	3 (2?)	0	0	4	33	27	82	*41·2*
Total	31	15	3	9	14	82	40†	194	
Percentage of grand total	*16·0*	*7·7*	*1·5*	*4·6*	*7·2*	*42·2*	*20·6*		

* Figures in brackets indicate number of introductions thought to be derived from epidemics at the nearby Woodford Homes.

† This total includes 3 introductions occurring within 2 weeks of the start of the study.

N.B. The number of introductions investigated bacteriologically for the five rows are, respectively, 31, 5, 7, 60 and 0; the 0 includes 3 examined only late in the incident.

Table 3. *Relation of mode of introduction to development of cottage outbreak*

Mode of introduction	No. of introductions	% with secondary cases	Total no. of cases		Mean clinical attack rate on children (%)	Mean bacteriological attack rate on children present throughout (%) (no. of outbreaks available in brackets)
			Children	Staff		
(*a*) By a healthy carrier	39	51·3	74	22	14·2	59·4 (25)
(*b*) By a convalescent infected outside the cottage	5	(60·0)	12	1	13·7	43·0 (2)
(*c*) Introducer infected primary case before becoming ill him-/herself	8	(75·0)*	25	10	21·5	69·7 (7)
(*d*) Introducer thought to be the primary case (cottage swabbed)†	59	45·7	113	14	14·2	38·2 (47)
(*e*) Introducer thought to be primary case (cottage not swabbed)	83	7·2	86	10	10·2	— —
(*d*) + (*e*)	142	23·2	199	24	11·9	— —
Total	194	32·0	310	57	12·7	47·6 (81)

* In 1 incident the incubating carrier, and in another the only apparent secondary case, were 'suspect' as having carried the streptococci for more than 7 days before sickening; they do not therefore appear in this table as 'cases'.

† Three cottages swabbed only once late in the incident are regarded as not swabbed.

Table 4. *Relation of carrier state of primary case to occurrence of secondary cases in the cottage*

		'Early spread'		'Convalescent spread'	
Carrier state	Tonsil state	No. of carriers	% with secondary cases	No. of carriers	% with secondary cases
Heavy nasal	In	14	29	9	(22)
	Out	8	(13)	8	(75)
Light nasal	In	30	10	13	23
	Out	10	10	3	(33)
Throat only	In	62	6	33	9
	Out	6	0	5	(20)
All carriers (rates standardized for degree of carriage)	In	106	11·1	55	15·3
	Out	24	5·3	16	36·1
Heavy nasal	Standardized for tonsil state	22	26·0*	17	33·9
Light nasal		40	10·0	16	25·3
Throat only		68	4·9	38	11·5

* This trend is significant; P = about 0·02.

Notes. Seventeen patients were not swabbed on discharge and only one spread infection; twenty-nine of the primaries were negative on return from hospital and spread occurred in 2 cottages (7 %) and was attributable to a healthy carrier contact. There were 12 introductions due to double primaries or in which the introducers' tonsil state was not known; one spread.

We have no certain explanation for the independent effect of the tonsils. Tonsillectomized children convalescent from sore throat carried streptococci in their noses for the same period as those with tonsils (Holmes & Williams, 1958*a*, Table 7); but there is some indication in the data collected but not included in Table 7 of that paper, that the period of carriage of large numbers of streptococci was longer in the tonsillectomized children. There is no evidence that they were more likely to carry streptococci in their saliva.

For equivalent degrees of carriage, convalescent carriers appeared to give rise to spread more often than incubating persons, as might be expected from the greater period of exposure.

Of the fourteen convalescent carriers who generated secondary cases, eight were discharged from hospital carrying as heavily as at any subsequent swabbing; six were heavier carriers at their later examination than at discharge and seem to have infected the secondary cases only after an interval.

Of twenty healthy carriers who were regarded as introducers of cottage epidemics, twelve (60 %) were heavy and four (20 %) light nasal carriers; six had had their tonsils removed (Table 5, cols. 3, 4).

The 'dangerous' carriers, whether incubating, convalescent or healthy are therefore similar: they are commonly heavy nasal carriers (45–60 %), and they have more often had their tonsils removed than the general run of infected children (Table 5).

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Table 5. *Characteristics of 'dangerous' carriers*

		'Dangerous' carriers							
		Primary-case introducers who started cottage outbreaks (from Table 4)		Healthy carriers who started cottage outbreaks		Primary cases who failed to generate infection, on admission to hospital, (from Table 4)		All hospital admissions with streptococcal infections*	
Carrier state	Tonsils	No.	% of total	No.	% of total	No.	% of total	No.	% of total
Heavy nasal	In	6	21 } 45	7	35 } 60	10	9 } 15	41	13 } 21
	Out	7	24 }	5	25 }	7	6 }	27	8 }
Light nasal	In	6	21 } 28	3	15 } 20	27	23 } 31	85	26 } 33
	Out	2	7 }	1	5 }	9	8 }	24	7 }
Throat only	In	7	24 } 27	4	20 } 20	58	50 } 55	136	42 } 46
	Out	1	3 }	0	0 }	6	5 }	14	4 }
All	In	19	66	14	70	95	81	262	80
	Out	10	34	6	30	22	19	65	20
Total	—	29	—	20	—	117	—	327	—

* This figure differs from that for the total number of illnesses by the number of children with otitis not admitted to hospital and the number not swabbed exactly on the day of admission.

Table 6. *Influence of streptococcus type on attack rates in cottage introductions*

				In introduction with children affected						
				Cases of streptococcal illness						
Type	No. of cottage introductions	Total no. of cases	No. of introductions with children affected	No. of cases among children	Mean child cases per introduction	Mean attack rate on total cottage population (%)	No. of incidents adequately swabbed	Mean bacteriological attack rate on total population (%)		Ratio, bacteriological attack rate to clinical attack rate on total cottage population
2	32	79	30	63	2·1	15·6	11	48·3		2·1
5	31	72	30	68	2·3	16·6	15	37·3		3·0
22	24	48	21	39	1·9	15·1	11	39·2		1·8
Lily	23	34	23	32	1·4	9·4	8	50·1	43·3	4·9
12	15	32	14	25	1·8	14·8	9	43·0		3·1
9	12	31	12	28	2·3	17·0	7	47·4		2·2
4	11	12	8	9	1·1	11·1	1	71·5		2·5
28	9	9	6	6	1·0	9·5	0	—		—
Corby	8	13	6	9	1·5	12·3	4	33·0		3·2
25	5	5	5	5	1·0	10·0	1	8·3		1·0
1	4	4	4	4	1·0	9·3	0	—		—
3	4	6	4	5	1·3	9·5	2	37·5		3·4
6	4	7	4	4	1·0	9·0	3	75·7	45·5	10·0
Angas	4	7	4	7	1·8	15·0	2	53·0		2·8
11	3	3	2	2	1·0	7·5	0	—		—
19	2	2	1	1	1·0	8·3	1	16·7		2·1
36	2	2	2	2	1·0	9·0	0	—		—
18	1	1	1	1	1·0	8·3	0	—		—
Totals	194	367	177	310	1·75	13·9	75	43·7		—

Note. It is not possible to calculate attack rates for the staff in the cottages so that the rates given in this table are based on the 'total' child population in the cottage, that is, all children present in the cottage at any time during the outbreak.

The heavy nasal carriers who started cottage outbreaks were on the average each responsible for 1·9 secondary cases, while the light nasal and throat carriers generated 1·3 and 1·2 cases, respectively. Tonsillectomized carriers who started outbreaks generated no more than non-tonsillectomized (1·7 and 1·6).

Influence of the streptococcus

Eighteen different serological types of streptococcus were introduced into the cottages during our survey. Six of these (*Types 2, 5, 22, Lily, 12* and *9*) were each introduced twelve or more times (Table 6), and were together responsible for 137 (71 %) of the total introductions and 255 (82 %) of the 310 cases of infection in children.

The six types most frequently introduced were also, with the exception of *Type Lily*, those that produced the highest clinical attack rates, with means of 14·8–17·0 %. Only one other type (*Angas*) had a rate as high as this. The bacteriological attack rates (i.e. the proportion of children in the cottage who yielded the streptococcus at any time during the outbreak), perhaps because based on smaller numbers, showed less stability, but there was no suggestion that they were higher with the more frequent types. Indeed, the weighted mean attack rate for the first six types is 43·3 % and that for the remainder (on only fourteen outbreaks) 45·5 %.

The last column of Table 6 gives the ratio of the bacteriological to clinical attack rates; it is thus a measure of the number of children infected for each child ill. *Type 6* clearly differed from the rest in having a mean ratio of 10; the three outbreaks had ratios of 12·3, 10·4 and 7·3. There was also a suggestion that *Type Lily* had a consistently high ratio. Of those types that affected any number of cottages only *Type 22* had a ratio of less than 2·0.

The spread of the six 'Epidemic Types' (Table 7) could be partly explained by the fact that 59 % of their introducers were nasal carriers, compared with only 37 % of the introducers for the other types ($d = 22$ %, S.E.$_d = 8{\cdot}5$ %). But even allowing for this, the 'Epidemic Types' seemed to have a greater tendency to give rise to secondary cases since 41 % (23/56) of their nasal-carrier introductions led to spread of infection, compared with 13 % (2/16) of the nasal-carrier introductions of other types ($d = 28$ %, S.E.$_d = 13$ %).

Influence of type of cottage

No very striking differences were found between the *baby* and *nursery* cottages as compared with the *school-age*, nor between the *reception* and *permanent* cottages.

There was a suggestion that infection spread less often in the cottages that had the smaller number of children exposed. Only 7 (15 %) of 46 introductions into cottages with a total population of six to nine children were followed by secondary cases compared with 56 (38 %) of the 148 introductions into cottages with 10 or more ($d = 23$ %, S.E.$_d = 8$ %).

The change in the cottage population during the period of the outbreak was measured by the ratio of the number of children present at any time in the outbreak to those present throughout, or for 1 month after a single-case introduction. Except that all 5 cottages with ratios of 2·0 or more had secondary cases, the

Table 7. *Influence of type of streptococcus on spread from cottage introductions*

	Primary case thought to be cottage introducer (*d* and *e* of Table 3)				Introduction attributed to healthy, convalescent or incubating carrier (*a*, *b* and *c* of Table 3)	
	Primary case a nasal carrier on admission to or discharge from hospital		Primary case never a nasal carrier			
Streptococcus type	No. of introductions	No. with secondary cases	No. of introductions	No. with secondary cases	No. of introductions	No. with secondary cases
2	11	4	10	0	9	5
5	16	6	5	0	9	3
22	7	5	8	1	9	7
Lily	9	3	10	1	4	2
12	7	3	2	0	6	4
9	6	2	5	4	1	0
Subtotal	56	23	40	6	38	21
Other types	16	2	27	2	14	5

Note. In 3 of the introductions thought to be due to the primary case, there were two simultaneous primaries; these are excluded from this table.

turnover did not appear to influence the frequency with which introductions were followed by secondary cases. There was, nevertheless, a positive correlation between the final clinical attack rate and the turnover ratio ($r = +0{\cdot}26$; $0{\cdot}05 > P > 0{\cdot}02$).

No relation was detected between the attack rate and the proportion of the children in the cottage who had had their tonsils removed.

It will be shown below that the 194 cottage outbreaks belonged to sixty-three different 'Village epidemics'. We therefore sought evidence for any change in the infectivity of the streptococci in the course of the Village outbreaks, but were unable to find more than a suggestion that the clinical attack rate was highest in the second cottage to be invaded; there was no change in the ratio of bacteriological to clinical attack rate during the Village epidemic.

Dynamics of cottage outbreaks

In studying the method of spread of infection within the cottage it was possible to recognize four broad categories (Table 8). In the first (rows *a* and *b*), with 22 introductions, the initial spread to the secondary cases appeared to be from the incubation-stage carriage of the primary, who later became sick. In fourteen instances this spread seemed, from the serial intervals of the cases and the available bacteriological results, to be entirely from the primary case; in eight, intermediary healthy carriers might have been responsible. For twelve of the twenty-two incidents with early spread all the cases occurred within 7 days of the primary.

In the second category (rows *c* and *d*), with 26 introductions, no secondary cases occurred until the primary case had returned from hospital, and the spread that occurred then was considered to be due to his or her convalescent carriage, either directly or through healthy intermediaries. The interval between the first and second cases was at least 5 days (in one instance when the primary was discharged unusually soon) and averaged about 19 days. Although there was commonly a cluster of secondary cases at one time, this was less marked than in the incidents with early spread; 64 % of all the clinical illnesses in the latter and 31 % of those in the former occurred within a single week. The final attack rates of the two types of outbreaks did not differ to any notable extent.

Three outbreaks seemed to be entirely due to spread from a convalescent carrier who had been infected elsewhere and discharged from hospital into the affected cottage (row *e*).

A fourth category (row *f*) comprised six incidents in each of which all the patients seemed to have become infected from a single healthy carrier. As indicated in the footnote to Table 8 several of the outbreaks in rows *a* to *e* were initiated by healthy carriers, and the later spread in some of these should perhaps really be attributed to infection from the healthy-carrier introducer rather than to spread from the primary case.

A further analysis was made in an attempt to determine the potential value of exclusion of convalescent carriers from the cottage. When the introducer of an outbreak was infected from a convalescent carrier, we assumed that this outbreak could have been prevented by adequate chemotherapy, as could any further outbreaks in other cottages attributed to spread of infection from the first. Similarly,

Table 8. *Mode of spread to secondary cases in cottage outbreaks*

	Introductions		Secondary cases		Mean no. of cases within 7 days of primary case	No. of incidents with all cases within 7 days of primary	Max. no. of cases in 7 days	Mean span of onset (days)		Final attack rate on total child population (%)
	No.	% of incidents with spread	No.	% of total				Cases 1 and 2	All cases	
Initial spread apparently due to										
(*a*) Incubation-stage carriage of primary, probably directly	14	22·6	44	26·0	2·9	6	3·0	3·3	33·7	26·4
(*b*) Incubation-stage carriage of primary, probably via healthy carriers	8	12·9	25	14·8	2·4	6	2·6	3·6	25·9	19·0
(*c*) Convalescent carriage of primary, directly	14	22·6	39	23·1	1·1	1	2·2	20·3	31·2	24·7
(*d*) Convalescent carriage of primary, possible via healthy carriers	12	19·4	31	18·4	1·1	0	1·4	18·7	39·4	21·0
(*e*) All spread due to a convalescent carrier infected elsewhere	3	4·8	12	7·1	(1·3)	0	(2·0)	(8·0)	(51·7)	(15·7)
(*f*) All spread due to one introducer, a healthy carrier	6	9·7	11	6·5	(2·5)	4	(2·7)	(5·5)	(18·5)	(21·2)
(*g*) Mode of spread doubtful	5	8·1	7	4·1	—	—	—	—	—	—
(*h*) No spread (including 4 two-case introducers)	132	—	0	—	—	—	—	—	—	—
Total (means based on all rows except last)	194	—	169	—	1·8	—	2·2	13·9	34·0	21·8

Note. The twenty healthy-carrier introductions with secondary cases (see Table 3) are distributed over the rows of this table as follows: row *a*—2 introductions, row *b*—2, row *c*—2, row *d*—5, row *e*—1, row *f*—6, row *g*—2.

with secondary spread from the convalescent carriage of an introducer, the source of whose infection was unknown, it was assumed that all the secondary cases could have been prevented. A similar analysis was made for hospital infections and for infections derived from incubating carriers. Altogether, 184 of the 367 infections could be allocated to one of these three sources and 63 others were the primary cases of Village introductions (see below p. 229) (Table 9). Judged from these figures, 25 % of all cases would have been prevented if infection from convalescent carriers had been eliminated. It is almost certain, however, that this is an underestimate since there were 120 infections that could not be allocated to any of these sources.

Table 9. *Numbers of infections ultimately attributed to various recognized sources*

Type	Total no.	% attributed, directly or indirectly, to infection from: Convalescent carrier	Incubating carrier	Hospital	% regarded as new introductions to the Village
2	79	28	18	15	8
5	72	36	18	14	11
22	48	44	10	1	8
Lily	34	26	12	0	18
12	32	28	19	0	13
9	31	0	58	0	13
Total for above six types	296	29	20	8	10
Other types	71	7	0	13	47
Total	367	25·0	16·4	8·7	17·2

Note. The source of infection for the remaining cases was not discovered.

One curious fact emerges from Table 9: of the secondary cases of *Type 9* infection whose source could be recognized (constituting 58 % of all the *Type 9* infections) all were derived from incubating carriers and none from a convalescent carrier; we can find no explanation of this.

Carrier rates among healthy children in the cottages

It was not practicable to make bacteriological investigations of the cottage contacts of all children developing streptococcal illnesses, but we were able to do so in most of the cottages in which two or more children were ill, as well as in a reasonable proportion of the single-case outbreaks. The period for which observation was continued depended on the continuance of cases of illness, and also, partly, on the carrier rates observed.

The carrier rates observed among the children present in the cottage at examinations in the week before, and at various times after the sickening of the first child are shown in Table 10. The rate was consistently lower in the cottages experiencing only a single case than in those in which clinical spread occurred.

Table 10. *Carrier rates in cottages experiencing outbreaks*

Day of swabbing (day of first case = day 0)	Single-case introductions				Introductions with secondary cases: Spread concluded†				Introductions with secondary cases: Spread not concluded			
		Mean % carrier rate				Mean % carrier rate				Mean % carrier rate		
	No.	All children	All except convalescents	N/T ratio,* all children	No.	All children	All except convalescents	N/T ratio, all children	No.	All children	All except convalescents	N/T ratio, all children
− 7 to − 1 incl.	8	8·6	.	0·86	.	.	.	.	4	28·8	.	0·85
0 to + 6 incl.	25	15·8	14·2	0·51	.	.	.	.	24	29·4	28·5	1·00
+ 7 to + 13 incl.	10	11·8	8·6	0·71	5	44·4	47·8	0·67	17	45·5	41·2	0·69
+ 14 to + 20 incl.	15	9·7	5·3	0·43	10	39·6	34·5	0·59	20	39·0	33·8	0·69
+ 21 to + 27 incl.	12	17·1	14·8	0·37	10	37·5	37·7	0·57	12	51·4	38·4	0·64
+ 28 to + 34 incl.	5	(25·4)	(21·2)	(0·65)	16	39·0	29·4	0·37	7	38·4	23·9	0·58
+ 35 to + 48 incl.	6	(14·5)	(11·8)	(0·87)	20	40·3	35·5	0·40	6	(48·7)	(40·8)	(0·35)
+ 49 to + 62 incl.	0	(—)	(—)	.	14	35·5	28·4	0·34	6	(47·3)	(37·0)	(0·44)
+ 63 to + 90 incl.	2	(41·5)	(43·0)	(0·23)	13	24·4	18·3	0·33	3	(60·3)	(44·4)	(0·38)

* N/T = nose carrier rate/throat carrier rate.

† For each row all illnesses had developed before the first day entered under 'day of swabbing'.

There was an indication that the carrier rate tended to increase during the first 2 weeks after the onset of the first case, perhaps because returning convalescents infected more healthy contacts, and this impression is confirmed when the records of individual cottages are examined. Thus, of 37 cottages examined on the day on which the primary sickened or within 6 days afterwards, and again between days 14 and 27, 26 had higher carrier rates at the second swabbing, 4 the same, and 7 lower rates. Of 19 cottages first swabbed between 7 and 13 days from the day of the primary's sickening, however, only 9 showed a rise at a second swabbing between days 14 and 27, and 8 showed a fall.

Carrier rates seemed to be lower at the examinations made after clinical spread had concluded, but it is noteworthy that several cottages still had high carrier rates 10 weeks after the occurrence of the first case. Such falls in carrier rates that were observed seemed to be almost equally due to clearance of carriers and to changes in cottage population involving departure of carriers or their dilution by newcomers.

There was a clear relation between the carrier rate at examinations made in the first 6 days and the final bacteriological attack rate (Table 11); the correlation coefficient was $+0{\cdot}766$ ($P < 0{\cdot}001$). There was a similar but less marked trend with clinical attack rate ($r = +0{\cdot}26$, $0{\cdot}10 > P > 0{\cdot}05$).

Table 11. *Relation of initial carrier rate to final attack rate*

Carrier rate on healthy children at examination within 7 days of first case (%)	No. of introductions	Final attack rates (%)	
		Bacteriological	Clinical
0–9	14	21	14
10–	10	32	12
20–	6	38	13
30–	3	44	12
40–	2	39	17
50–	3	67	17
60–	3	74	20
70–	1	64	18
80–	1	89	22

The twelve swabbings carried out between 1 and 7 days before the first child sickened were usually concerned with the aftermath of a previous incident. At four of the swabbings no carriers were found; at three the only carrier was the child who was subsequently to be the first case of illness; and in the remaining five, one or more healthy carriers were found. In three of these one of the carriers was the presumed introducer of the infection.

Five cottages were examined between 8 and 14 days before the first illness and 2 were found to have carriers of the epidemic type. Five were examined more than 14 days before the first illness and again 2 had carriers; one of these was notable in having 80 % of the children carriers 17 days beforehand. This was a single-case incident due to *Type 6* in a nursery cottage, and there were no changes in the population between the day of swabbing and the day on which the child sickened.

Duration of outbreaks and serial intervals between cases

The duration of the cottage outbreaks varied greatly; the longest interval between the first and last case that appeared to follow a single introduction was 210 days, and there were several incidents that lasted for 60–90 days. These differences result largely from the different ways in which the infection spread—whether from incubating, convalescent or healthy carriers. Presumably the carrier state of convalescents and healthy contacts must also play a part, although in the swabbings given in Table 10 the nose/throat carrier ratio is not significantly lower in the cottages in which spread has concluded than in those in which the outbreak was still active.

Table 12. *Serial intervals between cases in nineteen large outbreaks*

No. of clear days elapsing between successive cases (all illnesses)	No. of cases		No. of cases that were in newcomers to the cottage		Notes
– 6	67		0		.
7–13	12		0		.
14–20	9		0		.
21–27	7		5		*a*
28–34	2		.		*a*
35–41	3		2		.
42–48	2	17	1	10	.
49–55	1		1		.
56–62	1		.		*b*
133	1		1		*c*

Notes. *a*, one case infected with another type earlier; *b*, one child was ill following re-infection and had been infected much earlier; *c*, child present in cottage 110 days before sickening although not present at the time of initial spread.

There is no doubt that prolongation of clinical spread can also be due to the arrival of new children into an infected cottage. In Table 12 are shown serial intervals between cases in the 19 well-swabbed incidents with 4 or more cases among children or staff. The serial intervals were usually less than a week, and of the seventeen persons sickening more than 3 weeks after the last previous case, ten were newcomers to the cottage and had not been present at the time when the earlier cases had occurred, two had been infected earlier with another type, and one was a re-infection. (It has to be noted, however, that one of the newcomers was in the cottage 110 days before going sick.) Children infected at the time of the initial spread of infection often continued to carry the streptococcus for some time, and it seemed clear that some at least of these prolonged carriers were able to transmit their infections to others even after 3–5 weeks. Of the seventeen instances of children or nurses sickening 21 or more days after the last previous case, six were known to have one or more heavy and seven others one or more light nasal carriers among their contacts.

When the cottages were classified according to the turnover of children during the outbreak (as described on p. 219) it was found that 9 (39 %) of 23 spreading

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outbreaks in cottages with no, or practically no change in their population (ratios of 1·0 or 1·1) lasted for more than 20 days. In 26 spreading outbreaks in cottages with a greater turnover, 58 % lasted for more than 20 days, but within this group increased changes in the cottage population did not seem to have any distinct effect.

There were twenty-three incidents in which new cases of illness occurred after the 20th day from the first case and in which the healthy and convalescent children were swabbed between days 25 and 45. Nineteen (83 %) of the cottages had one or more nasal carriers present, and 3 of the others were known to have had a nasal carrier at about day 20. Of 39 cottages with no new cases after the 20th day, 25 (64 %) had one or more nasal carriers. It is clear, therefore, that the occurrence of late cases usually implied the presence of nasal carriers, but the converse was not true.

As is to be expected from the details just given, the interval between the first and second cases bore little relation to the final extent of an outbreak. Thus, in twenty-nine incidents the first 2 cases occurred within 5 days and the mean final and clinical attack rate on the child population of these cottages was 20·4 %; in thirteen incidents with a span of 6–10 days the rate was 25·0 %; and in six with a span of 11–15 days the rate was 21·5 %.

Bedroom spread

For forty-eight of the outbreaks with more than one case of illness among the children we had records of the particular bedroom occupied by each child. In only four of the forty-eight was there any suggestion that spread of infection occurred in the bedrooms, and one of these was an outbreak in a cottage for babies under 18 months of age, in which the babies in one room have no contact with those in other rooms. In thirty of the forty-eight outbreaks it seemed clear, from the distribution of the children who became ill or were infected first, that bedroom spread played little or no part. In the remaining 14 cases the evidence is somewhat equivocal, usually because infection was already widely distributed at the time of the first swabbing of the cottage.

Schoolroom outbreaks of infection

Owing to the limited bacteriological investigations, much less information was available about schoolroom outbreaks of infection than about cottage outbreaks. There were fifteen occasions when one or more cases seem to have been infected at school—that is they attended the same class but were living in a different cottage from the primary case. In nine of these incidents there was a single classroom secondary, in four there were 2, and in two there were 3 secondary cases. Of the last, one was a *Type 12* outbreak in the nursery school occurring at the time of the widespread *Type 12* infection, and one was a *Type Lily* outbreak in one of the school classes. In five instances heavy nasal carriers, and in four light nasal carriers, are known to have been present in the class before the occurrence of the first illness. In several instances these carriers were also held responsible for the spread of infection in their cottage.

VILLAGE EPIDEMICS

We showed in Part I (Holmes & Williams, 1958*a*) that, although the incidence of streptococcal infections varied from month to month, there was no period of very greatly increased prevalence. This was due to the fact that the Village experienced a series of overlapping epidemics due to different types of streptococci. The general picture is illustrated in Fig. 1, which gives the monthly sore-throat attack rates

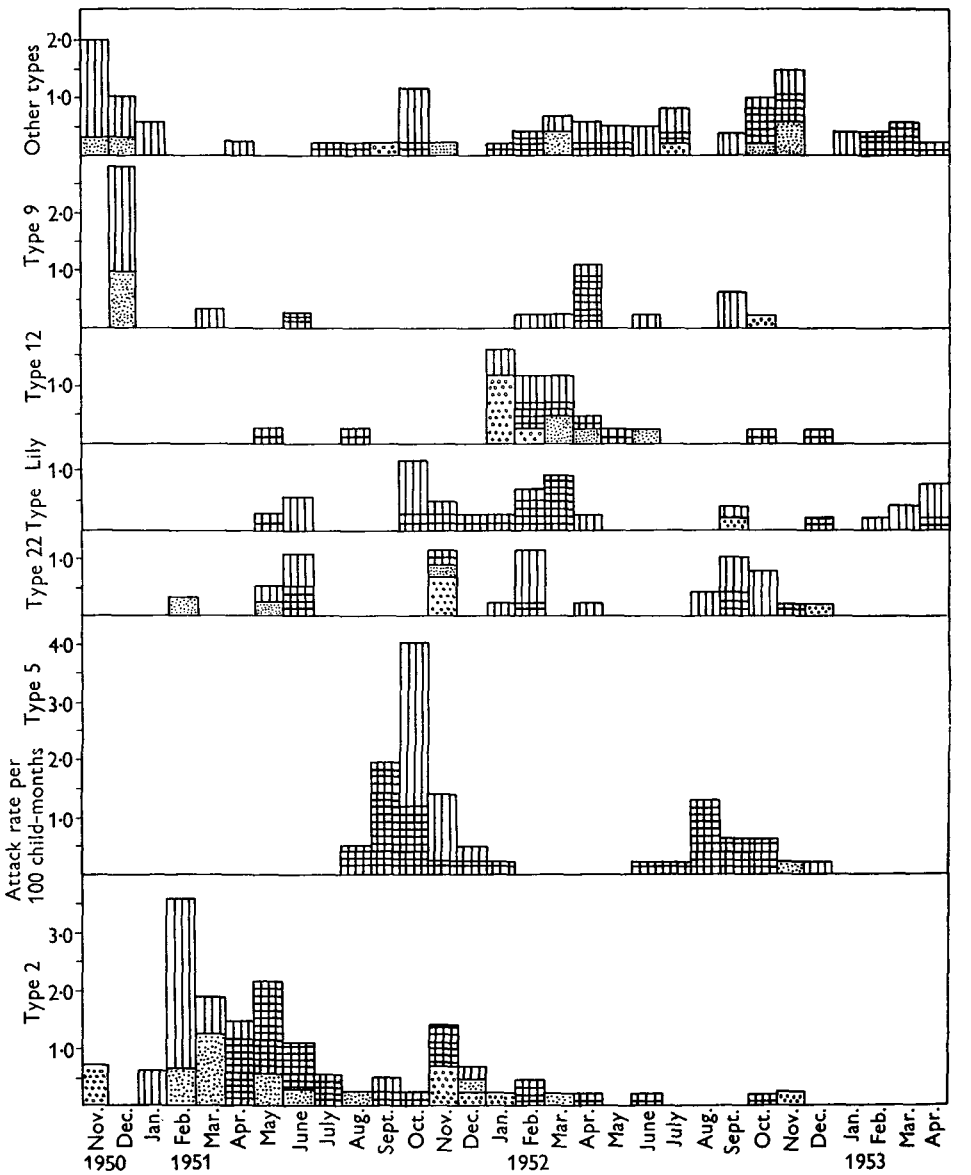

Fig. 1. Incidence of infections due to different types of *Streptococcus pyogenes*. The shading indicates the proportion of all cases occurring in the four divisions of the child population: reception nursery, ▨; reception school-age, ▦; permanent nursery, ▩; permanent school-age, ▥.

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per 100 children, and the contributions to this rate of children in the different cottage groups.

The outbreak due to *Type 2* was the largest, commencing sharply in January 1951 and decreasing slowly from its initial peak over some 16 months. The initial spread was largely confined to *school-age* children in the *permanent* group, but later all groups were affected. The outbreak due to *Type 12* was similar to the *Type 2* outbreak in having an initial high incidence and a slow decline; it also affected all population groups. Two separate *Type 5* outbreaks were seen, the first starting in the *reception school-age* group and later spreading to the *permanent* group, and the second practically confined to the *reception* group.

The highest attack rate for any one type in any month was just over 4 % (*Type 5*, October 1951); an attack rate of 3·6 % in one month was seen with *Type 2*, and of 2·8 % with *Type 9*. The height of the columns in Fig. 1 indicates the attack rate on the whole child population, but the components of the columns do not indicate directly the attack rates on the populations of the various subgroups. When the attack rates were computed on the basis of the subgroup (*permanent nursery*, *permanent school*, etc.) attack rates of up to 10–15 % were recorded, but these were naturally based on small numbers of children.

Village introductions

A streptococcus type was regarded as newly introduced into the Village if either: (*a*) the child first ill with it was a new arrival considered to have been infected outside the village; or (*b*), not knowing the source of infection in the first case, the streptococcus was thought to have been absent from the Village (cases or known carriers) for 1 month or more. On these grounds there were 63 separate Village introductions, including 4 observed in the first month of the study. If the interval under (*b*) had been 2 months instead of 1, the number would only have been reduced by 6.

Some 80 % of all introductions resulted in 5 or fewer cases of illness, and only 8 (13 %) had more than 10 cases.

Source of Village introductions

For 40 (64 %) of the 63 Village introductions the source of the infection is quite unknown; on the average these introductions occurred about 3½ months after the last previous case of illness in the Village due to the same type of streptococcus. In 14 cases (22 %) the introduction seemed to be attributable to a new arrival in the Village; 10 of the introductions appeared to be by healthy carriers. Another 9 introductions were evidently derived from the nearby Woodford branch of Dr Barnardo's Homes; 5 of these were due to the transfer of children infected at Woodford into Barkingside cottages, and 4 were due to cross-infection in the hospital—in which children from Woodford, as well as from Barkingside, were nursed. In several cases the transfer of infected children occurred during the spring and summer of 1951 when a large group of 'reception' children was transferred from the Woodford to the Barkingside Home. Two of the hospital cross-infections

occurred in early 1952; none occurred after the opening of a new isolation block in the Barkingside hospital in February 1952.

In 10 of the 63 introductions a nurse was the first person to be infected. In one of these cases the infection was derived from Woodford and in two others the nurse was definitely thought to have been infected outside the Village; the source of the other 7 introductions is not known. Five of the 30 child-introducers with no recognized source of infection were attending schools outside the Village, at which they might have become infected but we know nothing of most of their class-mates.

Thirty-five (56 %) of the introductions were in *reception* cottages. This represents a rate of about 0·81 introductions per 100 child-months of exposure, and is considerably greater than that of the *permanent* cottages, which was 0·33 per 100 child-months.

Factors influencing the outcome of a Village introduction

The nature of the cottage first affected did not have any pronounced effect on the outcome of an introduction, although spread was slightly more common from the *permanent* cottages than from the *reception*. Similarly, spread was slightly more frequent from *school-age* than from *nursery* or *infant* cottages, even though the *school-age* cottages had fewer introductions than the *nursery*.

Table 13. *Relation of number of cases in first cottage affected to spread through Village*

No. of cases in first cottage affected	No. of introductions	Mean no. of cottages affected	Mean no. of cases in Village
1	49	2·3	3·8
2	6	3·7	6·7
3	5	7·4	15·2
4	1	8·0	25·0
12	1	1·0	12·0
3 or 7	1*	12·0	29·0

* Two cottages affected almost simultaneously.

Spread to other cottages was clearly dependent on the extent of spread in the first cottage affected (Table 13); 49 introductions with only one case in the first cottage spread, on the average, to 2·3 other cottages and generated 3·8 cases, while 6 introductions giving 2 cases in the first cottage spread to 3·7 with 6·7 cases, and 5 cottages with 3 cases spread to 7·4 cottages with an average of 15·2 cases. The one incident with twelve cases in the first cottage included some children with scarlet fever; in consequence that cottage was held in quarantine, and this doubtless explains the absence of spread to other cottages. A similar trend is seen when Village spread is related to the bacteriological attack rate in the first cottage, although for only 26 of the introductions was the cottage examined bacteriologically: 9 of 10 introductions with infection rates of less than 20 % in the first cottage failed to spread to any other cottage and one resulted in a total of 6 cases in the Village, giving a mean of 1·5 cases per introduction. The means for introductions with 20–39 %, 40–59 %, and 60–79 % infection rates were, respectively,

6·3 (3 introductions), 9·5 (6) and 13·6 (5). One introduction giving an infection rate of 83 % generated 7 cases and one introduced simultaneously into 2 cottages with infection rates of 40 and 92 %, generated 29 cases.

The number of cases arising from a Village introduction was, as was to be expected from the analysis of cottage outbreaks, related to the carrier state of the primary case of infection. There were 9 introductions where the apparent Village introducer was a heavy nasal carrier; only 1 (11 %) of these failed to spread and the mean number of cases per introduction was 15·8. Of 20 introductions by light nasal carriers, 12 (60 %) failed to spread and the mean number of cases was 4·2, or, excluding a single introduction generating 29 cases, 2·9. There were 33 introductions with simple throat infections in the primary, 22 (67 %) of which failed to spread, and the mean number of cases was 3·0, or, excluding 2 introductions with 17 and 38 cases, 1·5. (In one additional introduction, due to the transfer of an infected group of children, no single primary case could be recognized.)

For twenty incidents, the introducer was a *school-age* child attending the school in the Village grounds; these involved an average of 4·4 cottages; 18 introductions by *school-age* children attending no school or a school outside the Village involved an average of only 2·6 cottages. On the figures available, however, this difference is not significant.

The types of streptococci that produced the largest proportion of cases were naturally the types that occurred in the most marked epidemics (Table 14). Of the twelve types introduced on more than two occasions, only two—*Type 1* and *Type 25*—failed to spread, although *Type 3* never produced more than 2 cases at an introduction.

The interval between successive Village introductions of one streptococcal type did not appear to affect the spread at the second introduction; in fact the proportion of second introductions with more than 5 cases was greatest when the second introductions occurred within 2 months of the first. The number of cases arising from the first introduction had, however, a striking effect (Table 15); no outbreak of more than 10 cases followed an outbreak of more than 5 cases, and, of 7 second introductions following a first that had generated more than 10 cases, none spread at all.

The total number of cases of streptococcal infection, regardless of type, occurring in one 2-month period had no detectable effect on the outcome of introductions occurring in the subsequent 2-month period, nor had the season of introduction.

DISCUSSION

Among the factors that have been suggested as responsible for initiating epidemics of streptococcal infection, three—the introduction of new types of streptococci, the introduction of new and susceptible children, and the occurrence of the 'dangerous-carrier' state—seem to be the most relevant to our study. We could detect no effect of season or of concomitant epidemics and it is difficult to envisage that changes in, for example, the nutritional state of the children could lead to the epidemiological picture that we observed.

Table 14. *Village introductions, by type of streptococcus*

Type (in same order as Table 6)	Total introductions to Village, assuming minimum interval of 1 month	No. of Village introductions with no secondary cases	Streptococcal illnesses in children or staff: Nos. in introductions that spread	Streptococcal illnesses in children or staff: Mean no. per Village introduction	Mean no. of cottages involved, per Village introduction	Mean attack rate (%) on children (from Table 6)
2	4*	2	2, 75	19·8	8·0	15·6
5	8	5	40, 2, 25	9·0	3·9	16·6
22	4	1	3†, 6, 38	12·0	6·0	15·1
Lily	6	2	3, 20, 2, 7	5·7	3·8	9·4
12	4	3	29	8·0	3·8	14·8
9	4	2	12, 17	7·8	3·0	17·0
4	5	3	4, 5	2·4	2·2	11·1
28	5	3	2, 4	1·8	1·8	9·5
Corby	3	1	8, 4	4·3	2·7	12·3
25	5	5	—	1·0	1·0	10·0
1	4	4	—	1·0	1·0	9·3
3	3	0	2, 2†, 2†	2·0	1·3	9·5
6	1	0	7	7·0	4·0	9·0
Angas	2	1	6	3·5	2·0	15·0
11	1	0	3	3·0	3·0	7·5
19	2	2	—	1·0	1·0	8·3
36	1	0	2	2·0	2·0	9·0
18	1	1	—	1·0	1·0	8·3
All types	63	35	—	5·8	3·1	13·9

* Twelve cases in one cottage, and one in another may have been secondaries to two additional introductions, not included in the table, occurring during a *Type 2* Village epidemic.

† In these introductions all cases occurred in one cottage.

N.B. Occurring within the first month of the study, although included in column 2, were:

Type 2, 1 introduction affecting a total of 2 cottages, and two patients.
4, 1 introduction affecting a total of 1 cottage, and one patient.
28, 1 introduction affecting a total of 2 cottages, and two patients.
Corby, 1 introduction affecting a total of 1 cottage, and one patient.

Table 15. *Influence of number of cases arising from one Village introduction on number arising from a second introduction of the same streptococcal type*

No. of cases from previous Village introduction	Number of introductions with: 1 case	2–5 cases	6–10 cases	>10 cases	Total
1	14	4	3	5	26
2–5	3	3	1	3	10
6–10	1	1	0	0	2
>10	7	0	0	0	7
Total	25	8	4	8	45

(When the sixteen entries are grouped symmetrically to give a fourfold table, Fisher's 'exact' test gives a probability that the observed distribution would occur by chance as 1 in 23.)

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The analyses both of the individual children's experience (Holmes & Williams, 1958*c*) and of the Village as a whole (Table 15) demonstrate the importance of new types. It is clear that individuals were seldom re-infected when re-exposed to a given type, and the Village as a whole never suffered two consecutive large epidemics due to the same type. This experience is closely parallel to that of Griffith, in his study of epidemics in public schools (*Report*, 1938).

Six different *Types—2, 5, 9, 12, 22* and *Lily*—were more 'communicable' during our study than the others, but it is difficult to judge whether this was because of some inherent characteristic or of the chance circumstances of their introduction. The latter seems more likely. Only two of the twelve types introduced more than twice failed to spread to at least one other cottage, while 15 of the 27 introductions even by the six prevalent types failed to spread altogether. It might be postulated that one of the characteristics of a streptococcus that makes it 'communicable' is a tendency to colonize the nose as well as the throat. There is some evidence for this, in that nasal carriage was somewhat more frequent both among children ill with, and among healthy carriers of, the six 'epidemic' types than among other carriers, but the difference was not great. Some types seemed to have consistent characteristics in their spread through the Village; *Type 6* had a notably high ratio of infections to illnesses, and *Type 9* seemed particularly apt to spread from incubating carriers.

The importance of population changes in the genesis of epidemics is implicit in the fact of type-specific immunity; on a smaller scale it was clearly seen leading to the long continuance of particular cottage outbreaks. The effect does not, however, seem to be a very large one and there were not the striking differences that one might have expected between the *'reception'* and *'permanent'* cottages. This may be because of the successive invasions with different streptococci. Or, it may be that, as Wannamaker (1954) suggested, carriers tend to become less dangerous soon after they acquire their infection, and that the population changes in the *reception* cottages were rarely frequent enough to introduce susceptibles at a time when the carriers were in a highly infective state. We have not been able to make a quantitative estimate of the diminution in infectivity of nasal carriers and, although it is certainly true that in most cases nasal carriers who spread infection had not been infected for more than 3–6 weeks, this may reflect the exhaustion of susceptible children in the cottage rather than exhaustion of the carrier's power to spread the infection.

Our results offer abundant confirmation of the importance attributed by Hamburger and his colleagues (Hamburger, Green & Hamburger, 1945*a*, *b*) to the nasal carrier in spreading streptococci—both in the initiation of a cottage outbreak and probably also in its late continuance. Although from the figures of Table 4, convalescent carriers seemed to spread infection more often than the incubating carriers, it has to be remembered that the period of carriage in convalescence is immensely greater than that in the incubation period; nasal carriers in the incubation stage must be, on a time basis, far the most infective.

Convalescent carriers seemed to produce their secondary cases most often about 2 weeks after their discharge from hospital. Sometimes this was because they were

discharged carrying streptococci only in the throat and became nasal carriers later. It was notable that even when spread was attributable to a convalescent carrier who carried large numbers of streptococci over a long period, there was commonly a cluster of cases occurring within a few days of one another, as though some particular action or state of the nasopharynx was required for spread to occur. There was no evidence of spread by foodstuffs, but on the small scale of our cottage outbreaks such might occur without its being detected.

In the earlier papers (Holmes & Williams, 1958*a*, *c*) we showed that, on the whole, the tonsillectomized children fared slightly better than children, of the same age and exposed in the same way, who had not had their tonsils removed. In studying the spread of infection a disadvantage of tonsillectomy is apparent, namely, the greater relative frequency of nasal carriage among the tonsillectomized children who become infected. The 'dangerous carriers', who spread infection to their cottage-mates, were significantly more often tonsillectomized than the general run of infected children.

For guidance on control measures we need particularly to recognize the relative importance of the various persons from whom infection may be spread and the place in which the spread takes place. In any study of the source of the infection in a particular individual there are two major difficulties: that we cannot hope to know all the infected carriers to whom he has been exposed; and that, even knowing his contacts, we have to rely on judgement when we specify one among them as being the person from whom he was infected. All our conclusions have to be read with these points in mind, and with the further proviso that a large proportion of the sources, particularly of the cottage introductions, could not be recognized. Despite these difficulties we feel that the analyses are worth presenting because no similar attempt at a quantitative assessment of a number of small outbreaks seems to have been attempted, and because we believe that the results have sufficient validity to justify advocacy of some particular control methods rather than others.

In this situation the school seemed to be of less importance than the cottage as a focus of infection. Although 16 % of cottage introductions were attributable to school contacts, the figure for all cases of infection was only 12 % because secondary cases were more common in the cottages than in the school rooms.

Within the cottage it was even more difficult to obtain evidence on the place of spread, but despite the fact that the bedding of the heavy nasal carriers, and of a good many other children, was heavily contaminated with the streptococci, we could obtain very little indication of spread of infection in bedrooms. It seemed, therefore, that the communal play-rooms and dining-rooms might be more important—an impression conforming with that gained in the U.S. Air Force (Wannamaker, 1954).

It appears unlikely that environmental control methods will reduce the incidence of streptococcal disease greatly. With the school a relatively unimportant source, air disinfection with ultra-violet irradiation in the classrooms could hardly have any great effect. In a recent trial in schools in Southall (*Report*, 1954) this procedure was shown to reduce the incidence of absence due to these illnesses, but in

these schools its apparent usefulness may well have been associated with the small risk of infection in the family.

Similarly, one would not expect any great effect from oiling or disinfection of bedclothes. For some time it was the custom at Barkingside (as it is even in the majority of hospitals) for blankets not to be removed from a bed when a child went to hospital with a sore throat, and in *reception* cottages it was not infrequent for another child to be put into the bed in the meantime. We have no full records on the subject, but we never gained the impression that a child became infected in this way.

It is clear that the routine sulphamezathine treatment of sore throats did not eliminate streptococci from the throat (Holmes & Williams, 1958*a*, Table 7) and that the convalescent carriers were potent in spreading infection. We estimate that at least 25 % of all the infections occurring in the Village would have been prevented if no children had become convalescent carriers, and this figure is certainly a minimum because the source of about half the infections was unknown and must often have been a convalescent carrier. In this connexion it is, however, interesting to note that Breese & Disney (1956) observed a secondary attack rate of over 20 % in families in which the index case was treated with penicillin.

From all the results reported in the literature (e.g. Denny, Wannamaker & Hahn, 1953) convalescent carriage could have been virtually eliminated by adequate penicillin treatment of the acute illness, and this is certainly supported by our observations on children with otitis who were treated with penicillin. Nevertheless, the sulphamezathine treatment allowed the children to develop some immunity, at least to the homologous serotype. Whether the children treated with penicillin developed any immunity cannot be judged from our analyses but from other work (e.g. Denny, Perry & Wannamaker, 1957) it is probable that they did not. In deciding on the appropriate management for a population such as that at Barkingside one has therefore to weigh the disadvantage of allowing convalescents to remain carriers and generate fresh cases of infection, against the disadvantage of treating the patients so that they do not carry in convalescence (and are probably also at less risk of developing complications), but are liable to become re-infected when they return to their cottage.

Some 16 % of the infections were attributed to persons in the incubation stage of an acute illness; this estimate is probably more accurate than that for convalescent carriers, but may still be an underestimate. The incubating carrier presents a more difficult problem in control, and in some circumstances might well justify a measure of quarantine on the contacts of a primary case, or their chemoprophylactic treatment.

SUMMARY

In a residential home for children, 367 cases of streptococcal illness were observed in a period of 30 months. The children lived in groups of about twelve in separate cottages. There were 194 occasions on which a streptococcus was thought to have been newly introduced into and produced illness in a cottage; on 132 of these 194 occasions there were no secondary cases of illness. The remaining 62 cottage introductions were followed by one or more secondary cases.

In 27 % of the 194 introductions, the primary case of illness seemed to have been infected from a healthy person in the cottage. In all, 30 % of introductions of a new streptococcus into a cottage could be attributed to recognized contacts with one or more known infected children.

The most important factor determining spread within the cottage seemed to be the carrier state of the primary case, spread following more often when the primary case had streptococci in the nose either on admission to hospital, or in convalescence.

There was no evidence that spread within cottage bedrooms was of great importance.

In about 35 % of the incidents with spread, the initial spread to secondary cases seemed to be from the incubation-stage carriage of the introducer; in 42 % it was from his or her convalescent carriage.

The carrier rate in the healthy cottage-contacts was generally higher in cottages experiencing clinical spread of infection than in those that had single-case introductions. There was a strong correlation between the carrier rate in the first week after an introduction and the final bacteriological attack rate, and a weaker correlation with the final clinical attack rate.

Continued spread of infection in a cottage was commonly due to the arrival of new children and was almost always associated with the presence of nasal carriers of streptococci.

The 194 cottage introductions could be grouped into sixty-three overlapping Village epidemics, each apparently deriving from a new importation of the particular type into the Village, although the evidence for this was often merely the absence of known infections within the previous few months. Only 13 % of the introductions resulted in more than 10 cases, and some 80 % had 5 or fewer. Introductions were more frequent in the cottages receiving children new to the homes than in those for the more permanent residents.

The principal factor found as determining the spread from the first cottage to others was the attack rate in the first cottage. Introductions in cottages for school-age children, and especially those in which a child attending the school in the Village grounds was the first to be attacked, also seemed to lead to spread more often than others.

The interval between successive Village introductions of one type did not appear to affect the extent of spread at the second; but the number of cases occurring in the first of two introductions had a notable effect: in no case did two successive introductions both result in a large number of cases of illness.

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