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AN OUTBREAK OF MILK-BORNE SCARLET FEVER AND TONSILLITIS IN DONCASTER

BY

R. WATSON, M.A., M.B., D.P.H.

Medical Officer of Health, Doncaster County Borough

Many outbreaks of tonsillitis and scarlet fever due to the drinking of infected milk have been reported, but the outbreak which occurred in Doncaster in December, 1936, had several points of interest which appear to make it worth while adding to the literature on the subject. The outbreak was a comparatively small one, extending over a period of not more than twelve days from December 9 to December 20 inclusive, and as the infected milk, amounting to about fifty gallons a day, all came from one farm and was distributed by one retailer unmixed with milk from other sources, investigation was made comparatively simple. The number of cases affected, however, is large enough to warrant certain conclusions being drawn.

Source of Infection

The first cases came to my notice about midday on December 11, and by the evening of that day fifteen cases of scarlet fever had been notified, all in the same part of the town. Suspicion fell on a certain milk supply, and arrangements were made for four samples of this milk to be taken for bacteriological examination that afternoon. Later in the day sufficient evidence had been collected to warrant a notice being issued under the Milk and Dairies Acts and Orders prohibiting the sale of this milk for human consumption for a period of twenty-four hours.

The farm from which the supply came was situated in the Doncaster rural district administrative area, and, together with Dr. Penman, the medical officer of health for the rural district, I visited this farm on the morning of the next day, December 12. Here the only thing to be discovered was that one of the milkers had a sore throat which, he stated, he had had "for about a week." During the morning a report was received to the effect that the four samples of milk taken the previous afternoon had been plate-cultured and had all proved to be negative for haemolytic streptococci; however, no animal inoculations had been carried out. It was accordingly considered that if the milker was removed from duty the milking utensils properly sterilized, and milking carried out under supervision, the milk could be released as from the morning of the next day, Sunday, December 13. As a precaution the retailer undertook to advise his customers to boil the milk, and this advice was also given by the local press and by house-to-house visits by the health visitors. This was done until it was possible to complete arrangements for the pasteurization of the whole supply. Pasteurization became effective as from midday on December 15, and the milk when not pasteurized was then used for other purposes and not distributed direct to consumers.

Bacteriological Investigations

In the meantime it had been found that the milker suspended from duty had a child who had been suffering from otorrhoea since December 2, and that he had been dressing the ear. On December 14 arrangements were made with Dr. Griffith of the Ministry of Health Laboratory for the typing of the haemolytic streptococci which had been found to be present in the discharge from this child's ear, and in swabs taken from the throats of the milker and several of the patients who had been admitted to the isolation hospital. On December 15 the cows forming the herd supplying the infected milk were examined by the chief veterinary officer of the West Riding County Council, Major D. S. Rabagliati, and group samples were submitted to Dr. Griffith for examination. The only clinical abnormality discovered in the herd was that one of the cows was suffering from an induration of one teat, the condition being diagnosed as a "crushed teat." It was stated to have been crushed by a tread about a fortnight previously. There was no external lesion.

Dr. Griffith was able to isolate *Streptococcus pyogenes*, Type II, from the child's ear, from the throat of the milker, from the throats of all the patients from whom swabs were submitted for examination, and from the milk secreted by the cow with the "crushed teat." This cow eventually developed an acute mastitis in the two forequarters of the udder. She was isolated for further observation by Dr. F. C. Minett of the Research Institute of the Royal Veterinary College, London, and he informs me that she continued to excrete *Streptococcus pyogenes*, Type II, for several weeks. The chain of evidence as to the source of the outbreak was therefore definitely established. It is of interest to note that a sample of the milk taken from bulk on December 14 failed to show the presence of haemolytic streptococci on plate culture, and that the retailer himself failed to show the presence of Type II organisms at this period, although his throat swab repeatedly gave heavy growths of haemolytic streptococci of a different type.

Age and Sex Distribution

In considering the effects of the outbreak it should be noted that the figures given in this account refer only to those cases occurring in the borough of Doncaster.

Only a few cases associated with the milk supply were notified in the rural district area, and these do not materially affect the total figures.

Very early in the outbreak it was noticed that many patients had only tonsillitis with no signs of a rash. In an attempt to obtain a complete picture of the effect of the mass infection I have recently completed an investigation of all the families known to be taking the milk during the period in which it was infected. The results of this inquiry are shown in the following tables and charts.

The milk was delivered by the retailer to 380 families in Doncaster, and of these 205 families (54 per cent.) were affected to a greater or less degree, the infection being fairly equally distributed over the whole area of supply. The 380 families contained 1,343 persons and were made up as follows: children aged 0 to 4 years, 62 ; children aged 5 to 14 years, 218 ; persons aged 15 years and over, 1,063. The number of persons attacked and the attack rates are shown in Table I.

TABLE I.—*The Number of Persons Attacked and the Attack Rates in Varying Age Groups*

Age Group (Years)	No. of Persons at Risk	Cases of Scarlet Fever	Cases of Sore Throat	Total Cases Affected
0–4	62	26 (41.9%)	8 (11.3%)	34 (53.2%)
5–14	218	53 (23.9%)	37 (16.9%)	90 (40.8%)
15 and over	1,063	56 (5.3%)	184 (17.4%)	240 (22.7%)
Totals..	1,343	135 (10.0%)	229 (17.0%)	364 (27.1%)

TABLE II.—*Cases of Scarlet Fever and Sore Throat due to Infected Milk ; Age and Sex Distribution (December, 1936)*

Age Group (Years)	Scarlet Fever Cases			Sore Throat Cases			Total Cases Attacked		
	Males	Females	Total	Males	Females	Total	Males	Females	Total
0–4	16 (26.2%)	10 (13.5%)	26 (19.2%)	4 (4.2%)	4 (3.0%)	8 (3.5%)	20 (12.7%)	14 (6.7%)	34 (9.3%)
5–9	13 (21.3%)	20 (27.0%)	33 (24.4%)	8 (8.4%)	7 (5.2%)	15 (6.5%)	21 (13.4%)	27 (13.0%)	48 (13.3%)
10–14	10 (16.4%)	10 (13.5%)	20 (14.9%)	11 (11.4%)	11 (8.4%)	22 (9.6%)	21 (13.4%)	21 (10.3%)	42 (11.5%)
15–19	3 (5.0%)	9 (12.1%)	12 (8.9%)	11 (11.4%)	7 (5.2%)	18 (7.9%)	14 (8.9%)	16 (7.7%)	30 (8.2%)
20 and over	19 (31.1%)	25 (33.9%)	44 (32.6%)	62 (64.6%)	104 (78.2%)	166 (72.5%)	81 (51.6%)	129 (62.3%)	210 (57.7%)
Totals ..	61 (100%)	74 (100%)	135 (100%)	96 (100%)	133 (100%)	229 (100%)	157 (100%)	207 (100%)	364 (100%)

TABLE III.—*Cases of Scarlet Fever in Doncaster other than those due to Infected Milk (1935 and 1936)*

Age Group (Years)	Male	Female	Total
0–4	36 (21.8%)	27 (13.4%)	63 (17.2%)
5–9	61 (36.9%)	84 (41.6%)	145 (39.5%)
10–14	49 (29.7%)	54 (24.7%)	99 (27.0%)
15–19	5 (3.0%)	7 (3.5%)	12 (3.3%)
20 and over	14 (8.6%)	34 (16.8%)	48 (13.0%)
Totals..	165 (100%)	202 (100%)	367 (100%)

These figures are interesting as they show how, with presumably the same infecting organism, the incidence of a rash symptomatic of scarlet fever appears to vary according to the age of the person attacked. Having regard to the number at risk in the different age groups, they also seem to indicate how much more children are affected than adults, especially children under 5 years of age. They also bear out findings collected by Professor Picken (1936) to the effect that in a milk outbreak adults may form a large proportion of the total cases suffering from clinical scarlet fever. The distribution of cases, of both scarlet fever and sore throat, according to age and sex is shown in Table II. For comparison with the scarlet fever group the distribution of these cases for the years 1935 and 1936 (excluding those cases associated with the milk outbreak) is appended (Table III). These latter figures should form a fair basis of comparison, as they do not include any cases which were associated with a milk-borne epidemic.

So far as cases of clinical scarlet fever were concerned in the milk outbreak the percentage of the total cases occurring among adult males was nearly four times the normal expectation. Among adult females the percentage was about twice the normal. Children of school age in proportion showed a lower percentage incidence than the normal rates would lead one to expect. The effect of the outbreak must, however, be judged on the total number of cases of tonsillitis and scarlet fever, for which, unfortunately, no comparison with the normal expectation is possible. From the figures in Table II it will be seen that among males 51.6 per cent. of the total cases of scarlet fever and tonsillitis were adults, and for the adult females this figure was 62.3 per cent. The only comparable figures are those respecting the Chelmsford outbreak, to which the Doncaster figures approximate fairly closely. It is of interest to know that both the Chelmsford and the Doncaster outbreaks were caused by a Type II haemolytic streptococcal infection of milk. No evidence has been forthcoming to account for the high percentage of the adults attacked ; possibly this might be due to a previous absence of Type II infection in the areas.

Course of Outbreak

The accompanying charts, which show the day-to-day occurrence of cases, whether of tonsillitis or scarlet fever, according to the ascertained date of onset of the disease, are of some interest in showing the course of the outbreak and the effects of action taken to bring it to an end. Chart 1 shows the number of " milk " cases, of both scarlet fever and tonsillitis, the " seasonal " cases of scarlet fever being superimposed. From the chart it would appear that the milk outbreak can be limited between December 9 and December 20. The two cases occurring on December 5 and 6 respectively might not be associated with the milk outbreak, and the two cases appearing on December 23 and 27 respectively were, so far as can be ascertained, secondary cases. Other ascertained secondary cases were noted as follows: December 13, one case ; December 14, one case ; December 17, three cases ; December 18, three cases ; and December 19, two cases. There may have been more secondary cases, but when definite proof of this secondary character could not be established the cases have been counted as original milk infections.

The omission of these secondary cases makes it clear that there were no new cases from mass infection of the milk after December 17, and this would correspond with the fact that infected milk ceased to reach consumers after midday on December 15. The odd cases with an

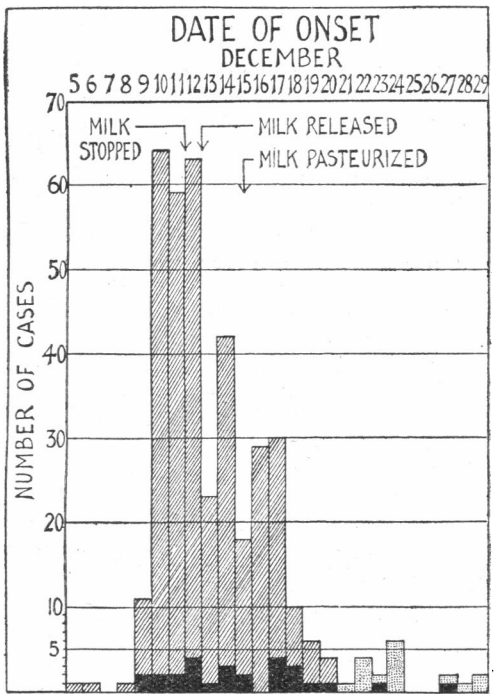

CHART 1.—The shaded area represents those cases of scarlet fever and tonsillitis due to infected milk. Superimposed is a black area representing seasonal cases of scarlet fever only, which arose at the same time as the milk-borne outbreak but were not due to infected milk; similar cases over and above the few milk-borne cases which appeared after December 20 are shown by the dotted area.

onset later than December 17 can be explained either by a prolonged incubation period, a delay in consuming milk obtained on December 15, or possible non-proven secondary cases. The normal incubation period is thus indicated as being from two to three days, and so the milk was apparently first infected not earlier than December 6. It will be remembered that the milk was stopped for a period of twenty-four hours on the evening of December 11, so that no infected milk reached consumers on December 12. This would account for the marked fall in the number of new cases on December 15, but does not explain the even more marked reduction on December 13. This chart appears to show that, assuming an incubation period of three days, a gross infection of the milk took place on December 7, 8, and 9, and for some reason the infection was reduced on December 10, to reappear on December 11, despite the negative bacteriological findings in the milk on that date. This rise, due to apparent recurrence of infection, was partially checked by the withholding of the milk on December 12, and finally stopped by the pasteurization of all the milk on December 15. Boiling of the milk was never adopted by consumers generally, and of those who did boil it very few did so before December 14, so that this appears to have had very little effect upon the course of the outbreak.

I understand that there is an increasing belief that milk outbreaks such as this are caused through cows themselves becoming infected by some human agency. In this instance there is no doubt that the cases dating from December 16 were all due to milk which had been infected by the cow found to have an infected teat from which *Streptococci pyogenes* Type II were isolated on December 15, as the infected milker could not have directly infected the milk after December 11. The infection of the cow's teat was clearly at an early stage when first discovered, but Dr. Minett has reason for believing that this cow was

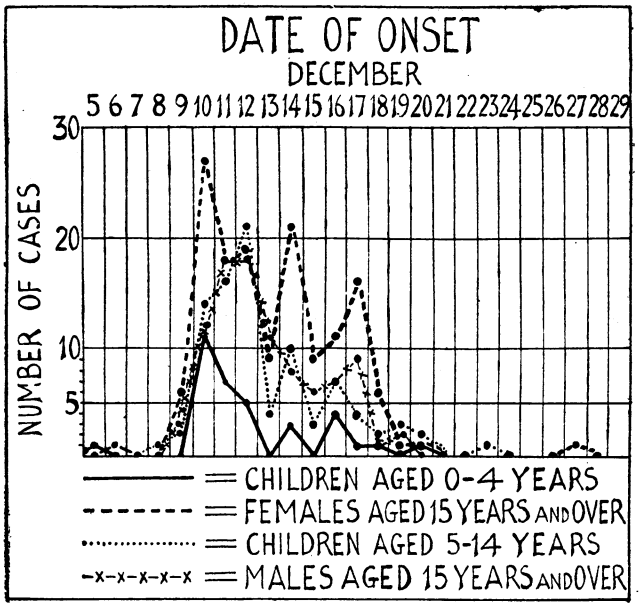

CHART 2.—This shows the day-to-day occurrence of cases of scarlet fever and tonsillitis associated with infected milk in different age groups and sexes.

infected by December 6 or 7, and possibly even a few days earlier than this, and so was responsible for the whole outbreak. I understand that recent work carried out at the Research Institute of the Royal Veterinary College would indicate that the possibility is very unlikely of the first part of the outbreak being due to direct infection of the milk by the milker, who probably began with a sore throat about December 5, but nevertheless this possibility would appear to fit in with the otherwise unexplained fall in the number of cases on December 13. It should also be remembered that the milker was doing all the necessary home treatment for his child's otorrhoea as from December 2, and there thus appears to be yet another possible method of spread of infection both to the cow and possibly a gross infection to the milk by means of the milker's hands.

Chart 2 shows the cases split up into the following groups: children aged 0 to 4 years, children aged 5 to 14 years, males aged 15 years and over, and females aged 15 years and over. It is of interest in that it indicates the high proportion of the children under 5 attacked in the early stages of the outbreak, and it shows, too, that the children in this age group and the adult females formed the majority of those who were first attacked. It will be noted that the peak numbers for school children and adult males come two days later than do the peak numbers for the other groups. As compared with adult females, adult males escaped fairly lightly in the later stages of the outbreak. No explanation has been found for these variations.

Clinical Manifestations

I do not intend to deal at length with the clinical manifestations seen in the outbreak, but a few words must be said on this subject. The disease was usually characterized by a very acute onset leading to rapid prostration. The first symptom was commonly described as a burning

or shooting pain in the throat, and many patients were almost able to state the hour at which this symptom occurred. Some two hours afterwards they had difficulty in getting undressed when they realized that it was imperative that they should go to bed. There were, of course, several cases of a very mild nature, but the majority of patients were suffering sufficiently severely to necessitate their staying in bed for several days.

The throat conditions appeared to vary, but could be roughly divided into the three main types described by Dr. Camps in the Chelmsford cases (1935). The cases of scarlet fever were noted as being of a much more serious type than those normally seen. Rashes, when present, tended to be marked and to be of a rather coarse nature; desquamation followed the normal course. In the more severe cases pain was a marked feature, and the tonsillar glands were easily palpable. An interesting point is that one dog (an Airedale) appears to have been affected, though the nature of the infection was not confirmed bacteriologically. It became ill a day before any human case occurred in that particular household, became rapidly collapsed, with marked swelling and obvious pain in the region of the fauces, and was described as being "paralysed" for a week.

Complications

The chief complications noted were adenitis, otitis media, quinsy, and polyarthritic symptoms. One case of nephritis, one of acute appendicitis, and one with a diffuse herpetiform eruption were associated with initial throat infections. These complications made their appearance at intervals varying from three days to three weeks after the onset of the illness, but the majority were seen at the end of the first week. The arthritic symptoms tended to appear a little later than this, and were very persistent. They were not the transient joint pains common with streptococcal throat infections; many patients were still complaining of these arthritic pains three months after the outbreak had ended.

Although the figures are too small to be of much value, it is interesting to compare the occurrence of complications among those cases nursed in hospital and those nursed at home, as shown in Table IV.

From these figures it would appear that, except in the 0 to 4 years age group, hospital treatment does not appear to have had any beneficial effect upon the reduction of complications. All cases treated in hospital received streptococcal antitoxin (scarlatina) serum, but only two cases nursed at home received any serum. It might be argued that only the worst cases were admitted to hospital, but this was by no means the case, as in the early days of the outbreak the presence or absence of a rash was often the deciding factor in the request for removal to hospital, and many of the cases showing no signs of a rash were acutely ill. Nor is it believed that the assessment of complications was very much more accurate among cases nursed in hospital. As will be mentioned later, the possible cause of the comparatively large numbers of complications among the hospital patients was that cross-infection occurred owing to the admission of cases of scarlet fever associated with a coincident "seasonal" outbreak of the disease.

For all cases the percentages of complications were as follows: in the 0 to 4 age group, 38.2 per cent.; in the 5 to 14 age group, 25.5 per cent.; in patients aged 15 and over, 25.4 per cent. This gave a total incidence of 26.6 per cent. These percentages are high and the results serious. Apart from many cases suffering from resultant debility there is little doubt that a late spread of infection from the cases of adenitis and otorrhoea resulted in an increased number of cases of scarlet fever in the district during the three months succeeding the outbreak, and has been responsible for an outbreak of tonsillitis with associated scarlet fever in the Doncaster Royal Infirmary which necessitated the closing of one unit for a short period.

Two deaths have definitely been associated with the epidemic—one in a child aged 2 years suffering from severe scarlet fever, who died within twenty-four hours of the onset of the disease, and one in a male aged 33, who developed adenitis following a severe attack of tonsillitis. One other death was that of a woman aged 38, who developed clinical scarlet fever on December 6 and cannot therefore be definitely associated with the outbreak, although she was taking the milk which later proved to be infected. In all cases death appeared to be due to toxaemia. Unfortunately post-mortem examinations were not possible, nor were swabs taken at the time of the original throat infections for bacteriological examination. It is worth while noting that no cases of puerperal fever occurred during this period or the succeeding months, and that only one case of erysipelas was definitely associated with the outbreak.

TABLE IV.—*The Incidence of Complications in Cases Treated in Hospital as Compared with those Treated at Home*

Age Group (Years)	Adenitis	Otorrhoea	Quinsy	Arthritic Symptoms	Total Cases	Total Complications	Percentage Incidence of Complications
Cases Treated in Hospital							
0–4	—	1 (5.0%)	—	2 (10.0%)	20	3	15.0
5–15	6 (16.2%)	5 (13.5%)	—	4 (10.8%)	37	15	40.5
15 and over	1 (2.2%)	1 (2.2%)	1 (2.2%)	7 (15.5%)	45	10	20.2
Totals ..	7 (6.8%)	7 (6.8%)	1 (0.9%)	13 (12.7%)	102	28	23.8
Cases Treated at Home							
0–4	5 (35.7%)	5 (35.7%)	—	—	14	10	71.3
5–14	4 (7.5%)	1 (1.9%)	2 (3.8%)	1 (1.9%)	53	8	15.1
15 and over	18 (9.2%)	—	11 (5.6%)	22 (11.3%)	195	51	26.2
Totals ..	27 (10.3%)	6 (2.3%)	13 (4.9%)	23 (8.8%)	262	69	26.4

Administrative Problems

From the administrative point of view several problems were raised. Once the outbreak had begun it was quickly realized by the general practitioners that the cases of tonsillitis and scarlet fever were of the same origin, and the question arose as to whether all cases should be

notified as scarlet fever. Several cases exhibiting no exanthem were in fact notified. This accounts for the fact that the figures in this report do not exactly correspond with those in a preliminary annotation published in the *British Medical Journal* (1937, **1**, 26). Rightly or wrongly it was decided to ask for notification only in cases of clinical scarlet fever. In view of the fact that direct case-to-case spread was very limited once the outbreak had begun, the notification of all cases of tonsillitis irrespective as to whether they were associated with the milk infection would have served no useful purpose. It would have been only of limited statistical value unless every case could have been properly investigated by the sanitary inspectors or health visitors as is done in all cases of notifiable infectious disease, and this would have placed an unbearable strain on these staffs whose services at that time were much more usefully applied in other directions.

With regard to isolation hospital accommodation the question of the presence or absence of a rash did not apply. It is true that the majority of cases admitted to hospital were cases of clinical scarlet fever, but cases of tonsillitis were admitted where nursing facilities at home were inadequate. One patient with tonsillitis who developed an acute appendicitis was admitted to hospital for operative treatment, as was also a case of diffuse herpetiform eruption clearly associated with a recent throat infection. One child who had been nursed at home with scarlet fever and had developed otorrhoea was removed to hospital as his mother was expecting to be confined. When home facilities were satisfactory, therefore, cases were nursed at home, but, nevertheless, between December 5 and December 29, 131 patients were admitted to hospital, 102 of these being cases associated with the milk outbreak. Luckily the hospital was nearly empty at the beginning of December, but even then the patients in the sanatorium section had to be given "Christmas leave" to create extended temporary accommodation for scarlet fever cases.

Patients were retained in hospital for three weeks unless complications had ensued. No "return" cases have come to my notice. Some patients developed secondary attacks of tonsillitis in their third week, and this was probably due to cross-infection from "seasonal" cases. Some of the "complications" may have been due to this cause. At the very beginning of the outbreak it had been realized that the sudden spate of cases would probably be infected by an organism of one serological type, and they were accordingly separated from the few cases of scarlet fever already in hospital. Unfortunately, as will be seen from Chart 1, what was apparently a "seasonal" outbreak of scarlet fever began about the same time as the milk outbreak, and with the rush of cases it proved impossible to segregate cases strictly according to their probable source of infection, and cross-infection occurred. These "seasonal" cases were mainly associated with haemolytic streptococci Type IV, though there was one case of Type I infection which ended fatally. The difficulties associated with cross-infection were, however, most noticeable during the three months succeeding the milk outbreak, when, as has been stated, there was an increase over the usual expected number of scarlet fever cases.

During the period of the outbreak typing of haemolytic streptococci from patients was carried out by Dr. Griffith at the Ministry of Health Laboratory. Following the outbreak, through the kindness of Dr. Camps of Chelmsford, all new scarlet fever admissions to hospital have been typed. The point of interest is that, despite the heavy Type II infection in the milk outbreak, this type completely disappeared during the six weeks following the end of the outbreak and was replaced by a bewildering number of different types, which made segregation in hospital a matter of great difficulty. About the middle of February Type II organisms again began to appear, only to disappear early in March.

The other main administrative actions taken early in the outbreak were to inspect the schools in the district chiefly affected, to exclude all children showing signs of sore throat and to recommend their parents to obtain medical advice, and to circularize the doctors in the district regarding the epidemic. This circular letter pointed out that few immediate secondary cases were to be expected, but that danger would arise from cases of otorrhoea, etc., resulting from the infection. The danger was made apparent early in January when a few cases of mastoiditis and adenitis found their way to the wards of the Royal Infirmary and precipitated the throat infection there already referred to. The circular also advised the following quarantine periods:

Cases of scarlet fever and severe tonsillitis—three weeks from the date of onset.

Cases of mild tonsillitis—one week after the subsidence of symptoms.

Cases with throats positive for haemolytic streptococci—one week if no clinical signs of infection appeared. (A good number of these cases were found as interest grew, and caused considerable administrative difficulties.)

Contacts, especially school children, were advised quarantine for one week unless negative throat swabs had been obtained.

The midwives were also circularized and advised to take special precautions for a considerable period, and the necessity for wearing adequate masks was re-emphasized. All visiting was stopped at the Municipal Maternity Home. Several administrative problems of a minor nature occurred during the course of the outbreak, and with these, as with the greater problems, much help was obtained from the Ministry of Health, and especially from a personal visit by the late Dr. Vernon Shaw, who visited Doncaster on December 16.

Summary and Conclusions

1. An outbreak of scarlet fever and tonsillitis due to infection of a milk supply with *Streptococcus pyogenes* Type II, and the administrative action taken to minimize the effects of the outbreak are described and discussed.

2. The distribution of cases shows a heavy attack rate among pre-school children, and also that adults form a larger proportion than normal of the total cases.

3. The primary infection of the milk appears to have been by the cow becoming infected, but on the evidence obtainable locally the possibility of direct contamination of the milk by a milker cannot be entirely disregarded.

4. The complications subsequent upon infection are enumerated, and from this point of view, apart from children under 5 years of age, hospital treatment appears to have been of doubtful value.

5. The only adequate method of controlling an outbreak is by stopping the milk supply or by having it efficiently pasteurized.

6. To avoid a sense of false security being given in the search for haemolytic streptococci in milk small group samples must be taken.

My thanks are due to Dr. F. Griffith, Dr. F. C. Minett, and Dr. F. E. Camps for the help and for advice they have given to me in the preparation of this report.

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