

## **A large outbreak of streptococcal pyoderma in a military training establishment**

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### SUMMARY

An outbreak of streptococcal pyoderma in a military institution into which fresh susceptible recruits were regularly introduced involved more than 1300 persons over an eighteen-month period. Two M types were responsible for the great majority of the cases and an attack by one conferred immunity to that strain but not to the other. Lesions varied from trivial to disabling. Epidemiological studies indicated that contact – direct or indirect – through such things as gymnasium equipment and room dust was the means of transmission. The outbreak was eventually controlled by vigorous case finding, thorough treatment of cases and the strict application of hygienic principles to prevent spread. There were no late complications and throat streptococci were not involved. It is possible to control such an outbreak without isolation or the making of any significant concessions in the training programme even when large numbers of persons are living and working at close quarters.

### INTRODUCTION

Between July 1979 and December 1980 some 1300 cases of streptococcal pyoderma were bacteriologically confirmed at a large military training establishment in south-western England. The epidemic provided an opportunity to study some facets in the dissemination and control of the infection under fairly well-defined environmental and social circumstances.

Streptococcal pyoderma on a large scale is considered particularly prevalent in hot and humid conditions (Taplin *et al.* 1973). Regular outbreaks occur in Trinidad (Poon-King *et al.* 1973) and amongst school children in Alabama (Dillon *et al.* 1967) during the summer months; both have been associated with nephri-

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togenic organisms. On the other hand the disease is endemic in the Red Lake Indian reservation in Minnesota, USA, where cooler climatic conditions at times prevail (Anthony, Perlman & Wannamaker, 1967). During World War II in certain combat zones infected skin lesions accounted for up to 80 % of all soldiers attending sick call (Pillsbury & Livingood, 1968), and more recently a study in the Mekong delta region of Vietnam showed a significant incidence amongst front-line troops who spent much time on patrol knee deep in flooded rice fields (Allen, Taplin & Twigg, 1971). Fortunately the incidence of nephritis has been low in these groups.

Our outbreak occurred in a recruit training camp in which regular inspections and observations could be made throughout a regular programme of activity.

### *Background*

The infection, known to the subjects as 'Woodbury rash' after a common used for training exercises, is well recognized in the service. Clinically cases were generally mild and more often than not self-treatment was successful. Occasional severe cases required more active therapy and even hospital admission for a day or two. The few swabs that were taken showed a succession of streptococcal types, one generally dominant for a time and then replaced after a year or so. The potential significance of the situation was brought to the fore when a recruit collapsed and became anuric after a 30-mile march. He was found to have a rash, from which streptococci were isolated, and a high anti-streptococcal antibody titre. Immediate investigation revealed that an epidemic of pyoderma of substantial proportions was in progress.

### *The environment*

The camp is modern and consists of permanent buildings with all facilities. There is a permanent staff of around 900 and at any one time up to the same number of recruits in training. The majority of recruits are between 16 and 18 years old and have come straight from home or school. There are intakes of about 50 approximately every two weeks, each group making up a troop. Smaller groups of experienced soldiers drawn from any of the services attend shorter training courses. Accommodation is in barracks with 6 to 8 people to a room. Rooms are light and airy and beds are separated by at least 5 feet. There are three showers and one bath to approximately 28 men. Troops move from barrack to barrack during their stay and, though members of one troop tend to remain together, there is ample opportunity for mixing at meal times and during sporting activities. In addition movement of recruits between troops occurs when a recruit is 'back-trooped' (dropped back to a more junior troop) due to illness or failure to progress satisfactorily.

The course, which lasts 30 weeks, is physically very demanding. The gymnasium and swimming pool are modern, but such is the demand that one troop will follow another in their use all day long. A variety of obstacle and endurance courses, and day and night exercises on open heathland provide further opportunities for skin trauma to occur, resulting in sores caused by such things as chafing webbing equipment and multiple gorse punctures.

## METHODS

The clinical notes on each case were recorded on a specially designed chart. Selected data were subsequently transferred to the computer for correlation analysis.

Swabs, which were pre-moistened in phosphate-buffered saline, were either sent to the laboratory within a few hours of being taken or placed in Stuart's transport medium if they were to be kept overnight. Standard methods were used to identify and type isolates. After identification of haemolytic streptococci as members of the serological group A, the strains were typed at the Streptococcus Reference Unit, Central Public Health Laboratory at Colindale, using sera prepared against the T and M proteins and  $\alpha$  lipoproteinases (Opacity Factor OF). The serological studies were also performed at Colindale.

Environmental swabs or materials were placed in serum broth containing crystal violet 1 mg/l, incubated overnight and plated on an agar base supplemented with blood (3% v/v) and crystal violet. Direct plating was occasionally used.

## RESULTS

*Clinical*

Any form of skin lesion was likely to become infected. The commonest presentations were the following.

(a) Woodbury rash; this starts as multiple punctures, scratches or cuts generally of the lower limbs from contact with gorse, heather, bracken or hawthorn and develops into pustules which become ecthymatous or impetigenous.

(b) Rope burns on the thighs, backs of the knees, lower legs and on the palms of the hands.

(c) Grazed knees, elbows and shins.

(d) Blistered toes and heels.

(e) Webbing friction sores.

The lesions appeared as ulcers with conspicuously red borders which developed a purple tinge as they matured. The exudate tended to dry and form large impetigenous crusts. Cellulitis, abscess formation, lymphangitis and lymphadenitis were not uncommon, and bursitis occasionally complicated infection near joints. The disease also presented as secondary infection of athlete's foot, BCG and yellow fever vaccination sites, tattoos, insect bites, acne, otitis externa, and also as balanitis.

The skin infection in the majority of cases produced only minimal incapacity. Recruits were able to continue training while undergoing treatment, particularly in the early weeks. However, even then certain restrictions were imposed, which included the prevention of rope climbing, swimming, running, or the wearing of webbing.

Complications usually arose in the later weeks of training. At that time the level of physical activity was much higher and more time was spent on field exercises. The opportunities for skin trauma and secondary infection were correspondingly much greater. Around 10% of all cases of streptococcal skin infection caused

complications necessitating admission to the camp sick quarters. The length of stay varied from 2 to 10 days, with an average length of stay of 3 days. However, daily dressings thereafter, and a period of rehabilitation often delayed the return to normal training.

It is estimated that over the eighteen-month period around 200 man/weeks of training were lost. If the figures for 'back-trooping' and 'opt-out' (voluntary retirement from the course) are considered, it is probable that the loss of effective training time was even greater.

No association with streptococcal throat infection and tonsillitis was noted, and there were no cases of nephritis. All cases of heat exhaustion and dehydration were carefully recorded and investigated: there was no direct association with streptococcal infection.

### *The outbreak*

Over the first five weeks of investigation in June and July 1979 there were 98 cases from which streptococci were isolated, giving an approximate incidence of 20% overall, but varying from less than 5% to around 50% in the 12 troops in camp over that period. When questioned near the end of their training most recruits claimed to have had the infection at some time during their course. Physical inspections were carried out and all cases identified and treated. At the beginning of the three-week leave period (August–September 1979) instructions were issued that if lesions were present, recruits were to contact their home doctors for treatment. This period is not included in the summary as the incidence was not accurately determined by week of onset. Thereafter the course of the outbreak as reflected in the isolations made is presented in Fig. 1.

At the start of the next sixteen-week period (September–December 1979), cases were first diagnosed towards the end of the second week and numbers increased steadily such that by week 9, 30–60 new cases were presenting each week, usually distributed as one to three cases in each of the 13 to 14 troops in the camp. Occasionally as many as six or seven from one troop might present in one week but this was unusual.

Streptococcal isolation rates varied from one troop to another – between 25% and 40%. In all, 346 isolations were made. It was apparent that many mild infections were self-treated and not reported and the true incidence may well have been twice the isolation incidence.

Few reported more than one attack. New intakes first developed lesions in the third or fourth week of training.

In spite of advice advocating treatment at home, a number of recruits arrived back at camp after the second leave period still with active lesions and during the next few weeks the peak incidence of the outbreak was reached (January–March 1980). Despite one troop providing 16 new cases in a single week, the usual pattern of one or two cases each from a number of different troops was the general rule. During the 12-week term 445 new isolations were made.

At the start of the next term (April–August 1980) there were, once again, many residual cases. However, measures for containment, which are detailed later, were rigorously applied and a sudden drop in the number of cases found occurred early

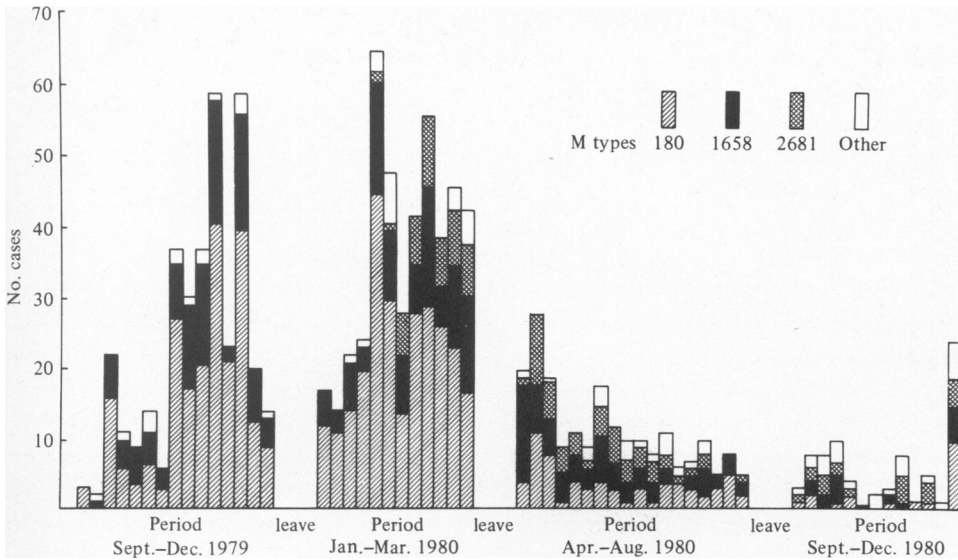


Fig. 1. Isolations of streptococci by type over the four periods of study.

on and the incidence further declined slowly over the rest of the period. During the 18-week term only 206 isolations were made.

In the final period under review the pattern was one of low endemicity even though the surveillance was of the same order as in previous terms. The rise during the last week was due largely to a high incidence in one particular troop who had been away from camp and were inspected just prior to completing the course.

Only six of the 1300 isolates have come from the permanent staff of the camp – all others were from the recruits.

### *Bacteriology*

In the first few months of the outbreak 99% of all streptococci isolated from the skin lesions belonged to one or other of two M types – (a) T 5/27/44, M provisional type 180, OF + ve (PT 180) and (b) T 11, M provisional type 1658, OF + ve (PT 1658). The former was first recognized in the camp in 1975 and the latter in 1977, both sporadically. Before then a variety of types were found amongst the few strains isolated. The strains were readily distinguished by their distinctive colonial appearances – colonies of PT 180 looked like applied disks with depressed centres whereas PT 1658 had low convex colonies. Only one type was found on any patient at one time. About 15% of those infected developed a second infection due to the other type from that causing the first. There were no cases of two separate incidents of infection being caused by the same organism in the same patient. Throughout the epidemic the relative proportions of the two strains have fluctuated little with an approximate 70% to 30% dominance of PT 180 to PT 1658. Fig. 1 presents the data in detail. About half-way through the outbreak a third

Table 1. *Streptococcal serology in recruits at the beginning and end of training*

	Percentage positive			
	No. sera	ASO	ADB	AHT
Recruits on entry	48	25	12	50
Recruits at end of training	58	55	60	75

ASO = Antistreptolysin O, positive titre  $\geq 200$ ; ADB = AntiDNAase B, positive titre  $\geq 400$ ; AHT = Antihyaluronidase, positive titre  $\geq 128$ .

strain, T14, M80, OF—ve (PT 2681) appeared. There is circumstantial evidence that it had been introduced from another military establishment. At first the organism was confined to one or two troops, but it gradually became more widespread and finally accounted for 25% of all strains isolated. None of the strains showed any general tendency to cluster by troop or, with occasional exceptions, by room.

The epidemic streptococci were rarely isolated except from active skin lesions. Even in patients they were obtained only once from the nasopharynx, twice from normal skin and twice from the perineum. They were not associated with respiratory disease. A variety of streptococci were isolated from the nasopharynx of newly arrived recruits, but none was of the epidemic types. In the later stages of the epidemic 9% of lesions were associated with other types whose origin is likewise not known.

Following rigorous therapy the organisms disappeared rapidly, but in untreated or partially treated cases they could be recovered from the lesions for some weeks.

*Staphylococcus aureus*, almost all resistant to penicillin, was associated with 80% of the lesions from which streptococci were grown. Many different phage types were demonstrated and there was no evidence of cross-infection with these organisms at either room or troop level. Where tested, staphylococci of the same phage type could be isolated from both the skin lesions and the nose of the patient.

### *Environment*

Epidemic streptococci were readily isolated from gymnasium mats, climbing ropes, changing room floors and benches, bedding of infected patients and room dust. They were not found in the swimming pool or in the ablution areas.

### *Serology*

One troop of men was bled within a few days of arrival, and their sera were examined for anti-streptolysin O (ASO), anti-DNA-ase B (ADB) and anti-hyaluronidase (AHT) antibodies. Sera from three other troops totalling 58 men, bled within a fortnight of the end of their courses, were similarly tested. The results are presented in Table 1 using the generally accepted criteria for minimal levels compatible with recent infections; 200 for ASO, 400 for ADB and 128 for AHT. There is some serological evidence of infection and the ADB is more regularly raised than the others.

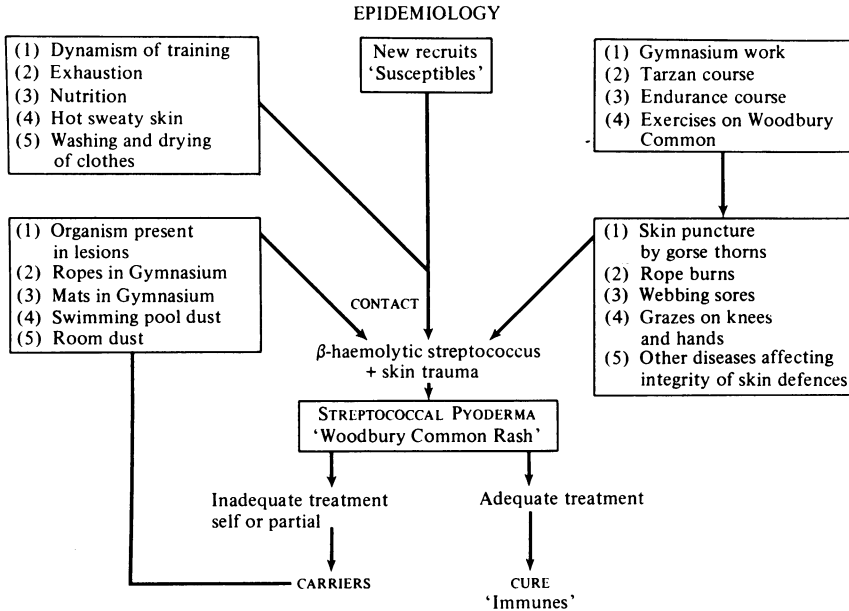


Fig. 2. Features considered to be of possible importance in the epidemiology of the outbreak.

*Epidemiology*

The sources of the organisms are not known. Both had been present in the camp for a few years before the investigation began. Carriage in the nose and throat, or on the skin or perineum was very rare amongst patients or contacts. In partially treated cases, however, long-term carriage of up to several weeks duration was the rule. It seemed therefore that the most likely means whereby the organisms were maintained in the camp was in the substance of active lesions.

Fig. 2 summarizes those factors considered to be of importance in the epidemiology of the outbreak. All infections were preceded by a breach in the skin. The boxes on the right indicate the activities leading to skin trauma and those on the left features associated or possibly associated with the maintenance and transmission of the infection. The organisms were isolated from the gymnasium equipment and facilities, and inasmuch as one troop tended to follow another in rapid succession, transmission by these routes was probably widespread.

Streptococci were sought in ponds and on vegetation in training areas (which local legend considers to be of importance), but none was found even when troops known to be infected were followed up immediately.

To determine if damp, hot skin which had been unwashed for 48 h might carry the organism while normal skin did not, swabs were taken from areas on the thighs without lesions of a heavily infected troop during the course of a three-day-and-night exercise in the field. No isolation was made.

Search was made for clustering of cases in rooms by those sharing quarters. While dominance of one of the two main types was occasionally demonstrable there seemed to be no general clustering of cases either by room or in time. Airborne transmission seems unlikely to be the major means of dissemination. The pattern of development of the epidemic was slow and unexplosive and the environmental findings suggest contact as the usual route. Over a period selected clinical, laboratory and other data for each case were placed on computer and an attempt made to associate various features such as age, serological susceptibility, stage of training where infection occurred, trauma, personal hygiene, stress (physical and mental), diet, temperature and humidity. Significant correlations were hard to detect, perhaps in part due to the fact that the social treatment, environment, age and ambition of virtually all the men involved were so uniform.

#### *Treatment and control*

All the cases were treated initially with Triplopen and thereafter with one injection of benzathine penicillin and oral flucloxacillin for 5–10 days. A local antiseptic (chlorhexidine, peroxide or povidone iodine) was used to clean the lesions and a dressing applied if appropriate. Follow-up swabs were not generally taken after the reliability of the regime had been proved.

Control was necessarily based upon the minimum disruption of courses. The training requirement was absolute and infected recruits could not be separated from the rest. Active case finding was established; 'freedom from infection' inspections were carried out where possible by the medical staff and by physical training instructors. Self-inspection was encouraged. In the great majority of cases the course could be continued during the period of treatment.

Regular health education lectures included information on the nature, recognition and control of the infection and the importance of proper treatment. One person in each room was issued with a povidone iodine spray (Disadine) and at the end of each day any lesions on any person in that room received a generous coating of antiseptic powder. Long trousers were used for gymnasium work in which transmission seemed likely to occur, i.e. rope-climbing and mat exercises. Finally the use of antiseptic soap was encouraged, though it was not generally adopted. It was recognized that, while more extensive ablution and clothes drying facilities were desirable, control could be achieved with existing facilities.

#### *Follow-up*

Twelve months after the end of the survey a further assessment was made over a four-month period. The total incidence had dropped to 1 to 2 new cases per day and it was felt that the disease was reasonably well controlled. However, analysis showed a peak distribution in the later weeks of the course with the following calculated incidence: weeks 1 to 2 zero incidence, weeks 3 to 15 two recruits per thousand, weeks 16 to 24 three per thousand, weeks 25 to 28 52 per thousand and weeks 29 to 30 57 per thousand affected. There was only a weak association with living under field conditions but a much stronger relationship with heavy physical activity such as endurance courses and the likelihood of sustaining minor skin trauma.



In December 1980 a new strain of Group A streptococcus T 11, M 11, OF +ve was isolated for the first time and in March 1981 was found in 90% of cases. Provisional type PT 1658 has apparently disappeared altogether though PT 180 is still seen from time to time.

#### DISCUSSION

Streptococcal skin sepsis seems to have become resurgent in recent years. Cruickshank (1953) suggested that the bullous or staphylococcal type of impetigo was far more frequently encountered than the non-bullous or streptococcal type. However, the Red Lake (Anthony, Perlman & Wannamaker, 1967) and Trinidad (Dillon *et al.* 1967) outbreaks drew attention to the continued significance of streptococci despite their sensitivity to most commonly used antibiotics. Outbreaks have been reported from cadet barracks (Leonard, 1967), detention centres (Colling *et al.* 1980), amongst naval personnel (Coburn & Young, 1949) and in combat troops in the tropics (McMitlan & Hurwitz, 1969).

In their reviews of potential predisposing and aetiological factors, Wannamaker (1970), and Peter & Smith (1977) drew attention to the high incidence in the tropics, in places where ventilation is poor and where hot humid atmospheres prevail, and to the fact that the greater prevalence is in the summer months. While the Red Lake outbreak in the northern USA showed that cold climates were not inimical to the disease, our and other studies suggested that hot and sweaty skin in physically tired men was particularly susceptible. There is general agreement that streptococci do not penetrate normal intact skin and that a breach of the epidermis by trauma, insect bite or invasion by scabies, mites or other parasites must precede the infection (Dudding *et al.* 1970).

Views on the carriage of streptococci on normal skin are controversial. Clinical observation and experimental attempts to induce carriage generally suggest that short-term carriage is most unusual even when treatments are given to remove from the skin debris, proteins and lipids, particularly sebum, which are known to be bactericidal (Leyden, Stewart & Kligman, 1980; Ricketts, Squire & Topley, 1951). On the other hand in situations of high endemicity streptococci have been found on intact skin (hands, nails, perineum) of cases and contacts (Allen, Taplin & Twigg, 1971).

Nasal carriage in many outbreaks was quite common, although associated respiratory infection rarely occurred. For example, in the detention centre outbreak, 19% of the case contacts were nasal or throat carriers or both, while in Vietnam 8% of cases had the same organism in the nose as on the skin lesions, but in neither episode was respiratory disease reported. The evidence from outbreaks such as the one described here is, however, that skin sepsis is not spread by the respiratory or airborne route. Contact direct from lesions or indirectly through clothing, equipment and poor hygienic practices seems the most likely means, but there is little published data to support the contention.

Group A streptococci causing skin sepsis differ from those causing respiratory disease (Parker, 1969). Each has distinct M antigens and often recognizably different T antigens as well. Certain strains are nephritogenic. Skin strains not

infrequently colonize the nose and throat but rarely if ever give rise to respiratory disease. Certain T patterns predominate among the majority of skin strains and many of the M proteins are unusual or new. The biological basis for the propensities of skin strains is not known though there are suggestions that they have a basic resistance to sebum to which most streptococci are rather sensitive (Hill, James & Maxted, 1963).

In all recently reported outbreaks the associated presence of *Staphylococcus aureus* was noted in 47% to 68% of cases – and the question has been raised as whether the staphylococcus might be the initiator of the lesions. It is now generally thought that this is not so for the following reasons. The bullous lesion from which staphylococci only are isolated is distinct from the non-bullous in which streptococci with or without staphylococci are found. The latter lesions appear the same whether or not staphylococci are present or are artificially introduced. While phage type 71 tends to predominate in bullous lesions, there is no consistent pattern amongst staphylococci isolated from non-bullous ones and the same organisms can often be found in the patient's nose and in the lesions. Many staphylococci found in bullous lesions produce an extracellular substance which inhibits  $\beta$ -haemolytic streptococci; staphylococci in non-bullous lesions either do not produce the substance or it is present in insufficient quantity to be of significance. Finally, if swabs are taken at the vesicular stage of the non-bullous lesions – i.e. before overt ulceration has taken place – only streptococci are generally found. There may well be an interaction between the two organisms in the developed lesion, but the initial event is purely streptococcal.

The serological response to skin infections differs from that to respiratory ones. The ASO titres are low or absent, although the skin organisms are known to produce as much streptolysin O as those which, after invasion other than through the skin, induce high levels regularly. The anomaly is thought to be due to binding and subsequent inhibition of streptolysin by skin cholesterol and other lipids (Kaplan & Wannamaker, 1976). The response to the enzymes DNA-ase B (Kaplan *et al.* 1970) and hyaluronidase (Potter, Siegel & Simon, 1968) are more regularly found, but are even then insufficiently reliable to be of value diagnostically. Protective type-specific antibody may be produced against the M antigens (Bisno & Nelson, 1974) and where it is present, reinfection by the same M-type is prevented.

While untreated pyoderma is usually self-limiting, over 90% of lesions contain large numbers of streptococci even 10 days after the onset, increasing the possibility of complications, such as local extension and metastatic spread to distant sites and, with some strains, glomerulonephritis. Disseminating streptococcal disease can still be life-threatening despite the continued sensitivity of the organisms to antibiotics (Goepel *et al.* 1980). Topical treatment alone fails to cure the infection and can be regarded only as ancillary to systemic therapy. While most authorities regard benzyl penicillin as the drug of choice, erythromycin and clindamycin are preferred by others. The efficacy of various regimes is summarized by Peter & Smith (1977).

The size and morbidity of some of the outbreaks has led to attempts at

prophylaxis for contacts and for those likely to be exposed to risk of infection. Intramuscular penicillin given to children in affected families reduced the incidence of secondary cases by 40% over a control group (Ferrieri, Dajani & Wannamaker, 1973). The effect is, however, short lived, and in a trial in Trinidad, two weeks after a group had been given a single injection of prophylactic penicillin they were recolonized by streptococci (Sharrett *et al.* 1974). In one outbreak of pyoderma the respiratory carriers were all given courses of penicillin, and while no further cases occurred thereafter, no bacteriological follow-up was undertaken (Communicable Disease Reports, 1978). Others have relied more on attention to hygiene, regular bathing with bactericidal soap and early local treatment of any skin lesions (Leonard, 1967; McMitlan & Hurwitz, 1969). However, streptococcal pyoderma occurs generally in circumstances which render the rigorous observance of such measures difficult to enforce and maintain. Our outbreak was unusual in the number of people involved, the relative mildness of most of the cases, its epidemiological pattern and the behaviour of the organisms involved.

The actual incidence is likely to be greatly in excess of just those cases in which organisms were isolated. From the presence of recent scars and from verbal information, most men in most troops suffered an attack of skin sepsis during the 30-week period of training, and a small number had more than one attack.

Some new recruits had serological evidence of previous acquaintance with streptococci, but immunity is type specific. The organisms forming PT 1658 are 'skin strains' and are not regularly isolated from the general population. Representatives of PT 180 are more widely distributed. Susceptibles were thus entering the camp at the rate of 50 or so each fortnight, providing a continuously fresh population for the maintenance of the disease within a relatively closed community.

The relative numbers of isolations of each fluctuated very little. They appear to be of moderate virulence and non-nephritogenic, and to have little if any affinity for respiratory tissues. While Parker found that in a series of patients there tended to be a sequence of streptococcal types during the course of their illness (Parker *et al.* 1968), our cases were remarkable for the purity and consistency of their culture isolates during an episode of infection. A further unusual feature is the long-term presence of the two strains in almost unchanged proportions in the affected population. Generally one strain dominates an epidemic, and when in Trinidad two major strains were present this was only for a short period of overlap when one strain was replacing another (Parker, 1969).

The third serotype might have been expected to become dominant since it was new to the camp. Instead it seemed, like the other strains, to have established an optimal level of endemicity. However, the most recent strain seems to have replaced the others almost entirely.

The lesions seem milder than those described from Vietnam. Our cases rarely required hospitalization, while the average hospital stay with the American troops was 6 days (Allen, Taplin & Twigg, 1971). Treatment and control were built round the infectious nature of the local lesion, the assumed spread by contact and the need for active surveillance. Early in the epidemic the recruits feared loss of

training time far more than the discomfort of streptococcal pyoderma. The incidence did not, however, begin to show much decrease until more general measures were introduced. The fall in infected rope burns after long trousers replaced shorts was significant, and it is believed that the application of antiseptics to wounds before they became infected was also effective.

Education led to early recognition and the more rapid sterilization of lesions with consequent shorter periods of infectiousness. While we felt that all these measures contributed to the gradual, but sustained fall in numbers of cases in recent months, the exercise could not be controlled and the part played by natural fluctuations in incidence could not be assessed. The degree of immunity to the two main types may have reached an order in which spread became difficult to sustain – which may be supported by the emergence of the new type PT 2681. Mass penicillin prophylaxis has been considered, but as at present the disease is in 95 % or more of cases not particularly serious, the potential hazards outweigh the uncertainty of success.

#### REFERENCES

- ALLEN, A. M., TAPLIN, D. & TWIGG, L. (1971). Cutaneous streptococcal infections in Vietnam. *Archives of Dermatology* **104**, 271–280.
- ANTHONY, B. F., PERLMAN, L. V. & WANNAMAKER, L. W. (1967). Skin infections and acute nephritis in American Indian children. *Paediatrics* **39**, 263–279.
- BISNO, A. L. & NELSON, K. E. (1974). Type specific opsonic antibodies in streptococcal pyoderma. *Infection and Immunity* **10**, 1356–1361.
- COBURN, A. F. & YOUNG, D. C. (1949). *The Epidemiology of Haemolytic Streptococcus during World War II in the United States Navy*. Baltimore: Williams and Wilkins.
- COLLING, A., KERR, I., MAXTED, W. R. & WIDDOWSON, J. P. (1980). Streptococcal infection in a Junior Detention Centre: a five year study. *Journal of Hygiene* **85**, 331–341.
- COMMUNICABLE DISEASE REPORTS (1978). Streptococcal disease in a detention centre. 78/5.
- CRUICKSHANK, R. (1953). The epidemiology of some skin infections. *British Medical Journal* **i**, 55–59.
- DILLON, H. C., MOODY, M. D., MAXTED, W. R. & PARKER, M. T. (1967). The epidemiology of impetigo and acute glomerulonephritis. Results of serological typing of Group A Streptococci. *American Journal of Epidemiology* **86**, 710–723.
- DUDDING, B. A., BURNETT, J. W., CHAPMAN, S. S. & WANNAMAKER, L. W. (1970). The role of normal skin in the spread of streptococcal pyoderma. *Journal of Hygiene* **68**, 19–27.
- FERRIERI, P., DAJANI, A. S. & WANNAMAKER, L. W. (1973). Benzathine penicillin in the prophylaxis of streptococcal skin infections: a pilot study. *Journal of Paediatrics* **83**, 572–577.
- GOEPFEL, J. R., RICHARDS, D. G., HARRIS, D. M. & HENRY, L. (1980). Fulminant *Streptococcus pyogenes* infection. *British Medical Journal* **ii**, 1412.
- HILL, M. J., JAMES, A. M. & MAXTED, W. R. (1963). Some physical investigations on the behaviour of bacterial surfaces. The occurrence of lipid in the streptococcal cell wall. *Biochimica et Biophysica Acta* **75**, 414–424.
- KAPLAN, E. L., ANTHONY, B. F., CHAPMAN, S. S., AYOUB, E. & WANNAMAKER, L. W. (1970). The influence of the site of infection in the immune response to Group A streptococci. *Journal of Clinical Investigation* **49**, 1405–1414.
- KAPLAN, E. L. & WANNAMAKER, L. W. (1976). Suppression of the anti-streptolysin response by cholesterol and by lipid extracts of rabbit skin. *Journal of Experimental Medicine* **144**, 754–767.
- LEONARD, R. R. (1967). Prevention of superficial cutaneous infections. *Archives of Dermatology* **95**, 520–523.
- LEYDEN, J. J., STEWART, R. & KLIGMAN, A. M. (1980). Experimental infections with Group A streptococci in humans. *Journal of Investigative Dermatology* **75**, 196–201.

- McMITLAN, M. & HURWITZ, R. (1969). Tropical pyoderma in Vietnam. *Journal of the American Medical Association* **210**, 1734–1736.
- PARKER, M. T. (1969). Streptococcal skin infection and acute glomerulonephritis. *British Journal of Dermatology* **81** (supplement 1), 37–46.
- PARKER, M. T., BASSETT, D. C. J., MAXTED, W. R. & ARNEAUD, J. D. (1968). Acute glomerulonephritis in Trinidad: serological typing of Group A streptococci. *Journal of Hygiene* **66**, 657–675.
- PETER, G. & SMITH, A. L. (1977). Group A streptococcal infections of the skin and pharynx (first of two parts). *New England Journal of Medicine* **297**, 313–317.
- PILLSBURY, D. M. & LIVINGOOD, C. S. (1968). Dermatology. In *Internal Medicine in World War II*, vol. 3, ch. 10. Washington: Department of the Army.
- POON-KING, T., POTTER, E. V., SVARTMAN, M., ACHONG, J., MOHAMMED, I., COX, R. & EARLE, D. P. (1973). Epidemic acute nephritis with reappearance of M-type 55 streptococci in Trinidad. *Lancet* **i**, 475–479.
- POTTER, E. V., SIEGEL, A. C. & SIMON, N. M. (1968). Streptococcal infections and epidemic acute glomerulonephritis in South Trinidad. *Journal of Paediatrics* **72**, 871–884.
- RICKEYTS, C. R., SQUIRE, J. R. & TOPLEY, E. (1951). Human skin lipids with particular reference to self-sterilising power of skin. *Clinical Science* **10**, 89–93.
- SHARRETT, A. R., FINKLEA, J. F., POTTER, E. V., POON-KING, T. & EARLE, D. P. (1974). The control of streptococcal skin infections in South Trinidad. *American Journal of Epidemiology* **99**, 408–413.
- TAPLIN, D., LANSDALE, L., ALLEN, A. M., RODRIGUEZ, R. & CORTES, A. (1973). Prevalence of streptococcal pyoderma in relation to climate and hygiene. *Lancet* **i**, 501–503.
- WANNAMAKER, L. W. (1970). Difference between streptococcal infections of the throat and skin (first of two parts). *New England Journal of Medicine* **282**, 23–31.