AN ACUTE OUTBREAK OF STAPHYLOCOCCAL ENTEROTOXIN FOOD POISONING

EDMUND D. COOPER, M.D., F.R.F.P.S. (GLASG.), D.P.H., F.R.S.H., *Medical Officer of Health, City of Cape Town*

It was mainly due to G. M. Dack and his co-workers[1] (1930) that the attention of the medical profession was focused on certain groups of staphylococci as a cause of food poisoning outbreaks.

In 1950 Feig[2] reported that 78% of all food-poisoning outbreaks occurring in the USA were due to this group of organisms. In contrast to this finding, it would appear from a study of the medical literature that in the UK the largest proportion of food poisoning outbreaks are caused by the salmonellas. The reason for this difference is not obvious.

According to Carter[3] very few food outbreaks were reported upon or investigated in Glasgow—with its population of over a million individuals—before 1946. The true position regarding food poisoning in that city is in no way reflected by the available laboratory records or investigations. Since the attention of epidemiologists and public-health personnel has been focused on food as a cause of serious and acute illness in Glasgow, 519 incidents have been reported between 1946 and 1955, in which no less than 1,676 persons were involved. Only 6% of the total food-poisoning incidents during this period were due to staphylococci.

Du Bos[4] (1948) reports that fatalities are rare in staphylococcal food poisoning, only 8 deaths having been recorded in several thousand reported cases, and that of these only 4 could reasonably have been directly attributed to the food-poisoning episode. He suggests that the severity of the attack varies with the amount of enterotoxin ingested.

An Australian Outbreak

In 1954 Crowe[5] reported an explosive outbreak of food poisoning in the state of Victoria, Australia, due to the staphylococcus, in which phage typing was employed for the first time, in an effort to trace the source of infection. Food poisoning in this particular incident was promoted by hot summery conditions, and affected 80 individuals, 1/5th of the total number of persons who had partaken of a lunch of cold mutton and ox tongue at the annual agricultural show of a small Victoria town. The symptoms were classical. Within 2½-3 hours after the lunch a number of persons developed severe nausea, vomiting, abdominal cramp, prostration and collapse. Observers described the scene dramatically. Collapsed patients were lying round the showgrounds and in the garden of the local doctor's house. Others were vomiting from cars into the street gutters. The scene resembled a battlefield in miniature.

Food poisoning is not a notifiable disease in Australia, and it was only several days later that the State Department were apprised of the outbreak. Dumped foodstuffs left over from the lunch were found in the showgrounds and yielded heavy growths of coagulase-positive staphylococci which, on phage typing, were found to be identical with those recovered from the noses of 3 persons who served at the lunch in question.

An American Outbreak

Wain and Blackstone[6] report a staphylococcal food-poisoning outbreak amongst persons attending a picnic at Mansfield, Ohio, USA, on 17 September 1955, in which 120 individuals were brought to the local hospital out of 306 persons at risk. A further 124 persons were also affected, but not sufficiently seriously to attend or be admitted to hospital. Notwithstanding the severity of the symptoms during the time of the attack, most individuals recovered within 24 hours, whether they had received treatment or not.

All affected gave a history of consuming ham, and all without exception volunteered that the ham had no abnormal taste or odour and could only be described as being delicious. The hams in question were slow-baked all night on 16/17 September, and then boned and sliced by an individual who was suffering from open acne pustules of the face. Thereafter the sliced hams were returned to the ovens, which were kept at 200°F until 2 p.m., when they were collected and transported in the back of a station wagon, on a day when the mean temperature was 90°F, before being consumed at 6 p.m. that evening. Cultures obtained from the acne lesions proved to be identical with those recovered from the hams in question.

Wain and Blackstone[6] also refer to the short incubation period of 1-6 hours in staphylococcal food poisoning and suggest that if the onset of symptoms is longer than 11 hours, staphylococcal gastro-enteritis can usually be ruled out.

They also adduce that the staphylococcal enterotoxin acts on the peripheral sensory nerve structures of the viscera and not on the vomiting centre in the fourth ventricle.

Practically every reported outbreak reveals that the incriminated food was handled or prepared by an individual who, on later investigation, was found to be a nasal carrier, or was suffering from acne or other septic skin lesion. In addition, it also appears to be a *sine qua non* that the food shall be stored at kitchen temperatures for several hours before serving. The reports indicate that the order in frequency of foods responsible are: (1) Meat products, particularly ham, (2) cream and custard products, (3) poultry.

THE PRESENT OUTBREAK

On Boxing Day, 26 December 1957 one of the largest local sports clubs in Cape Town was the sponsor of a bowls tournament not limited to their own members.

The club, which had recently been established, had erected a completely modern and up-to-date club house, included in which was a modern kitchen, whose design and size is such that provision of a limited number of lunches and teas for its members is possible. The refrigeration space available was strictly limited, and conformed to the needs and requirements for which the kitchen had been designed; but was never intended to store the quantities of foodstuffs necessary to provide lunches for the number of persons who were present at the club on the day in question.

The club's catering arrangements fell under the control of an honorary catering committee of members, and European and non-European cooks, waiters and kitchen staff were employed.

Three or four times a year the club sponsors a bowls tournament, when as many as 300 people can be expected to participate. On these occasions, with the assistance and help of club members, it has been the general practice of the club to provide cold lunches for this number.

For the tournament on 26 December 1957, when about 250 persons were expected, large orders were placed with outside suppliers for corned beef, salted fresh tongues, pickled herrings, vegetables, fruit, and ice cream. The tongues and corned beef were cooked in the club kitchen on the afternoon of 24 December and were left to cool overnight in the pantry. The weather over this period was particularly hot and humid.

On the morning of 26 December at about 11 a.m., during the preparation of the lunch by voluntary and paid members of the staff, two of the permanent kitchen staff made complaint of nausea and associated abdominal pain, followed shortly afterwards by vomiting. No association between this train of symptoms and the food which was being prepared was appreciated by the club secretary or the voluntary kitchen helpers, and the two staff members concerned, being incapacitated and of no further use to the club, were sent home by taxi.

Lunch was served from about 1.30 to 2.30 p.m., and about 1-1½ hours afterwards the first casualties occurred. The numbers affected mounted with alarming rapidity, until in a very short time large numbers of the members and their guests were lying all over the club house and grounds in various positions of prostration and collapse. Eye witnesses described the scene as a shambles, and likened it to the after-effects of the detonation of a high-explosive bomb.

Many who felt the first onset of symptoms, or saw what was happening to others, hastened to their cars and made for home as quickly as they could. The local ambulance service was busily employed all the afternoon removing casualties to hospital, where first-aid treatment was given to many before they were sent home; a number, however, whose condition was serious, had to be admitted to the wards.

Food poisoning is not a notifiable condition in South Africa, but the City Health Department heard of the episode at 7 p.m. the same day (Boxing Day), and was thus in a position to get first-hand information from unaffected club members still present on the scene, and to collect samples of the food remaining from the lunch. A check of the staff members the following day revealed no one with any evidence of septic skin lesions.

Detailed information furnished by about 120 affected individuals of this outbreak indicated that, with the exception of only 2 persons, all had either consumed cold tongue or corned beef, or both. Except for these two, the individuals who had not partaken of these meats were unaffected. Of about 290 persons who attended this function, no less than 220 were affected—some more seriously than others. No deaths occurred; although one individual, seriously ill, was kept in hospital for over 3 weeks.

Additional epidemiological evidence that these two items were responsible for the outbreak was the fact that it subsequently came to the notice of the Department that 2 non-European families who had partaken of pilfered portions of the meat on 25 December (Christmas Day) had been similarly affected and were in such a serious state that they also had to be hospitalized.

Bacteriological Investigations

Specimens of all available foodstuffs which were served at the lunch were collected and submitted to the Government Pathological Laboratory (Union Health Department), Cape Town, for bacteriological examination. A report received some days later indicated that a coagulase-positive *Micrococcus (Staphylococcus) pyogenes aureus* (fermenting mannite and liquifying gelatine) was isolated from the following articles of food consumed at the lunch in question: Ice cream, corned and sliced beef, pickled herring, strawberries, sliced tongue (from large part), french salad, beetroot soup, butter, and unsliced tongue. No *Micrococcus pyogenes aureus* was isolated from mayonnaise, milk, or the remains of the brine still present in the opened and empty barrel of pickled herring. More significantly, coagulase-positive *Micrococcus pyogenes aureus* was recovered from one sample of brine obtained from one of the brine vats at the premises of the butcher who supplied the corned beef.

All the positive cultures so obtained were submitted to the Bacteriological Department of the University of Cape Town for phage typing and comparison with cultures obtained there from the vomitus and faeces of victims of the outbreak admitted to the Groote Schuur Hospital.

As difficulty was experienced in carrying out phage typing of the culture material obtained from the foodstuffs and from the patients, the culture material was submitted to Dr. R. E. O. Williams of the Streptococcus and Staphylococcus Reference Laboratory, Central Public Health Laboratory, Colindale Avenue, London, who very kindly furnished a report from which the following results of phage typing are extracted:

Vomitus. Of 10 specimens of vomitus (K1-K10), 9 gave *Micrococcus pyogenes aureus* on culture. On phage typing, 2 of these cultures (K4 and K6) were found to be phage-group I, type 29, and 2 (K8 and K9) phage-group III.

Stools. Of 10 specimens of stool (K11-K20), 9 gave *Micrococcus pyogenes aureus* on culture. On phage typing, 4 of these cultures (K13, K16, K18 and K19) were found to be phage-group I, type 29.

From the one hospitalized patient who was seriously ill cultures of *Micrococcus pyogenes aureus* were obtained from stool specimens and material from a skin vesicle (K21 and K23). Both of these cultures were found to be phage-group I, type 29.

Cultures from food remains. The cultures of *Micrococcus pyogenes aureus* which were obtained from food remains on being phage typed gave the following result:

Ice cream	phage-type 29+
Sliced corned beef	phage-type 29
Fresh cream éclair	phage-types 29 and 77
Strawberries	phage-types 29, 79 and 77
French salad	phage-type 29+
Beetroot soup	phage-types 29 and 77
Unsliced tongue	phage-type 29

Other cultures were phage-typed as follows:

Nasal swabs from a member of the kitchen staff	phage-types 29 and 77
Brine from one of the vats in the butcher's shop	phage-type 29

From these data it would appear that phage-group I, type 29, was responsible for the outbreak in question. According to Dr. Williams, group I, type 29, is an unusual staphylococcal strain and, in his experience, has not in the past been implicated as the cause of a food-poisoning outbreak.

Commentary

As it was only those persons who had consumed cold tongue or cold corned beef who were affected in this food-poisoning outbreak it can with certainty be inferred that these two articles contained large quantities of preformed enterotoxin, and that this was occasioned by the fact that owing to inadequate cold storage facilities they were permitted to cool down overnight in a hot pantry adjacent to the kitchen at a time of high atmospheric temperature.

The recovery of phage-type 29 from brine obtained from one of the brine vats of the butcher supplying the corned beef appears to be more than significant.

Phage-group I, types 29 and 77, were also isolated from a nasal swab from one of the permanent kitchen staff at the club. While it could be considered that this individual might have been responsible for the outbreak of food poisoning, the fact that a culture which conformed to phage-group I, type 29, was isolated from a specimen of brine from one of the brine tanks situated in the shop of the butcher who supplied the corned beef for the lunch in question is more indicative that this was the source of the present outbreak.

The recovery of this phage type from the tank in question, and its presence in foodstuffs such as French salad, strawberries and beetroot soup—articles of food not usually responsible for food-poisoning outbreaks—leaves me with no alternative but to postulate wide dissemination of this organism from the corned beef *via* preparation tables, knives and hands of the kitchen staff to practically every article of food used at this function, as well as to the nares of one of the food handlers on the permanent staff.

It is also categorically possible to suggest that, notwithstanding the one infected brine tank and its meat contents,

this outbreak would not have occurred—or had it occurred its effects would have been strictly limited—had suitable and satisfactory facilities been available for the rapid and immediate cooling and storage of those products likely to serve as a media for the multiplication of the various types of pathogenic organisms which play so important a part in outbreaks of this type.

SUMMARY

1. This would appear to be the most extensive outbreak of food poisoning caused by a coagulase-positive *Micrococcus* (*Staphylococcus*) *pyogenes aureus* enterotoxin which has been reported in the Union of South Africa. Of approximately 290 individuals at risk, no less than 220 were involved and developed the classical signs of this type of food poisoning.

2. The use of a kitchen not designed, nor suitable, for providing the large number of meals required on this occasion played a not insignificant part in the occurrence of the present outbreak.

3. The limited refrigeration facilities in keeping with the purpose for which the kitchen had been designed and which necessitated hot cooked corned beef and tongue having to be cooled down in a hot pantry at a time of the year when atmospheric conditions were ideal for bacterial proliferation were directly responsible for the severity and intensiveness of the outbreak.

4. The necessity for rapid and effective cooling of all foodstuffs which are to be retained for subsequent use could not be better brought out than by the present outbreak.

5. The importance of preventable food-poisoning outbreaks such as described here is not without significance to the catering industry, the medical profession, and the health departments of central and local authorities.

I would take this opportunity of thanking Prof. R. Turner and Dr. L. S. Smith, of the Union Health Department, Prof. A. Kipps and the staff of the Bacteriological Department of the University of Cape Town, and Dr. R. E. O. Williams and the staff of the Streptococcal and Staphylococcal Reference Laboratory, Colindale Avenue, London, N.W. 9, for reports on the bacteriological investigations and phage-typing results which are set out in this paper.

REFERENCES

1. Dack, G. M., Cary, W. E., Woolpert, O. and Wiggers, H. (1930): J. Prev. Med. (Baltimore), **4**, 167.
2. Feig, M. (1950): Amer. J. Publ. Hlth, **40**, 279.
3. Carter, H. S. (1954): Glasg. Med. J., **35**, 244.
4. Du Bos, R. J. (1948): *Bacterial and Mycotic Infections in Man*. Philadelphia: J. B. Lippencott.
5. Crowe, E. J. (1954): Med. J. Austral., **1**, 468.
6. Wain, H. and Blackstone, P. A. (1956): Amer. J. Dig. Dis. (N.S.), **1**, 424.