Personal education is of more value than impersonal propaganda. The education of the parents and other adults can take place in welfare clinics and through the medium of health visitors, district nurses and general practitioners. The education of the children, and this is very fruitful, should be through the teachers. With regard to the manufacturers of home appliances, one should aim to make the public critical of goods which they buy and, in time, public opinion will demand safe articles. On this aspect legislation might be an advantage.

Mr. John Bunyan: A system of first-aid treatment of injured hands has been tried and found satisfactory in some pits in the North of England. The arrangement is simple. A steel cabinet with a swing-down door is placed in convenient positions in the pit where hand accidents are most likely to occur. Every miner knows that on injuring his hand all he has to do is to lower the hinge lid of the cabinet where he will find a basin and bottles containing sodium hypochlorite and water which, on being emptied into the basin, provide the correct concentration of sodium hypochlorite. He immerses his injured hand and waits for the first-aid man who applies a simple silk envelope after ten minutes. The sodium hypochlorite removes the contaminated blood and blood clot and causes a clean blood clot to form, reduces traumatic inflammation and takes away pain. The envelope, which is simple to apply, gives complete protection to the wound and causes no pain on removal. Having thus protected the wound, it is not of such great moment that some hours may elapse before the patient can be adequately treated at hospital.

Dr. Percy Stocks, mentioning the work being done in America to reduce home accidents' thought that general appeals were unlikely to succeed and that each cause of accident would have to be attacked in detail. A necessary prelude to such action was to obtain good statistics and the General Register Office was doing three things to that end. Coroners' certificates were providing more detail than hitherto of the circumstances of fatal accidents and where they occurred. After lengthy discussion with experts over the water a classification both by nature of injury and cause of accident had been grafted into the International List, which was now designed for morbidity as well as mortality statistics. This contained definitions and enough detail of grouping to satisfy anyone. The teaching hospitals had in the past paid little attention to causes of accidents but now they were being asked to do so in the interests of prevention. It was hoped to secure sufficient information about in-patients during 1949 to provide some useful statistics in the full detail of the new list. This would mean hard work but he believed the hospitals could, in that way, make an important contribution to the eventual reduction of many kinds of accidents.

[December 6, 1948]

DISCUSSION ON FOOD POISONING

Dr. E. T. Conybeare: Administrative measures.

Prominent among the administrative measures concerned with food poisoning in the past have been:

(1) Encouragement by a Government department of two outstanding investigators of food poisoning.

(2) Provision of a laboratory service for the special investigation of bacterial food poisoning.

(3) The issue to medical officers of health of memoranda offering guidance in the investigation of food poisoning outbreaks and the provision of practical help to them in circumstances of exceptional difficulty attending such outbreaks.

(4) The promotion of legislation facilitating the ascertainment of cases of food poisoning

and useful in preventing it.

Before attempting to suggest possible lines of future development it may be as well to

consider the lessons of the past.

In 1890, Ballard, after ten years' work in which, without any accurate bacteriology to help him, he clearly discerned the infective origin of most food poisoning and distinguished the toxin from the infection form of outbreak, wrote: "What does all this indicate as an efficient precaution against food poisoning? The grand precaution of all is the very commonplace one signified by the word *cleanliness* and it should be the business of the conservators of public health to see that this is observed as well as the business of every master or mistress of a family."

In 1925 Sir W. Savage, after over twenty years' study of the subject, postulated that if salmonella outbreaks could be controlled food poisoning would practically cease to exist as a public health problem. Savage thought that the key to this was first, a complete knowledge of the reservoirs of salmonella infection and then of the paths by which these bacilli

could reach human food; to help in this Savage wanted bacterial food poisoning made notifiable, and more attention paid to the investigation of clinically mild and numerically small outbreaks.

Fig. 1 is a chart of the actual numbers of outbreaks of food poisoning recorded as "reported" to the Ministry of Health between 1927 and 1947. Up to 1939 the source of

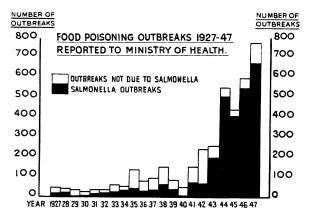


Fig. 1.

information was the Ministry's laboratory in London plus the special reports sent in by medical officers of health. After 1939 information was available through the regional laboratories of the Emergency Public Health Laboratory Service and its successor, the Public Health Laboratory Service. It is difficult to know the real meaning of the considerable increase in the number of reported outbreaks which started about 1942. Notification beginning in 1939 did not apparently produce any remarkable change. The war may have interfered. It is possible that from 1942 onwards when communal feeding became a more common practice and dried egg was used on a larger scale, there was a real increase in the number of outbreaks. Alternatively the apparent increase is only a result of the more frequent ascertainment of outbreaks resulting from the work of the Public Health Laboratory Service.

Past experience of administrative measures provides the following observations and suggestions:

Memorandum.—The current Ministry of Health memorandum on food poisoning is out of date. It was issued before food poisoning became notifiable and before the Public Health Laboratory Service came into existence and it does not mention staphylococcal enterotoxin. It should either be extensively revised or replaced by a new circular.

Notification.—Too much should not be expected of this procedure in connexion with food poisoning. Notification is bound to be an imperfect measure of incidence for a condition which, in its milder forms, escapes medical attention altogether and which, in any event, cannot be rigidly defined or recognized clinically with complete certainty. The main purpose of notification must still be to bring to the notice of the medical officer of health circumstances requiring further investigation or action of a kind which the general practitioner cannot be expected to undertake.

Hitherto the local data produced by notification, which was begun in 1939, have not been collected for central tabulation and analysis, but from January 1949, medical officers of health will be asked to include in their weekly return to the Registrar-General a note of the number of notified cases of food poisoning. These figures will be published as a total, and also by districts, in the Registrar-General's Weekly Return and, after correction for wrong diagnosis, the totals will be available in the Registrar-General's Quarterly Returns and, no doubt, will appear eventually in the Annual Statistical Review. It is likely that the data obtained in this way, apart from being more or less incomplete and therefore inaccurate as a measure of incidence, will be subject to fluctuations of an enigmatic kind comparable to those in the figures for the notification of dysentery to which Dr. J. Alison Glover has lately drawn attention. It would be a mistake to allow these data of notified cases to replace the collection of records of investigated outbreaks. The data obtained from notification should be combined with the information resulting from

the investigation of outbreaks so as to make possible a useful annual analysis by the Ministry of Health and the Ministry of Food.

Laboratory Services.—The regional laboratories of the Public Health Laboratory Service now cover practically every part of the country and are supported by central reference laboratories in which epidemiologically important procedures, like the typing of salmonellæ and staphylococci, can be done. It is now within the bounds of practical possibility for material from almost every notified case of food poisoning to be examined by competent bacteriologists.

It has been suggested that the routine laboratory investigation of food poisoning has become a waste of time. Although there are only a very small number of instances on record in this country in which, as a result of laboratory or other evidence obtained in the investigation of an outbreak of food poisoning, it has proved possible to take any effective preventive action, such as stopping the sale of infected food remaining unsold, the view that the routine laboratory investigation of food poisoning outbreaks is no longer worth while and that it ought to be entirely given up or limited in extent cannot be accepted. Present knowledge, at least in this country, is not sufficient to warrant this step.

Laboratory tests entirely divorced from epidemiological enquiries at the scene of an outbreak may be of little or no value. If they act too independently of one another neither the field epidemiologist nor the laboratory worker will ever solve such outstanding problems as are presented, for instance, by outbreaks in which, although food is a possible vector, it cannot be shown to contain a known bacterial pathogen or a chemical poison.

Legislation.—Ballard said nearly sixty years ago that the grand precaution of all was the commonplace one signified by the word cleanliness. To the extent that cleanliness of food depends on the cleanliness of persons it will not be easy to ensure it by legislation. It is, however, undoubtedly possible to legislate with some effect for environmental hygiene, i.e. for the cleanliness of places. The Food and Drugs Act 1938 contains special provisions in relation to premises used to prepare cooked meats for sale and to those used for the making of artificial creams, both of which are types of food liable to be concerned in food poisoning. The methods of food handling in catering establishments and other premises concerned in the sale and distribution of food rather than its preparation at present largely escape control by legislation. Here, as in food manufacture, the human link is the weakest in the chain.

There does not at the moment appear to be any special indication for further legislation relating, say, to catering establishments or to homes, which would be helpful in the prevention of food poisoning unless it be powers to prevent the employment in certain capacities related to food preparation or distribution of persons found to be healthy carriers of microorganisms such as salmonellæ or staphylococci of potentially dangerous types. On this line of action, there is also the possibility of requiring all persons engaged in such occupations to undergo routine laboratory tests to acquit them of a potentially dangerous carrier state.

Greater or more specific legal powers than those available at present do not appear to be required in connexion with organizations which manufacture or distribute food on a large scale. Legal powers tend to be too slow in action to be of practical importance; their greatest usefulness seems to be that their existence is often sufficient to dissuade those who might be willing to risk both the public health and the heavy damages which their victims might obtain in a Court of Law.

In time it may be that health education and a more critical climate of public opinion, rather than legislation, will produce standards of food handling in catering establishments and in homes that will offer a maximum protection against bacterial food poisoning. Notwithstanding our present difficulties in meeting demands for the new building and the new equipment which these standards entail, it is not too early to begin to inform the public more fully in this matter, starting perhaps by drawing attention to the causes of food poisoning outbreaks which originate in the home.

Dr. V. D. Allison: Most important among the enterotoxin-producing bacteria as a cause of food poisoning are the coagulase-positive staphylococci. The enterotoxin is pre-formed in the infected food, so there must be suitable conditions as regards the type of food, its moisture content, the temperature to which it is exposed and an interval of time between the actual infection of the food and its consumption by the victims, a time during which the organisms multiply and produce toxin. The clinical syndrome is now well recognized—short incubation period (one to seven hours with an average of three hours), acute onset with abdominal pain, nausea, vomiting and often diarrhæa, the symptoms lasting from three to twenty-four hours followed by rapid recovery, even from a state of collapse. Entero-

toxin can be produced in the laboratory from food-poisoning strains of staphylococci by growing them for forty-eight hours in 0.2% nutrient agar in an atmosphere containing 30% CO₂. Sterile filtrates from such cultures when taken orally by human volunteers in doses as small as 1-2 ml. give rise to the complete clinical picture of staphylococcal food poisoning.

The differentiation of serological types among coagulase-positive staphylococci, by Cowan (1939), Christie and Keogh (1940), and Hobbs (1948), and of bacteriophage types by Fisk (1942) and Wilson and Atkinson (1945), has been applied to field problems and has already added to our knowledge of the epidemiology of staphylococcal infections; the field is wide open for further investigation and progress. About 65% of coagulase-positive strains of staphylococci can be identified by phage typing and about 98% by serological typing—neither of the two methods is, as yet, practicable as a routine laboratory procedure.

The application of serological and phage typing to the investigation of outbreaks of staphylococcal food poisoning during the last three years has shown that the enterotoxin-producing strain may be isolated and identified from the suspected foodstuff in numbers ranging from 500,000 to more than 2,000 million organisms per gramme; it has also been isolated from the stools and vomit of victims and from the nose, nose and skin, or focus of skin sepsis in food-handlers who have manually handled the food, whether in the factory where there is large-scale production, in the restaurant or canteen where it is served, in the retail shop selling to individual customers, or in the home. These investigations prove beyond doubt that the main mode of infection of food by staphylococci is from the nose or hand of the food-handler to food which is manually handled, followed by storage of the food at temperatures which encourage multiplication of the organisms and production of enterotoxin. It can be readily appreciated that according to the stage at which infection of food occurs between preparation by the manufacturer and its ingestion by the victims, so the size of the outbreak will vary, from countrywide incidents down to canteen customers, shop clientele and family outbreaks.

During the last three years, I have phage-typed strains of staphylococci from 47 outbreaks of enterotoxin food poisoning. Strains from 26 of the outbreaks were isolated in this country from the incriminated foodstuff and frequently also from the nose or skin of a food-handler, and less often from the fæces and vomit of victims. The remaining 21 strains were received from Professor G. M. Dack, Chicago, Professor C. E. Dolman, Vancouver, Major A. R. Sandiford, Egypt, and Dr. E. S. Horgan, Khartoum. The foods from which the strains were isolated included beef, tongue, lamb, bacon, Vienna sausage, meat pie, glazed liver sausage, pork cheese, pressed beef, meat sandwiches, hake, potato salad, goat's milk, ice-cream, trifle and cheese. Bread, vegetables and fruit, fresh or cooked, have rarely been implicated as the vehicles of staphylococcal or indeed other food poisoning; in the case of bread and fruit this may be, in fact, due to the acid pH of the food. It is interesting to note that strains from 30 (64%) of the 47 outbreaks belonged to one phage and serological type designated "6/47, IIIc"; these included 13 strains received from the four sources abroad, the remaining 17 having been isolated in this country. Eight of the strains belonged to phage type "42D", including three from Chicago, one from Egypt and four from this country. In passing it may be noted that phage type "42D" is the strain commonly found in raw cow's milk and identified by Williams Smith as a frequent cause of bovine mastitis—one of the strains from Egypt was isolated from cheese which seems to be a not uncommon vehicle of staphylococcal food poisoning in that country as all four strains received from this country were isolated from cheese. On the other hand a strain isolated from a mixture of milk from cow, goat, sheep and camel which caused an outbreak in the Sudan was traced to the goat's milk and identified as phage type "6/47". Of the remaining nine strains in the series three could not be typed and six were divided among three other phage types. When it is realized that by the use of 22 phages it is possible to identify between 20 and 30 phage types and subtypes of coagulase-positive staphylococci, it would seem to be significant that strains from 81% of 47 outbreaks belong to only two phage types.

I would not for one moment suggest that all staphylococci of phage type "6/47" are enterotoxin producers, as this type appears to be one of the commonest types found in the nose of man, but another phage type, designated "3A", is at least as common in the nose and has not yet been found as a cause of staphylococcal food poisoning although it is outstanding as a cause of pemphigus neonatorum.

On occasions reheated foods have been implicated as the cause of enterotoxin food poisoning and it is, of course, known that staphylococcal enterotoxin resists boiling for at least thirty minutes. The staphylococci are killed by the heat treatment but the enterotoxin remains active and potent, and in such cases bacteriological examination by the laboratory of specimens of suspected food and fæces and vomit from victims will yield negative results. The difficulties in attempting to trace an outbreak of this type to its source can readily be appreciated. The mere finding of one or more nasal or skin carriers among the personnel

handling the suspect foodstuff is not alone sufficient grounds for implicating them as the source of the outbreak or suspending them from duty. But if, in the absence of cultures of staphylococci from victims or suspected food, we find that a food-handler is harbouring in his nose, or on his hands, a strain of staphylococcus belonging to a phage type (6/47) preeminent as an enterotoxin producer and cause of food poisoning, we are on surer ground in recommending his suspension from food-handling duties until he ceases to be a carrier.

Even in outbreaks in which the same phage or serological type of staphylococcus has been isolated from the stools of victims, from the suspect foodstuff and from the nose or hands of a handler of the food, there is another aspect to be considered. My colleagues, Dr. Mair E. M. Thomas and Dr. Joan Edwards at Colindale, have found that coagulase-positive staphylococci are present in the stools of approximately 33% of routine specimens sent to the laboratory, in the absence of any suggestion of staphylococcal infection of the gut or of food poisoning. In a series of paired strains of typable coagulase-positive staphylococci, isolated from the nose and stools of 31 individuals, the strains from 21 pairs were the same. In the remaining 10, strains from stool and throat were the same type in four instances; in six instances the strain from the stool belonged to a type different from that found in the nose, the throat either not having been swabbed or found negative. The conclusion I draw from this is, that the sources of coagulase-positive staphylococci isolated from the stool of an individual are either the nose, in a person who is a nasal carrier, or the food consumed by the individual or both. Which of us does not daily ingest in our food hundreds, perhaps thousands, of staphylococci derived from human hands and nose without ill-effect? It is perhaps not surprising therefore that they should appear in the stool. Such findings must make us cautious in interpreting as staphylococcal food poisoning results based solely on finding the same type of staphylococcus in a foodstuff, in the stool of a person who has partaken of it, and in the nose or on the hands of someone who has handled it. We must have, in addition, the typical clinical and epidemiological picture, and if possible a count of the number of viable staphylococci per gramme in the suspect food. I have recently had specimens from an outbreak of suspected staphylococcal food poisoning in which the same phage type of staphylococcus was isolated from the stools of victims and from the unheated suspect food—but counts in the latter were less than 100 per gramme. I would be very loth to accept such tenuous evidence as proof of staphylococcal food poisoning. If I were asked what would be the minimum number of viable coagulase-positive staphylococci per gramme of food I would accept as supporting evidence of staphylococcal food poisoning, I would feel diffident in giving a figure. Tentatively, I might suggest about 500,000 per gramme, but even that might be too high if the organism were a potent enterotoxin-producer.

In the majority of outbreaks of staphylococcal food poisoning the evidence against the staphylococcus is largely circumstantial, as in only a few instances is it possible or practicable to determine that the suspect strain is capable of producing enterotoxin, by the only methods at present at our disposal—the human volunteer or feeding to monkeys. The human volunteer is rarely available, and monkeys when available are capricious in their sensitivity and reaction to oral administration of staphylococcal enterotoxin. The kitten test for staphylococcal enterotoxin is no longer accepted, and there is a great need for a bench test. Attempts to devise such a test have so far only shown the many difficulties that have to be overcome to make it specific for enterotoxin.

The human nose is the commonest reservoir of coagulase-positive staphylococci, but investigations by my colleague Dr. Betty Hobbs show that the skin may be an important secondary reservoir in the absence of carriage in the nose and that the clean-looking, healthy, healing cut may be teeming with staphylococci, just as Colebrook and Ross (1947) obtained a profuse culture of group A streptococci which caused infection in a patient from the scab on the almost healed abrasion on a surgeon's elbow.

The basis of preventive measures is clean food, prepared by clean persons, using clean utensils in clean kitchens, with avoidance of manual handling of food after cooking or processing, and preservation of food in the refrigerator (Allison, et al., 1949).

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Dr. Joan Taylor: In recent years the number of proven salmonella infections has increased (Table I), but it is impossible to say whether this change in incidence is real or is the result of improved laboratory facilities.

| TABLE I.—HUMAN SALMONELLOSIS IN ENGLAN | AND | NGLAND AND W | LES* |
|--|-----|--------------|------|
|--|-----|--------------|------|

| | | 1923-39 | 1940-41 | 1942-43 | 1944-45 | 1946-47 |
|---------------------|------|---------|---------|---------|---------|---------|
| Salm. typhi murium | | 235 | 112 | 144 | 505 | 1,046 |
| ,, enteritidis | | 54 | 28 | 35 | 90 | 63 |
| ,, thompson | | 49 | 50 | 28 | 39 | 66 |
| ,, newport | | 29 | 19 | 21 | 108 | 39 |
| ", choleræ suis | | 14 | 3 | 4 | 2 | 3 |
| " bovis morbificans | | 8 | 3 | 9 | 9 | 11 |
| ,, dublin | | 8 | 6 | 4 | 4 | 31 |
| Other types | | 32 | 25 | 5 | 9 | 19 |
| Salm. anatum | | | 2 | 6 | 13 | 8 |
| " montevideo | | _ | 2 | 18 | 42 | 29 |
| " oranienburg | | | | 41 | 50 | 27 |
| ,, meleagridis | | | | 8 | 16 | 3 |
| ,, tennessee | | | | 4 | 6 | 6 |
| Other types | | _ | 19 | 41 | 68 | 86 |
| Т | otal | 429 | 269 | 368 | 961 | 1,437 |

^{*}The figures for the years 1923 to 1939 were compiled from the Annual Reports of the Chief Medical Officer of the Ministry of Health; those for subsequent years from the weekly returns to the Public Health Laboratory Service, together with those from the Salmonella Reference Laboratory.

Salmonella infections.—It is obvious that the types which were commonly found in this country prior to 1940 still occur more frequently than do other types. The most frequent cause of salmonella food-poisoning is Salmonella typhi murium followed by Salmonella enteritidis, Salmonella thompson and Salmonella newport. A line has been drawn across the chart dividing the types occurring pre-war from those occurring during subsequent years. The types below the line were new to this country, their appearance in human infections coinciding with the distribution of dried egg to the general public. An investigation of salmonella types occurring in dried egg (M.R.C. Special Report Series, No. 260) undertaken by the Public Health Laboratory Service showed that these new types were present in this food. Of the new types isolated, the five most commonly found in dried egg were those which accounted for the majority of the new types in human infection.

Animal hosts.—It has been said that animal reservoirs are the most important factor in human salmonellosis. Mice and rats are known to be carriers of Salmonella typhi murium and Salmonella enteritidis and a number of outbreaks of food poisoning have been traced to this source. Outbreaks of Salmonella dublin infection, a disease of bovines, have not uncommonly been traced to milk, but a recent survey of this infection in bovines (Field, 1948) has shown that though this disease is relatively common in Mid and West Wales, infection having been proved on 66 farms, no cases of human infection were reported during the period of the survey. Nevertheless, an outbreak of Salmonella dublin infection in Aberdeen (Henderson et al., 1948) was traced to milk. For infected milk to give rise to cases of human disease it is obvious that certain conditions such as temperature, humidity and time interval suitable for the multiplication of organisms are necessary, so that an infective dose is ingested by the patient. Poultry are not uncommonly infected with salmonella organisms. Excluding Salmonella pullorum and Salmonella gallinarum, Salmonella typhi murium and Salmonella thompson have been responsible for the majority of outbreaks of disease among chicks in recent years (Gordon and Buxton, 1946), nevertheless no cases of human infection due to these organisms were connected with this source. In contrast with this, cases of human infection due to Salmonella enteritidis and Salmonella typhi murium have been traced to the eating of infected duck eggs. In these birds the ovary becomes infected giving rise to infection of the egg contents. In the hen the ovary is almost never affected. The hen egg occasionally may be infected by the passage of organisms through the shell as the result of fæcal contamination. The importance of cats and dogs as a reservoir of salmonella infections is not yet known, but recently a case of Salmonella typhi murium infection in a child was traced to a pet cat, and Salmonella thompson was isolated from a dog with diarrhea though no human cases occurred.

A source of infection, sometimes overlooked, is the use of so-called rat and mouse "virus" preparations for the destruction of vermin. Recently, from a virus preparation used in a public restaurant were isolated the human and danysz varieties of Salmonella enteritidis.

It is illogical that efforts are made to stop the handling of food by human excretors of the salmonella group yet living cultures of these organisms may be used deliberately in kitchens. One outbreak (Dathan *et al.*, 1947) was traced to this source.

The rôle of the human carrier is not known as it is often difficult to decide whether a particular case is the cause or the result of the eating of infected food. After salmonella infections patients commonly excrete the infecting organism for four to six weeks and some remain carriers for months. The dose of organisms necessary to cause human disease is large; Hormaechi et al. (1936) found that 2,000 – 4,000 million organisms of Salmonella typhi murium sometimes caused mild diarrhea in human volunteers.

Dysentery.—Organisms of this group may cause an explosive outbreak of food poisoning, such as that which occurred at Holborn (Scott, 1934). About 20 persons were infected with Sh. sonnei after partaking of pease-pudding, the latter having been infected by a child convalescing from the disease.

Paracolon organisms.—The importance of this group of organisms as the cause of food poisoning is not yet known. They are a group, difficult to define, with some of the characteristics of the salmonella and the Bact. coli groups. Last year an outbreak of gastro-enteritis occurring among school children was investigated. From the fæces of cases was isolated a single serological type of paracolon bacillus. From another outbreak of food poisoning occurring in a P.O.W. camp in the Midlands was isolated the same serological type. In addition, this organism has been isolated from two sporadic cases of gastro-enteritis. Dr. Hobbs fed cultures to two human volunteers both of whom had symptoms similar to those of the other cases, thereby proving that the organism was the cause of the disease.

More research will be necessary to assess the importance of other members of the paracolon group as a cause of food poisoning.

In conclusion it must be emphasized that food poisoning caused by the organisms discussed is dependent on the assimilation of a relatively large dose, which in turn is dependent on the food, temperature, moisture and time interval being suitable for the multiplication of these organisms.

An appreciation of these factors and the education of food-handlers should lead to a fall in the incidence of food poisoning.

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