completely, and there are no signs of disease in the eyes—such as optic atrophy—to be seen subsequently.

In all probability, however, these cases are identical in origin, though differing in severity, with those cases in which blindness ensues after a somewhat similar but more severe initial illness, and in which pallor of the discs is subsequently found. It has been suggested, as an explanation of the pathology of these cases, that a basal meningitis seals temporarily the foramen of Majendie, and so causes distension of the third ventricle, which by pressing upon the optic chiasma interferes with the visual pathways.

The following cases will illustrate the conditions associated with amaurosis in infants, which I have attempted to describe in this lecture.

Mental Deficiency.

CASE 1.—Female infant. Was first brought up to hospital in 1906 because the parents thought that she had defective vision. She was prematurely born at the seventh month, and suffered from fits for the first two years of life. Fixation was very doubtful, and though the discs were noted as being suggestively pale, yet it was considered that there was not sufficient in the fundi to account for the defective vision.

She was seen again in February, 1914, when 9 years old. "Is an obvious idiot, does not seem to see, though on occasions appears to notice things. Can talk well but without sense. Lower limbs completely spastic, cannot sit up alone, no control of sphincters. Is quite a baby in her habits, sucks a teat, gets into severe tempers." The pupils reacted readily to light. The eyes were markedly divergent. The discs showed temporal pallor, but the fundi were normal in other respects.

She was last seen in January, 1921. "General condition much as before, idiot, spastic flexed legs, cannot walk, rolls head frequently from side to side. Eyes divergent, pupils active to light, sees light and looks towards it sometimes."

CASE 2.—Female infant, when first seen 7 months of age. Was brought up because she "took no notice." She was one of twins at the mother's sixth confinement. The other twin died from "bronchitis"; rest of family alive and well. She has had "fits of spasms and cryings" since a fortnight old. General condition seems good, but she does not yet hold her head up. Eyes parallel, no nystagmus, pupils react to light. It is difficult to obtain fixation, except occasionally with a very bright light. Media of the eyes clear, and fundi normal.

She was next seen two months later: "Did not fix the light; the fits have now ceased; can only just hold her head up; movements of the limbs appear good; is sleeping better; no teeth yet." Two months later again, aged 11 months: "Fixation is obtained with a strong stimulus. It appears to take a stronger stimulus to evoke fixation than with a normal infant. Has now cut two teeth."

CASE 3.—Female infant, aged 8 months when first seen. "Mother thinks that she does not notice things. First child, no previous miscarriages, fits for first six months; these have now ceased, good general condition, moves limbs well, eyes straight, no nystagmus, pupils react, fixation difficult to obtain, if at all. Media clear, fundi show a slight pigment deficiency." Seen two months later: "Fixation obtained more easily, occasional convergence." Seen two months later: "Mother says that the child does not seem to see as well as other children of the same age, and is backward. Cannot sit up by herself yet, no teeth, does not crawl. Will fix and follow the light, but seems slow in making up her mind to do so. It would appear that a more powerful stimulus is needed to arouse her attention."

The first case has been followed to its conclusion. Of the second and third cases this can hardly be said, but I would point out that no satisfactory lesion in the eye can be found for the defective vision, and that the initial fits, the failure to hold the head up, to sit up and to crawl, and the late appearance of the teeth, are suggestive features. Further, the manner of the fixation of the light by the eyes, when obtained, is very characteristic. These infants have a curiously languid way of performing this act.

Pigment Deficiency.

CASE 4.—Male infant, aged 4 months when first seen. Was brought to hospital "because he took no notice and rubbed his eyes." "Seventh child; other children alive and well and have good eyes; all are very fair, as also is the father; normal, healthy infant in other respects; eyes parallel; slow horizontal nystagmus; pupils react to light; fixation difficult to evoke, if at all; media clear, fundi show marked pigment deficiency, large choroidal vessel shown up on white sclera as background."

Seen five months later; now definitely follows objects. Mother says that he does not like bright light. Nystagmus had ceased until a few days ago when he got a bad cold, since when it has returned.

Temporary Amaurosis.

CASE 5.—Female child, aged 18 months. Quite well until one month ago; then "screaming convulsions," after which the child was apparently blind. Felt for its bottle and did not see it, crawled round its cot and hit its head. When seen both pupils were large and reacted very fully to light; fundi showed no abnormality. Seen again a week later: Child now sees well, pupils normal size and react to light readily.

These cases are taken from the report of an investigation, carried out at the Royal London Ophthalmic Hospital by the Lang research scholars, which appeared in the *British Journal of Ophthalmology* for August.

BACTERIAL FOOD POISONING FROM MUTTON.

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To the medical officer of health, the pathologist, and, to a lesser degree, the general practitioner the sudden outbreaks of serious and sometimes fatal disease still commonly, but erroneously, referred to as "ptomaine poisoning" are always of great interest. Although more than one hundred such epidemics have been reported in this country, and although about 80 per cent. of these were due to flesh foods of various kinds,¹ no instance is recorded where mutton was proved to have been the food primarily responsible for the transmission of the disease. In May of this year, however, there occurred in Manchester a limited outbreak of food poisoning in which, in all probability, this relationship was present.

Summary of Chief Features of Outbreak.

Six separate households were affected, the number of individuals attacked being 25. Abdominal pain, vomiting, diarrhoea, and muscular cramps were present in most cases. There was often profuse sweating, and in a few cases fever and delirium were reported. One patient, D. G., aged 40, died on the third day of illness; in the other cases recovery took place after an illness of from about 24 hours to 7 or 8 days. The average period of incubation was found to have been from 17 to 20 hours.

The clinical diagnosis of bacillary food poisoning was rapidly confirmed by the isolation from the tissues and secretions of D. G.² of a bacillus of the food poisoning (Gaertner) group, further particulars of which are given later. Also, the blood of most of the individuals attacked agglutinated in relatively high dilution (1:250 in one case) both a known bacillus of the food poisoning group (*B. suispestifer*, type Mutton) and the bacillus isolated from D. G. Other organisms of the food poisoning group were agglutinated in lower dilution or not at all. Bacteriological examination of the faeces and urine from a few cases was negative.

All the individuals attacked had recently eaten some kind of meat food purchased from the same retailer. Four persons in one house became ill after eating "veal chops," but in every other case the illness followed the consumption of some form of mutton (leg, shoulder, breast of mutton; mutton chops). Six patients ate, in addition, some beef roasted in the fat in which some of the mutton was cooked. A notable negative instance was provided by the case of

¹ Savage; Food Poisoning and Food Infections, 1920.

² The public health authorities were first notified of this outbreak through the coroner's office.

a child aged 4 (suffering from measles) who alone, among seven inmates of one household, had no mutton and did not suffer from symptoms of food poisoning. Further evidence of a similar kind was obtained. The meat in question was said to have been of very good appearance and flavour, and in most cases to have been well cooked. In some instances it was consumed only when served hot.

The only part of this suspected food which could be obtained for examination was the remains of a leg of mutton of which nine of the persons affected had partaken. From different parts of this joint, though not from some parts, a bacillus was isolated which agreed in its cultural characters, its pathogenicity, and, so far as tested, serologically with the bacillus isolated from D. G. The carcass from which this leg of mutton was obtained was one of six bought by the retailer a few days before the commencement of the outbreak. These were imported frozen carcasses which, after passing through the hands of various dealers, were stated to have been received by the retailer in bags in a "bright, hard" condition.

A full investigation at the retail shop gave no evidence of past or present intestinal disorder amongst the butcher's family or staff nor of any other likely source of specific contamination. The only non-imported meat sold there for a month prior to the outbreak was veal from four calves bought at the Manchester meat market. These four carcasses were passed as sound, and, with the exceptions already mentioned, no cases of illness could be discovered amongst the purchasers of this veal.

Remarks.

Here, then, was an outbreak in which, first, the nature of the disease was ascertained beyond reasonable doubt. Secondly, the rôle of mutton as the agent responsible for the transmission of the infection, in most of the cases at any rate, was clearly indicated. Many food poisoning epidemics have been elucidated to this extent, but only rarely is it possible to determine the actual source and occasion of infection, and this outbreak is no exception to the general rule. How and when this mutton became contaminated was not shown. It is probable that the carcass was extensively infected, but it remains uncertain whether infection took place at the retailer's shop or was already present when he received the meat. In the first case, it is quite possible that the mutton became contaminated with infective agents derived from the veal. The evidence obtained, however, supported the reverse view, that the veal chops giving rise to disease were infected from the mutton.

As to the primary source of infection in this, as, indeed, in most outbreaks, nothing is known. In a few instances the dangerous state of the meat has resulted from disease in the food animal, but this explanation is contradicted in the present case by the bacteriological findings, for it may be assumed that under such circumstances all parts of the joint examined would have been uniformly invaded.

The bacillus isolated from the spleen of D. G. had the general cultural characters of the inositol fermenting food poisoning bacilli. It was not agglutinated by an enteritidis serum, and only to 125/5000 titre by a paratyphosus B serum. With a *suipestifer*, type Mutton, serum it was agglutinated to full titre (1:8800). A *suipestifer*, Newport, serum of titre 1:20,000 failed to agglutinate in low dilution. From a *suipestifer*, Mutton, serum the bacillus D. G. absorbed completely its own and the primary agglutinins, and, from a high power anti-serum prepared with the bacillus D. G., *B. suipestifer*, type Mutton, absorbed the agglutinins for itself and for the homologous organism in equal proportion.

In the course of these tests other facts of bacteriological interest were noted, which may be referred to at a later date elsewhere. It may, however, now be stated that the causal organism of this outbreak was a strain of *B. suipestifer* (or *aertrycke*), type Mutton, the organism most commonly responsible for food poisoning in man.

A CASE OF SPORADIC INFECTION BY THE *BACILLUS AERTRYCKE*.

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SINCE the original *aertrycke* epidemic, described by De Nobele¹ in 1899, cases of infection by organisms similar to the one then found, with fully recorded post-mortem notes, appear to be rare. The work done by Boycott,² Bainbridge,³ and H. de Morgan⁴ gives us a very valuable survey of the bacteriology of the paratyphoid group, but no information is given by these investigators on the post-mortem changes due to the *Aertrycke* bacillus. Slight reference is made to two post-mortem cases by Perry and Tidy,⁵ in which they isolated *B. aertrycke* from the gall-bladder and from the spleen in both cases. Bainbridge⁶ recorded a case of *B. suipestifer* infection in which the organisms isolated never formed gas in any carbohydrate medium, and a similar strain was isolated and described by Bock.⁷ The following case, in which *B. aertrycke* was isolated, is therefore of some interest.

Clinical History.—A boy, aged 3 years, was admitted to hospital on July 24th, 1920, with a history of suddenly becoming ill on July 17th. The symptoms complained of were diarrhoea and vomiting. On admittance the child looked pale and anæmic, and was in a state of collapse. No further history could be obtained. The child rapidly became worse and died on the same day.

Post-Mortem.—There were numerous petechiæ on the body surface. The lymphatic glands were enlarged generally. The various serous surfaces showed numerous petechiæ, whilst the mucous membrane of the bladder also showed the presence of numerous small hæmorrhages. The lungs showed extensive changes. Marked broncho-pneumonia was present in the lower lobe of the right lung, the upper and middle lobes being distinctly oedematous. The upper lobe of the left lung was also oedematous and collapsed, the lower lobe being stretched over and adherent to the diaphragm. The spleen was found to be lying in an abscess cavity, the upper boundary of which was formed by the diaphragm. This cavity contained three-quarters of a pint of pus of café au lait colour. The upper three-quarters of the swollen spleen was necrotic and lying in pus, whilst from its anterior aspect pyogenic membrane swept forwards and upwards to complete the abscess walls. The tip of the spleen projected downwards outside the cavity and contained a white infarct. The intestines showed changes in the Peyer's patches and solitary lymph follicles, which were firm, swollen, pigmented, and somewhat worm-eaten in appearance. These solitary follicles also showed irregular depressions at their centres, the appearances suggesting a healing enteritis. Numerous white infarcts were present in the kidneys.

Bacteriology.—Broth cultures were obtained from the contents of the splenic pulp and from the gall-bladder. The Gram-negative actively motile bacilli isolated in pure culture from both sources gave the following cultural characteristics: A moist, white, semi-translucent growth on agar, practically odourless; uniform turbidity in broth; acid, and after some days, a faint trace of gas, in glucose, maltose, and mannite; lactose and saccharose unaffected; litmus milk, at first acid, finally and permanently alkaline; gelatin not liquefied, and no indol produced. The bacilli, when first isolated, could not be agglutinated by specific sera for *B. typhosus*, *B. paratyphosus* A, *B. paratyphosus* B, *B. enteritidis* of Gaertner, or by a weak human serum obtained from another case of *aertrycke* infection (strain unknown).

Repeated subcultures were made over a period of three months, during which time the organisms gave unmistakable signs of gas production in glucose, maltose, and mannite, though lactose and saccharose were still unaffected. The volume of gas formed gradually increased in the earlier successive subcultures. At this period agglutination experiments still gave negative results, but 12 months after isolation, when a series of experiments were carried out with Dreyer's technique,⁸ positive results appeared. For this purpose standard killed cultures of *B. paratyphosus* A, *B. paratyphosus* B, *B. enteritidis* of