made from tube A showed numerous colonies of bacillus coli and numerous blue colonies, easily detected, for in many places they were quite separate and isolated; these had all the appearances of colonies of bacillus typhosus: in the hanging drop they were typical, when tested with typhoid serum in dilution 1 in 100 clumping was instantaneous. Subcultures in the different media proved them to be bacillus typhosus. The Drigalski plate made from tube B (1 in 1650 malachite green) exhibited crowds of blue colonies (bacillus typhosus) and a very limited number of red colonies with red halo—i.e., bacillus coli communis.

So that in this experiment the great number of bacillus coli present in the original fluid (at least 100,000 per 1 cubic centimetre) did not interfere with the multiplication of the added bacillus typhosus; in fact, in the tube B (1 in 1650 malachite green) it was by far the predominating growth.

Experiment 4.—To 10 cubic centimetres of domestic sewage (at least 100,000 bacillus coli communis per 1 cubic centimetre) added a trace of bacillus typhosus. 10 cubic centimetres of distilled water to which the same amount of bacillus typhosus culture had been added showed just the faintest sign of turbidity; so that the amount of bacillus typhosus added to the 10 cubic centimetres of sewage was indeed very small. With a loop of the sewage typhoid mixture inoculated one tube (A) bile salt broth malachite green 1 in 1600, and another tube (B) bile salt broth malachite green 1 in 1500. Incubated for 24 hours at 37° C. tube A shows good growth with gas, tube B seems to have little if any growth. Made Drigalski plates with one loop from each tube. The result was this: plate from tube A (malachite green 1 in 1600) shows numerous colonies of bacillus coli (red with red halo); plate from tube B (malachite green 1 in 1500) has 21 (blue) colonies of bacillus typhosus, and about 50 to 60 small violet colonies (streptococci), but no red colonies of bacillus coli. The blue colonies of plate B were tested by agglutination, in hanging drop, and in the other media—gelatin, MacConkey fluid, neutral red broth, litmus milk, lactose-peptone water, and proved typical bacillus typhosus.

In this experiment, then, from sewage containing a mere trace of bacillus typhosus the enrichment of this microbe in, and recovery from, bile salt broth malachite green 1 in 1500 was easy, but it did not succeed from bile salt broth malachite green 1 in 1600.

Experiment 5.—The semi-fluid stool of a case of typhoid fever about the end of the third week (a female patient in Elizabeth Ward of St. Bartholomew's Hospital) was used thus: one loop of this stool was planted in bile salt broth malachite green 1 in 1600 (tube A), and one loop in bile salt malachite green 1 in 1500 (tube B). After 24 hours' incubation at 37° C. both tubes show growth. With one loop of each made a large Drigalski plate and incubated at 37° C. After 24 hours plate from tube A shows amongst a large number of neutral large colonies smaller blue colonies, easily recognised by their colour and aspect as those of bacillus typhosus. In hanging drop cylindrical motile bacilli clump instantly with typhoid serum in dilutions of 1 in 50 and 1 in 100; in addition to these in the marginal part of the plate some red colonies with red halo (bacillus coli) could be recognised. Plate from tube B contains no red colonies; only some neutral large colonies and numerous blue colonies of bacillus typhosus. Subcultures were made of the blue colonies of both plates and complete proof was obtained that we were dealing with the true bacillus typhosus.

From this experiment it is evident that in this case the bile-salt broth malachite green was of decided value.

Experiment 6.—Milk was bought in a small shop; it contained about 1000 bacillus coli communis per 1 cubic centimetre. To 100 cubic centimetres of this milk bile salt (sodium taurocholate) was added in the same proportion as in the former experiments, and malachite green in the proportion of 1 malachite green in 1500. Then to 10 cubic centimetres of sterile water added one loop (2 milligrammes) of a typhoid culture; the resulting emulsion was very faintly turbid. Of this fluid added one loop (2 milligrammes) to the above 100 cubic centimetres of milk; this was then incubated at 37° C. After 24 hours' incubation, plated on Drigalski medium one loop of the infected milk. This Drigalski plate on inspection after 24 hours' incubation showed, amongst neutral colonies, a large number of small blue colonies of the appearance of typhoid colonies. In the hanging drop and in agglutination test they complied with those of bacillus typhosus. Subcultures were made and their identity with bacillus typhosus was established.

Here, then, the proof of enrichment of bacillus typhosus in the bile-salt malachite-green milk (1 in 1500) was shown in a decided manner; there was no enrichment of bacillus coli, although the original milk was rich in this latter microbe.

Experiment 7.—This experiment is of special interest as it shows that there is no difficulty in recovering by this method the bacillus typhosus from specifically infected oysters. 12 native oysters (bought in a small shop) were placed in a tank containing 3000 cubic centimetres of sea water, as delivered by the Great Eastern Railway Company (Lowestoft). To 10 cubic centimetres of domestic sewage (St. Bartholomew's Hospital), containing at least 100,000 bacillus coli communis per 1 cubic centimetre, one loop (2 milligrammes) of typhoid culture was added, well shaken, and poured into the tank and well distributed in the 3000 cubic centimetres of sea water. The oysters were quite fresh and active, since within a quarter of an hour they all opened, emitting plenty of air bubbles. Next day six oysters that were found well closed were taken out, well brushed and washed under the tap, and then opened. After draining off the liquor within the shell as much as possible the bodies were placed into a sterile dish and well minced. The total amount of turbid fluid obtained from the minced material of the six oysters was about 12 cubic centimetres. Three tubes, each containing 10 cubic centimetres of bile-salt broth malachite green (1 in 1500), received each $\frac{1}{4}$ cubic centimetre of the above oyster fluid and were then incubated at 37° C. Next day all showed good growth. Of each of these tubes one large Drigalski plate was made

with one loop and incubated at 37° C. After 24 hours one of these plates (No. 1) showed numerous blue colonies of the nature and appearance of typhoid colonies; the other two plates (Nos. 2 and 3) were negative quâ-typhoid colonies. Of Plate 1 several of the blue colonies were selected for examination in the hanging drop and for agglutination test; both tests positive for bacillus typhosus. Subcultures fully confirmed the preliminary diagnosis.

We see, then, that a positive enrichment of the bacillus typhosus taken up by the six oysters was effected in one out of three bile-salt broth malachite-green tubes, which was quite sufficient for identification.

St. Bartholomew's Hospital.

A FATAL CASE OF CHOREA ASSOCIATED WITH DOUBLE OPTIC NEURITIS AND HYPERPYREXIA IN A CHILD, AGED THREE AND A HALF YEARS.

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At a meeting of the Society for the Study of Disease in Children held on Nov. 15th last the notes of a case of rheumatic hyperpyrexia occurring in a child, aged six years, were read by Dr. G. H. Lock. The extreme rarity of rheumatic hyperpyrexia occurring in one so young was commented upon, as also the paucity of the literature in relation to that complication. In the discussion that followed I related that I also had had experience of such a complication in a child even younger still—a girl, aged three and a half years, with chorea, which I had not yet published, but that in the interim I would look up the notes and report to the society at its next meeting. As rheumatic hyperpyrexia is so exceedingly rare in the young the following notes of the case may prove of general interest as well as to the members of the Society for the Study of Disease in Children, and I therefore take this opportunity of placing it on record. During an experience of over 20 years of children's ailments this is the solitary example of rheumatic hyperpyrexia that has come under my notice. At the time of the fatal onset the child was not obviously suffering from rheumatic fever, but her illness commenced with that complaint; it was followed a fortnight after by chorea, and when I saw her she had mitral disease and rheumatic nodules. One can therefore with some show of reason attribute the hyperpyrexia to a fatal and additional dose of the rheumatic poison.

A female, aged three and a half years, some six weeks before I saw her, which was in June, 1890, suffered from swollen hands and feet. 14 days after this attack she developed choreic movements which were general. When I saw her she spoke with difficulty and the movements were most marked in the face. They were much worse when she was being watched. She had a mitral bruit and there were numerous subcutaneous nodules of the size of a bean on the back of the head and about the ankles and feet. The temperature was 100° F. A full diet was ordered together with two minims of Fowler's solution three times daily, to be increased by drop doses every other day. On July 10th the arsenic mixture, which had reached ten minim doses, was stopped because of conjunctival irritation and a discharge from the eyes. By August 2nd she had wasted considerably and there was pityriasis tabescentium. The subcutaneous fibrous nodules had disappeared. The mitral bruit was then very harsh and distinct. There was double optic neuritis. She was taking her food badly and was languid and drowsy, but the choreic movements were less. Her morning temperature on this date, which had been previously subnormal, was found to be 101·6° and by midnight it had reached 106°. She was quite conscious until 4 P.M. on the 2nd when she became delirious. The movements at this time were greatly augmented. The face was flushed and the skin was hot and dry. A three-grain dose of antifebrin was given at 7 P.M., repeated at 9.40 P.M. and again at 12 midnight. Apart from the production of much sweating at the back of the neck the drug did not materially alter the condition. At 1 A.M. on the following day the temperature had fallen a little more than 2°, but quickly ran up to 106·4° at 2 A.M., when she was sponged, which reduced the temperature to 103·8°. During this period the breathing was rapid, the face was

livid, and the lips were blue, and she swallowed with difficulty. She died at 4 in the morning.

Necropsy.—There was slight thickening of the mitral valve. Peyer's patches were injected but the intestines were otherwise quite normal. The brain was normal.

Welbeck-street, W.

IS BUBONIC PLAGUE STILL LURKING IN THE CITY OF GLASGOW?

By THOMAS COLVIN, M.D. GLASG.,

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IN August, 1900, the inhabitants of Glasgow were startled by the announcement that plague had appeared in their midst after an absence of 235 years from the shores of the British Isles. The first detected cases were three patients whom I sent into Belvidere, the city hospital for infectious diseases, from 57, Thistle-street, on the south side of the river Clyde, with a provisional diagnosis of an acute infectious malady simulating enteric fever. These cases were ultimately proven to be clinically, pathologically, bacteriologically, and experimentally bubonic plague. In all 36 cases were traced and of these 16 died, which gave a mortality of 44·4 per cent. The last patient was discharged from Belvidere on Nov. 3rd, 1900, and ten days later Glasgow was declared free from plague. The origin of the outbreak was never discovered and the rats which infested the area where the disease appeared were on examination found free from plague.

A year later, in August, 1901, two cases, which were no doubt plague, occurred in the plague-infected area of the previous year. For a clinical note of these cases I am indebted to Mr. Robert Murdoch. He states that on August 23rd, 1901, he was called to see a boy, aged 12 years, in Crown-street, who was extremely ill, with a febrile temperature, and a painful swelling in his groin. No wounds or abrasions of any kind were seen on the boy's leg to account for the bubo. Two days later the boy's father took suddenly ill with the symptoms of an acute pneumonia only with this characteristic difference—that instead of the usual rusty sputum of simple pneumonia he coughed up at intervals pure blood. He died suddenly after two days' illness. Mr. Murdoch, becoming suspicious of plague, consulted Dr. John Stewart and both gentlemen were of opinion that the boy had bubonic plague and that the father had died from pneumonic plague. Dr. A. K. Chalmers, the medical officer of health, was at once communicated with and the boy and the dead body of his father were removed to Belvidere. Every one who was known to be in contact with these cases was removed to the sanitary reception house, and the man's house and his rag-store, where the infection was presumed to have been caught, were disinfected in the same manner as was done the previous year in plague cases.

It was a knowledge of these two cases which I only learnt incidentally from Dr. Stewart many months after their occurrence that gave me the key to the solution of the origin of an outbreak of plague in Liverpool that happened two months later, or in October, 1901. At the time I was deeply interested in this outbreak, for I was asked by Dr. Chalmers to give him a report of an illness of a patient of mine, in whose house two Liverpool ladies had stayed for three days and on their return home sickened with plague, one of them dying from that disease. I have carefully studied every known fact connected with this outbreak, including the official report for which I am indebted to Dr. E. W. Hope of Liverpool, and including also correspondence with the young lady, who is still in Liverpool, who unconsciously carried the infection from Glasgow into that city, and I now submit evidence that is strongly presumptive that the contagion was conveyed from the one place to the other by infected clothing.

On August 15th, 1901, I was called to see a young woman who had an indefinite illness. She had nausea and vomiting, a febrile temperature, and extreme prostration, but the only definite symptom was an acute pain in the left iliac region. Even to this day she is certain of the site of the pain, for among other remedies tried to soothe the pain was a fly-blister applied over its region. She had no buboes nor did she complain of pain in her axillæ or in her groins. There was no vaginal discharge and on bimanual examination

nothing definite could be made out. The only diagnosis that I could make at the time was an acute ovaritis or some obscure uterine affection. In reviewing that diagnosis in the light of Mr. Murdoch's cases the following most suggestive facts are to be noted. 1. An isolated case of plague occurring in a city which is supposed to be free from plague would never suggest plague when no apparent buboes are present. It is the association of two or more cases that puts the medical man on the alert. 2. This case occurred in Adelphi-street, about 100 yards from Thistle-street, and in the plague-infected area of the previous year. 3. It occurred about the same time as Mr. Murdoch's cases in the latter part of August. 4. Acute ovaritis according to Galabin and other writers is a very rare affection, and as a rule follows septic mischief after delivery or abortion or gonorrhoea. My patient had not the slightest evidence of any of these three conditions. Moreover, she was the very antithesis of those neurotics who often complain of ovarian pain, for she never had a day's illness before, unless during confinement, nor has she ever been ill since and is at present in excellent health. 5. Simpson states that the chain of glands and lymphatics in the abdominal cavity may be affected and form a painful iliac bubo which is tender to pressure. If on the right side the condition may be mistaken for acute appendicitis. The iliac bubo may occur without any apparent enlargement of the inguinal glands. The previous history of this case, the clinical symptoms, and the after-history all point to an iliac bubo and not to acute ovaritis.

Now it was during the week that my patient was ill that her two friends from Liverpool stayed three days with her. Although they did not occupy the same bedroom, for there were five apartments in the house, they were in most intimate contact with the patient. On Sept. 21st, or about four weeks later, their mother sickened and died from plague after an illness of seven days with buboes in her axillæ. On Sept. 22nd one of the girls sickened and died from plague nine days later with axillary buboes. On Sept. 24th the other girl sickened with plague with a bubo in her groin and she recovered. A woman who assisted in laying out the mother's body also died from plague, while four children living in an adjoining house sickened with plague, three of whom died. Now as the mother was never in Glasgow and she was the first to sicken the infection was indirect, and direct infection was also excluded by the date of sickening, for the average maximum incubation period of plague is ten days. But the mother superintended the washing and laying aside of the clothes worn in Glasgow and thus caught the infection, and having evidently developed a virulent form of the disease infected her two daughters.

In proof of the contagiousness of the clothing I elicited a most important fact. Between the sickening of these three cases and the sickening of the four children nearly a month had elapsed, so here again the infection was indirect. But the very week that two of these children sickened their mother was wearing a blouse that had been given to her by one of the girls who had been to Glasgow, for the girl's mother being dead and the blouse being of a bright colour she could not wear it herself, for she was in mourning. The last time this blouse was worn by the girl was in Glasgow when in immediate contact with her friend, who was ill presumably with plague, for the blouse was never worn by her after she sickened with plague on account of her mother's death. I made strict inquiries whether the blouse had been washed or cleaned before being worn by the mother of the children and received a negative reply, for the blouse was silk and a new one and only worn in Glasgow.

Cantlie relates that plague in 1900 was introduced into Durban from Mauritius in a way similar to what I have described. A family of six came from a plague-infected part in Mauritius into Durban. Two months after their arrival one of their sons opened a deal box which contained some soiled linen that the family had brought with them which had not been previously opened and the lad died two days later from bubonic plague. Simpson also relates many cases of clothing being a carrier of plague and many of the cases of plague in China were traced to the practice of the Chinese wearing the clothes of those who had died from that disease. One of the cases in the Glasgow outbreak in 1900 was the wife of a man who was employed in Belvidere in disinfecting and removing clothes from plague houses. As this woman lived fully a mile from the plague-infected area and had no association with any known plague