doubt, and I fear there is not much ground at present for relaxation of the usual strict inquiries on this point. In spite of temperance societies and of efforts to educate the public mind, a far too large habitual consumption of fermented liquors, and especially of the strong whisky spirit, still goes on. This constitutes a continual menace to the acceptors of life risks. How great may well be roughly estimated from the favourable reports of those offices which have formed a separate department for abstaining insurers.

Probably more of the disputed cases in the event of early death are in some way connected with concealment of intemperate habits than with all other causes put together. We all know that excess of alcohol destroys the moral sense, and so prepares the mind of an intemperate proposer to make statements which are not justified by actual facts. As medical examiners we are bound to ascertain pretty closely and report the daily habitual consumption of these alcoholic drinks, always remembering that it is not an occasional actual intoxication that produces the greatest deterioration of life, but that frequent taking of alcohol during the day, in whatever form disguised, which is now so common. The sure result is disease or degeneration of the nervous tissues or other organs, and the shortening of the life-expectation proper to the special age, and on which the normal premiums are based.

CONCLUSION.

As we all know, several excellent and most reliable works upon life assurance have been published during the last half-century, notably by Drs. Brinton, Sieveking, S. Thompson, and (quite recently) de Havilland Hall. These are all of great value, and are at present our guides and counsellors in considering the various points, the sum of which regulates our opinions and valuations of the lives submitted for examination.

Somewhat modified views of many insurance risks are already acted on by all the best offices. But in the present times facts of to-day may require tomorrow to be reconsidered by the light of fresh knowledge, and I think I have brought before our Society enough to show that no finality has yet been arrived at in this department of medical science, but that, on the contrary, we shall ere long still further modify many of our views as to the value of diseased lives; and that still increased and more thorough knowledge of many diseased conditions will elevate the practice of insurance of invalid or defective lives into something more like exact science than has hitherto been at all possible or practicable.

Still, at present the only safe way for offices to which invalid risks are offered, is to proceed slowly and cautiously. The security of these offices, and with them the interests of their insurers, are closely bound up in a proper and sufficient estimation of the risks incurred. But, even so, I would repeat that there seems to be good reason to hope before long for at least a more modified view as to the absolute or comparative non-insurability of many of the lives now customarily rejected on account of present morbid conditions or proclivities.

I have thus been able to touch upon only a few of the great medical problems which await the future consideration of doctors, actuaries, and all others engaged in the great business and science of life assurance. Any changes which can be made must necessarily be most carefully thought out, and most discriminatingly acted upon. My purpose to-day will have been answered if I have succeeded in directing your attention, however imperfectly, to some of the great issues with which hereafter we, in our several vocations and localisations, shall necessarily have to deal, and the consideration of which is being continually forced upon our attention.

NOTES AND REFERENCE.

¹ See further and similar statistics given at the meeting at Marlborough House on December 20th, 1898, since this paper was written, in the *BRITISH MEDICAL JOURNAL*, December 24th, 1898. ² But it would appear to be not yet proven that these are the only sources of infection. Dr. Manfredi has recently shown the existence of tubercle bacilli in the dust of some of the streets in Naples. ³ *Lancet*, October 8th, 1898.

MEDICAL MAGISTRATE.—The Lord Chancellor has added the name of Dr. West Symes, Senior Surgeon to the Royal Halifax Infirmary, to the Commission of the Peace for the County Borough of Halifax.

A CASE OF TYPHOID FEVER WITHOUT ANY LESION OF THE INTESTINE,

WHICH GAVE THE WIDAL REACTION DURING LIFE, AND FROM WHICH THE *BACILLUS TYPHI ABDOMINALIS* WAS OBTAINED BY CULTURE FROM THE ENLARGED MESENTERIC GLANDS FOUND AT THE NECROPSY.

By J. H. BRYANT, M.D., M.R.C.P.,

Assistant Physician and Demonstrator of Pathology to Guy's Hospital.

I PUBLISH this important case, and give a brief review of the few similar cases which have been reported on account of their rarity, as an important vindication of the great value of a positive Widal reaction in the diagnosis of typhoid fever and to show the necessity of making careful bacteriological examinations at all necropsies in which the slightest doubt arises as to the nature of the disease which has caused the patient's death. Necropsies in this country, in the majority of instances, are performed in a most perfunctory manner, a macroscopical examination only of the viscera often being considered sufficient. In this way many points of interest are lost, many erroneous conclusions are deduced, and many opportunities of advancing our knowledge of the causes and nature of disease by research are missed.

With our present imperfect knowledge of the pathological changes produced in the body by the action of micro-organisms, I maintain that a systematic bacteriological examination of the diseased structures should be made in all cases. If cultures had not been taken from the enlarged mesenteric glands in the case reported below, the cause of death would have been assigned in all probability to the broncho-pneumonia, and the diagnostic value of the serum reaction as an indication of typhoid fever would have been depreciated. I am indebted to Dr. Hale White for permission to publish the clinical notes of the case.

Male, aged 1 year and 9 months, was admitted on September 20th, 1898, for diarrhoea and vomiting. The mother stated that previously he had always been healthy. For the first nine months she fed him from the breast, since then on cow's milk, bread and butter, mutton broth, vegetables, and occasionally boiled fish. For some time previous to admission the child was always very thirsty, and in consequence was given a large quantity of milk. In July wasting was first noticed.

Family History.—Father healthy, mother healthy but delicate before marriage; she has had six children and a miscarriage at three months. The six children were all full term. One died of convulsions, aged 7 weeks; another of pneumonia when 7 months old. One sister, aged 7 years, was admitted into Miriam Ward on September 6th, and a brother, aged 5, into Stephen Ward on September 23rd, both suffering from a similar disease to that of the patient.

Present Illness.—Two weeks previous to admission diarrhoea and vomiting came on. A doctor was called in, and the symptoms were relieved by medicine. On Sunday, September 17th, the mother stated that the child appeared to be quite well; on the 18th diarrhoea and vomiting again commenced, but this time the symptoms did not yield to treatment. On the 19th, as the child was worse, he was brought to the hospital and admitted.

Condition on Admission.—Temperature 103.8°, pulse 136, respiration 48. He was very anæmic and wasted, and was noticed to be lying on his side with his legs and head flexed on the trunk. The abdomen was slightly distended and a little rigid. The edge of the spleen could be felt 2 inches below the costal margin. The edge of the liver could also be felt just below the costal margin in the right nipple line. The areas of liver and splenic dullness were increased downwards.

Respiratory System.—A small patch of impaired resonance was found at the right base behind, just below the angle of the scapula. *Râles* and rhonchi were heard all over the chest behind on the right side; over the area of impaired resonance the *râles* were slightly consonating in character. The heart sounds were normal.

October 20th to 24th. The head still remained flexed on the chest. The child was drowsy. *Râles* and rhonchi were still heard over the back. The highest temperature was at 2 A.M. and 6 A.M. on October 21st, when it reached 105.2°, the pulse-rate being 150 to 160, and the respiration-rate 48. The bowels were opened six times on the 21st, seven times on the 22nd, six times on the 23rd and 24th, and ten times on the 25th.

On October 26th the child was a good deal worse, the diarrhoea was very bad, the bowels were opened eleven times, the motions being of a greenish colour and of watery consistence. The temperature again reached 105.2°, the pulse-rate being 160, and the respiration-rate 60.

On October 27th the child was much worse, the bowels were opened nine times, and he was sick all day, being unable to keep anything at all down. The abdomen was very distended. The highest temperature was 104.8° at 10 A.M., the pulse being 150, and the respirations 56. Dr. Washbourn examined the patient and diagnosed typhoid fever. A specimen of blood was subsequently obtained from the patient, and it gave the Widal test, a very smart reaction being obtained both with 5 per cent. and 50 per cent. serum in two minutes. In the evening he became worse, and died at 8 P.M.

Necropsy.—Eighteen hours after death rigor mortis was well marked. There were no signs of decomposition. The body was thin and emaciated. The brain was not examined. There was recent pleurisy over the right

lower lobe and considerable subpleural ecchymosis over the posterior surface of the left upper, and upper part of the left lower, lobes. Projecting from the posterior and outer surfaces of both lungs were numerous yellowish-looking nodules of consolidation, surrounded by hæmorrhagic rings, and varying in size from $\frac{1}{4}$ to $\frac{1}{2}$ inch in diameter. On section they were seen to be early patches of septic broncho-pneumonia; it was expected on cutting into them to find that they were small pyæmic abscesses; they had not, however, broken down.

The heart weighed $1\frac{1}{2}$ oz., and appeared to be normal. The arteries, mouth, pharynx, œsophagus, and stomach were normal in appearance. The ileum also appeared to be normal. There was no ulceration, and the Feyer's patches were not swollen or discoloured. Nowhere in the intestine could any sign of recent typhoid ulceration be found, and there was not any appearance suggesting a healing or healed typhoid ulcer. The peritoneum was normal. The liver weighed 16 ozs., and had a normal appearance. The gall bladder and pancreas were normal. The mesenteric glands were much enlarged, and felt very soft; on section they presented a pinkish-grey colour, and appeared to be in a condition of acute inflammation; there was no sign of suppuration or caseation in any of them. The suprarenal capsules were normal. The kidneys weighed 3 ozs.; they were pale. The spleen was a little enlarged.

BACTERIOLOGICAL EXAMINATION.

Cultivations were made from a mass of the enlarged mesenteric glands, a broth tube and agar slant being inoculated from the centre of one of the glands, all possible precautions being taken to prevent any accidental contamination. The broth was kept in an incubator at a temperature of 37°C . In 24 hours it became quite cloudy and turbid. Motile bacilli with rounded ends, the majority being two or three times as long as broad, were found, also a few short oval forms and some very long threads.

On agar, after 24 hours at 37°C ., a profuse whitish semitransparent growth appeared. A good deal of growth was noticed also in the condensed fluid in the bottom of the tube. Microscopically bacilli were seen similar to those from the broth culture, and they were found to be very motile. They stained readily with methylene blue, but did not retain the stain when treated by Gram's method.

Litmus milk was inoculated from the broth and agar cultures. In 24 hours it had changed to a lilac colour; in 48 hours it had changed to a pink colour and had slightly coagulated.

Gelatine plates were prepared from the broth culture. In 24 hours there was no visible growth; in 48 hours small indistinct colonies were just visible but were difficult to distinguish.

In 72 hours: Plate 1. A large number of small translucent greyish or bluish-white colonies were seen. Plate 2. Fewer but larger colonies similar in colour seen. Plate 3. Still fewer but larger colonies, similar in appearance with rather irregular borders; no liquefaction of the gelatine in any of the plates; no other colonies seen.

An agar culture was made from some of the bluish-white colonies; these colonies examined microscopically showed bacilli similar in character and form to those above described. After 24 hours at 37°C the agar showed on its surface a profuse growth of raised greyish-white colonies.

A gelatine stab was made from the agar culture, and after 24 hours at 30°C . a few white colonies were seen on the surface of the stab and along the upper part of it. No gas and no liquefaction of the gelatine took place in 24 hours or subsequently.

A gelatine shake was also made. A slight clouding of the gelatine from the growth of innumerable small colonies was found after 24 hours. At the end of 48 hours the gelatine was very cloudy throughout and the

colonies were more visible. No liquefaction and no formation of gas took place.

A broth culture was made from the plates. After 24 hours at 37°C it was cloudy. It did not give an indol reaction. Motile bacilli similar to those described above were found.

A glycerine agar culture at the end of 24 hours at 37°C showed considerable growth of raised white colonies, and at the end of 48 hours the growth had increased.

Litmus milk was inoculated from the agar culture. In 24 hours a slight change in colour was noticed; in 48 hours a lilac colour was produced. No coagulation occurred, and after several days the lilac colour still remained.

Potato inoculated from the agar culture after 24 hours at 37°C showed a line of moisture along the inoculation streak. A coverglass preparation stained with methylene blue showed bacilli similar to those described above.

Gelatine and Glycocholate of Soda.—After 24 hours at 20°C a slight colourless shiny growth was visible. Examined on the fourth day showed a slight increase in the growth, which was still colourless and transparent-looking. Coverglass preparations stained with methylene blue showed long threads and numerous shorter bacilli with rounded ends and some smaller oval bacilli.

A broth culture was grown anaerobically at 37°C , and at the end of 24 hours the broth was cloudy; there was no gas formation. Microscopically very motile bacilli were found, some with rounded ends two or three times as long as broad, some oval, some long threads, some arranged longitudinally in pairs, and some showed vacuolation in the centre. A drop of 5 per cent. serum from a case of typhoid fever was added to a hanging drop obtained from this broth culture and marked clumping of the bacilli was produced in a few minutes.

Another broth culture was taken from the above, and in 24 hours the broth became cloudy. A hanging drop showed motile bacilli. Two of the hanging drops were treated with 50 per cent. and 5 per cent. serum respectively obtained from an immunised rabbit. The 50 per cent. caused clumping of the bacilli in about 5 minutes, and the 5 per cent. in about 10 to 15 minutes.

A culture of typhoid bacilli was treated in a similar manner with some serum obtained from the same sources, and in a very short time clumping occurred both with the 50 per cent. and with the 5 per cent.

REMARKS.

It will be seen from the above account of the case that on the afternoon preceding death Dr. Washbourn diagnosed the patient to be suffering from typhoid fever. The diagnosis was made partly from a consideration of the clinical features, namely, the continuous character of the pyrexia, the diarrhoea, distended abdomen, and enlargement of the spleen, and partly from the fact that his brother, who was lying in Stephen Ward, was similarly affected, and because his blood had given the Widal reaction. A specimen of blood was obtained from the patient after Dr. Washbourn's visit, and the serum which separated gave a very smart reaction, and it was so characteristic that Mr. Pakes, the Demonstrator of Bacteriology to the hospital, expressed a very decided opinion that the patient was suffering from typhoid fever. It was thought

that the illness had commenced about fourteen days previous to admission, so that death was considered to have taken place about the end of the third week from the onset.

Nothing unusual was anticipated before the necropsy took place. It was expected that the usual typical ulceration of the Peyer's patches of the lower part of the ileum would be found, and great surprise was expressed when no swelling, discoloration, ulceration, or other abnormalities whatsoever could be detected in the Peyer's patches, solitary glands, or mucous membrane of any part of the intestine. I thought at first an erroneous diagnosis had been made, and suggested that the symptoms might have been accounted for by the broncho-pneumonia which was found, although the character of the pyrexia was against this view. After finding the enlarged mesenteric glands, I suggested that, after all, it was most probably an anomalous case of typhoid fever without any lesion of the intestinal mucous membrane. Cultures from the enlarged mesenteric glands yielded an almost pure culture of the bacillus typhi abdominalis. The slight clotting of the milk inoculated from the first broth culture taken directly from the glands was probably due to a slight contamination with the bacillus coli communis. It will be noticed that coagulation did not take place until after forty-eight hours and then it was only slight. I could not find any colonies of the bacillus coli communis on the gelatine plates, although I looked and carefully examined for them, so that if present originally the number must have been insignificant. The bacillus obtained from the gelatine plates gave the characteristic positive and negative reactions of the bacillus typhi abdominalis—namely, did not produce gas in any media, did not cause milk to clot, did not produce indol, did not produce acid, did not liquefy gelatine, and further these bacilli obtained from a recent culture and treated with both 50 per cent. and 5 per cent. serum from a typhoid patient, and also from an immunised rabbit, clumped together in a manner characteristic of the bacillus typhi abdominalis.

The occurrence of typhoid fever without the characteristic intestinal lesions has been recognised for some time, and the chief credit for drawing attention to the importance of this condition is due to Osler. In the last edition of his *Principles and Practice of Medicine* he writes, "The wide existence of the typhoid bacilli has been repeatedly shown in cases which had the clinical feature of the disease, but without lesions in the small intestine. Typhoid fever is no more primarily intestinal than is small-pox primarily a cutaneous disease." He divides cases of typhoid fever into four groups:

1. Ordinary typhoid fever with marked enteric lesions.
2. Typhoid septicæmia, a general infection with the bacilli without special local manifestations.
3. Typhoid fever with localisations other than enteric—for example, lesions of the lungs, spleen, kidneys, and cerebrospinal meninges may exist with very slight or without intestinal lesions.
4. Mixed infections.

Brunschwig (1870), Litten, Moore, Church, Coupland, Phillips, and Goodall have recorded cases of typhoid fever without intestinal lesions, but as the diagnosis of these cases rested chiefly on the clinical evidence and the conclusions were not verified by bacteriological examinations, they are of little scientific value. Some of these cases were reported before the discovery of the bacillus typhi abdominalis, and it is therefore interesting to note that the possibility of such anomalous forms of typhoid fever was noticed so long ago. In future, as Chiari points out, the diagnosis of such cases must rest on the presence of the serum reaction and the demonstration of the bacilli.

Banti in 1887 described a case. The patient was a woman, aged 51. During life the usual clinical features of typhoid fever were noticed. Death took place on the twenty-eighth day of the disease. At the necropsy no lesion of the intestine was found, but the spleen and mesenteric glands were swollen. A bacillus morphologically resembling the bacillus typhi abdominalis was found in microscopical examination of sections of the spleen and mesenteric glands, but the bacillus was not identified by its cultural reactions, so that the evidence of the micro-organism being the bacillus typhi abdominalis is not convincing. This is the first case of the kind in which the bacillus typhi abdominalis is alleged to have been found.

Thue in 1889 described a case in which during life the fever was of a recurrent type, and the spleen was found to be considerably enlarged. At the necropsy slight swelling only of Peyer's patches were found. The bacillus typhi abdominalis is stated to have been obtained from the spleen and kidneys, but is not sufficiently identified as such.

Vaillard in 1890 reported the case of a young soldier who died after an illness of three days' duration. The chief symptoms were headache, epistaxis, pyrexia, constipation, retraction of the neck, and coma. At

the necropsy congestion of the lungs and meninges was found, but there was no intestinal lesion. The bacillus typhi abdominalis was obtained by culture from the spleen, lungs, and spinal cord; streptococci were also obtained from the spleen and meninges.

Karlinski in 1891 recorded three cases which occurred in an epidemic of typhoid fever. The characteristic symptoms of the disease were present during life, and after death no intestinal lesion was discovered; the spleen and mesenteric glands, however, were found to be enlarged. He states that he obtained the bacillus typhi abdominalis in cultivations from the spleen in all these cases. His evidence in favour of the micro-organism being the bacillus typhi abdominalis is not conclusive, however, on account of the insufficient technique of his bacteriological examination.

Guarnieri in 1892 described a case of typhoid fever which during life presented the characteristic symptoms of the disease. No intestinal lesion, however, was found at the necropsy, but the bacillus typhi abdominalis was obtained by culture from the biliary passages, liver, and spleen.

Du Cazal in 1893 recorded the case of a man aged 21, who died on about the twentieth day of the illness, and who during life presented the classical symptoms of typhoid fever—headache, epistaxis, continued fever, rose-red spots, and albuminuria. At the necropsy no lesion of any part of the intestine was found, the mesenteric glands were slightly enlarged, and the spleen was enlarged and weighed 420 grams; double pneumonia was also found. The bacillus typhi abdominalis was obtained in cultures made from the spleen and lungs.

Vincent, in 1893, described the case of a man, aged 35, who died about the twelfth day after the onset of a severe illness characterised by pyrexia, diarrhoea, purpura, and coma. At the necropsy the Peyer's patches were found to be normal; the mucous membrane of the intestine, however, was congested. The spleen weighed 230 grams; the mesenteric glands were not enlarged; bilateral pulmonary congestion was found. The bacillus typhi abdominalis and streptococci were obtained from the spleen, liver, kidneys, and heart.

Du Cazal, in 1894, recorded a case in which during life the ordinary clinical features of the disease were present. No lesion of the intestine or mesenteric glands was found at the necropsy. The bacillus typhi abdominalis was obtained in cultures from the spleen.

Osler mentions a case. The patient was a man, aged 60, who was admitted into hospital under his care. He had been ill for about two months, and on admission was found to be suffering from shortness of breath, and presented signs of pneumonia affecting the lower lobe of the right lung. Death took place twenty-four hours after admission. A diagnosis of senile pneumonia was made during life. At the necropsy the lower lobe of the right lung showed fresh pneumonia passing on to a condition of gangrene. There was no intestinal lesion. The organs were submitted to a bacteriological examination by Flexner, and pure cultures of the bacillus typhi abdominalis were obtained from the lungs, spleen, kidneys and liver.

Beatty, in 1897, reported the case of a man, aged 34, who died on the sixth day of the illness. Death was preceded by jaundice, hæmaturia, and coma. The spleen and mesenteric glands were found to be enlarged, but the intestines were found to be quite healthy. The bacillus typhi abdominalis was obtained by culture from the spleen.

Cheadle, in 1897, recorded a case. The patient was a boy, aged 3 years. During life the characteristic symptoms of typhoid fever were present. A brother and sister of the patient were also under observation, suffering from typhoid fever. On the twenty-first day of the disease a positive Widal reaction was obtained. On the twenty-sixth day of the disease typhoid bacilli were separated from the urine by Parietti's method. He died in the fourth week after the commencement of his illness. At the post-mortem examination no ulceration of the intestine was visible, and Peyer's patches appeared to be normal. The spleen was normal in size and appearance. The mesenteric glands were enlarged. The bacillus typhi abdominalis was obtained in cultures made from the spleen.

Nicholls and Keenan, in January, 1898, recorded a very carefully-investigated case. The patient was a man, aged 25. The most important symptoms and signs were headache, loss of appetite, general weakness, pyrexia, distended abdomen, rose-red spots on the abdomen, enlarged spleen and constipation. Cultures were taken from the blood during life, but were sterile. The blood gave the typical serum reaction. Death took place in the sixth or eighth week of the disease. At the necropsy no evidence of ulceration or healed ulceration was found in any part of the intestine. The spleen (375 grams) and mesenteric glands were enlarged and broncho-pneumonia was found. Bacilli, resembling in form the bacillus typhi abdominalis, were found in sections of the spleen, mesenteric glands, and Peyer's patches. Cultures from the spleen gave pure growth resembling the bacillus typhi abdominalis. The bacillus typhi abdominalis was also obtained from the liver. The bacillus tested with typhoid serum gave the typical Widal reaction. They give in their paper, a careful review of the subject, an attempt to explain the reason of the absence of the characteristic intestinal lesions, and draw particular attention also to an important paper by Sanarelli on the experimental production of typhoid fever.

An analysis of the 15 cases shows that this form of typhoid fever without intestinal lesions may affect patients at ages varying from 1½ to 60 years; 11 of the cases during life presented the characteristic symptoms and signs of typhoid fever, in 1 case the fever was of a recurrent type and the spleen was considerably enlarged, in 1 case purpura was a prominent symptom, in 1 case dyspnoea and signs of pneumonia were found and a diagnosis of pneumonia was made during life, and in 1 case jaundice and hæmaturia were noted. Death took place on the sixth, tenth, twelfth, twentieth day up to the sixth or eighth week from the commencement of symptoms. Enlargement of the spleen was noted in 8 of the cases, enlargement of the mesenteric glands in 9, pneumonia and pulmonary congestion in 5, and con-

gestion of the meninges in 1. The bacillus typhi abdominalis was obtained from the spleen in 14 of the cases, from the liver in 4, from the kidneys in 3, from the lungs in 3, from the mesenteric glands in 2, from the heart in 1, from the biliary passages in 1, and from the spinal cord in 1.

The above analysis serves to show the possible widespread distribution of the bacillus typhi abdominalis. The fact also that in such a large proportion of these cases the symptoms during life are looked upon as being typical of typhoid fever, indicates that in whatever organ the chief nidus of the growth of the bacillus may be, the resulting toxins will produce the characteristic clinical features of the disease. It is also interesting to note in connection with this last-mentioned point the excessive diarrhoea (*vide* Chart p. 777) which occurred in my case, which again shows, as has often been pointed out, that the diarrhoea has no direct relation, and is in no way dependent on the extent or severity of the ulceration which is usually found.

As an appendix, I add, with the kind permission of Dr. Pye-Smith and Dr. Hale White, the reports and temperature charts of the brother and sister, both of whom were in Guy's Hospital at the same time suffering from typhoid fever.

L.M.W., aged 7 years. Under the care of Dr. Pye-Smith, admitted on October 6th, 1898, for drowsiness and pyrexia. She has had measles, and when a baby had "snuffles," and was much wasted. Some years ago she had a discharge from both ears, which lasted for some months. She has had "cataract" in both eyes. According to the mother she has always been delicate. In May and June last she was in Martha Ward on account of a fractured thigh.

The patient appeared to be quite well on Friday, September 30th, 1898; nothing definite was noticed on the following day, but on Sunday she was drowsy and lying about the house. She had no cough, was not sick, although complaining of nausea. On Monday, October 1st, she "moped about" and her mother gave her some castor oil. On Tuesday, October 2nd, she was in a similar drowsy condition; on the 3rd she was brought to the hospital, and was then treated with some sulphate of magnesia medicine. On the 4th she was again brought to the hospital, and was then admitted.

She had had epigastric pain and also slight diarrhoea. According to the mother's account the motions were thin and dark.

Condition on Admission.—The pulse was 118, regular, full, and compressible; respirations 40; and the temperature 102.8°. No typhoid spots were visible. The abdomen was full and resonant all over. The spleen could not be felt. The tongue was furred except the middle, tip, and edges, which were red.

October 7th. The patient was sponged two or three times on account of the temperature rising above 105°. The bowels were opened after the administration of a glycerine enema; there was nothing abnormal noted about the faeces. Typhoid fever was suggested as the probable diagnosis.

October 9th. A rather bright red typhoid spot was seen about 3 inches above the umbilicus.

October 10th. No more spots seen; rhonchi heard over the chest.

October 12th. Bowels opened after an enema glycerini.

October 14th. The temperature dropped to 99° in the morning, but again went up to 101.8° later in the day.

October 17th. The spleen was felt.

October 20th. The patient was less apathetic.

October 21st. Bowels well opened.

October 25th. Temperature went up to 103.4°.

October 28th. Spots were again noticed. The temperature went up to 102°. During the last few days she passed a large quantity of urine, and a good deal of it was passed into her bed.

November 4th. The temperature still remained above normal. She was very apathetic.

November 5th. Urine passed more frequently into the bed.

November 7th. The bowels had not been opened for five days.

November 8th. A glycerine enema was administered with very little result; it caused a good deal of straining. A rectal examination was made, and a good deal of hard faeces was found in the rectum. An enema olei olivæ was ordered, and it was to be followed by an enema saponis if necessary.

November 10th. Patient very thin and anæmic; no spots; spleen not enlarged; no bronchitis. A specimen of blood was taken, and the serum gave a smart Widal reaction.

November 13th. The temperature had been normal for three days, and she no longer passed urine into the bed.

November 15th. In the evening her temperature was 96° and her pulse 48; she was very cold and collapsed. Stimulants (brandy, strychnine, ammonia, digitalis, and caffeine) were administered, and her condition improved.

November 18th. Condition improved.

November 21st. Pulse 43, digitalis mixture omitted.

November 24th. She was much better.

December 2nd. Steady improvement noticed. The brandy was omitted. Since November 11th the temperature had not been above 98.6° until December 1st, when it was 99.2°; this was accounted for by constipation.

December 10th. She was sent to a convalescent home.

A.W., aged 5. Under the care of Dr. Hale White. Admitted September 23rd, 1898, for "pain in the throat and stomach." He had previously suffered from measles and whooping-cough, and every winter he got attacks of bronchitis. About three weeks previous to his admission he was noticed to have lost his appetite and to have become weak and to be suffering from a dry cough; two weeks later he complained of sore throat and abdominal pain. He was unable to swallow solid food, and he coughed and vomited a brownish substance with very foul odour.

On September 19th this vomiting was very persistent, occurring about every hour and a-half.

Condition on Admission.—Apathetic. Pulse 112, regular, and compressible; respirations 28; temperature 101.8°. Tongue furred. Abdomen full; pain in the umbilical region, but not much tenderness. Spleen felt to be enlarged. No typhoid spots found. No abnormal respiratory sounds heard.

October 27th. A specimen of blood was taken, and the serum which separated gave the Widal reaction.

September 30th. Patient had no pain. No spots found.

October 3rd. Patient was dull and sleepy. The spleen could still be felt below the costal margin. The abdomen was a little distended, but there was no tenderness.

October 10th. The bowels were opened after an enema.

October 14th. Bowels again opened by means of an enema. Temperature did not rise above 98°.

October 18th. Bowels again opened by means of an enema. Farinaceous diet ordered.

October 25th. His temperature was 99.4° in the morning.

October 31st. He got up for the first time after tea.
November 8th. He was quite convalescent.
November 10th. He was sent to a convalescent home.

BIBLIOGRAPHY.

Banti, *Archiv. Italiane des Biol.*, December, 1887. W. Beatty, Enteric Fever without Intestinal Lesion, *BRITISH MEDICAL JOURNAL*, January 16th, 1897, p. 148. H. Brunschwig, La Lésion des Plaques de Peyer est-elle constante dans la Fièvre Typhoïde? *Thèse de Strasb.*, 1870, pp. 4-37. Cheadle, *Lancet*, 1897, ii, 254. Chiari and Kraus, *Zeitschrift f. Heilkunde*, 1897. W. S. Church, *St. Bart's. Hospital Reports*, 1882, xvii, p. 97. Du Cazal, *Bull. et Mém. Soc. Méd. des Hôp. de Paris*, 1893, p. 243. Du Cazal, *Le Bulletin Médical*, Paris, April 16th, 1894. Eiselt, *Gazette Hebdomadaire de Méd. Clin. de Paris*, June 20th, 1892. Flexner, *Journal of Path. and Bact.*, April, 1895. Flexner (Studies III), *Johns Hopkins Hospital Reports*, vols. iv, v, vii. Goodall, Two Cases of Enteric Fever fatal during the third and fifth week respectively, in which there was no Intestinal Ulceration, *Clin. Soc. Trans.*, 1897, p. 120. Guarnieri, Contributo alla patogenesi delle infezioni biliari, *Rivista generale Italiana di Clinica Medica*, 1897; *Ref. Baumgarten's Jahresbericht*, 1897, p. 234. E. Hodepnyl, On the occurrence of Typhoid Fever without characteristic Lesions of the Small Intestine, *BRITISH MEDICAL JOURNAL*, 1897, vol. ii, p. 1850. Karlinski, *Wiener med. Wochenschrift*, 1891, pp. 470 and 511, March 14th and 21st, 1892. M. Litten, Beiträge zur Lehre von Abdominal-typhus, *Charité Annalen*, vi, 1881, p. 103. J. W. Moore, *Path. Soc. of Dublin*, 1880. Nicholls and Keenan, Typhoid Fever without Intestinal Lesions, with the report of a case, *Montreal Medical Journal*, 1898, pp. 9-60. S. Phillips, *Clin. Soc. Trans.*, 1891. Sanarelli, Études sur la Fièvre Typhoïde expérimentale, *Ann. de l'Institut Pasteur*, 1894, 193 and 353. Thue, *Jahresbericht über die Fortschritte (Baumgarten)*, 1889, p. 196. Vaillard, De l'Infection par le Bacille typhique sans Lésions Intestinales, *La Semaine Médicale*, March, 1890, p. 94. Vaillard and Vincent, De l'Infection par le Bacille typhique sans Lésions Intestinales, *Bull. et Mém. de la Soc. Méd. des Hôp. de Paris*, 1890, p. 201. Vincent, Étude sur les Résultats de l'Association du Streptocoque et du Bacille Typhique, *Annales de l'Institut Pasteur*, February, 1893.

THE PATHOLOGY OF GAS POISONING, ILLUSTRATED BY FIVE RECENT CASES.

By J. LORRAIN SMITH, M.A., M.D.,
Lecturer on Pathology, Queen's College, Belfast.

THE following cases of poisoning by lighting gas occurred in Belfast in October, 1898, and January, 1899. I was asked by the coroner to examine the bodies, and in each case an exact determination was made of the percentage to which the hæmoglobin of the blood was saturated with carbonic oxide, the details of which are here recorded. As far as I am aware, no similar determinations have hitherto been made in cases of gas poisoning.

The lighting gas supplied in Belfast is a mixture of coal gas with about 40 per cent. of carburetted water gas. This mixture contains about 16 per cent. of carbonic oxide, whereas the coal gas contains only about 6 per cent. Water gas is now being extensively used in this country, and the serious extent to which its admixture with coal gas increases the risks to the public has just been reported on by a Home Office Departmental Committee.

On October 4th, 1898, S. H., a young woman, went to bed at 8.30 P.M., and was found dead at 6 next morning. The room occupied was of about 330 cubic feet capacity. It had no chimney. The ventilation by door and window was relatively small, as they were close fitting. The gas was found turned half on when the deceased was discovered.

Post-mortem Appearances.—A large amount of froth was observed about the mouth and nose. The lips were pink in tint. There was a distinct pink colour in the skin of the dependent parts. The left hand was extended. The right hand was flexed. *Post-mortem rigidity* was well marked.

Internal Appearances.—No pleuritic or pericardial fluid. Right heart collapsed; left heart moderately rigid. The cavities of the heart were nearly empty of blood. A few drops of blood were allowed to fall into a vessel of water, and at once showed the characteristic pink tint of carboxy-hæmoglobin. The lungs were slightly congested, but there was no oedema. The larynx and trachea were congested, and had a very pink appearance on the mucous surface. Some frothy mucus was in the bronchi. Emphysema very slight. The stomach contained half-digested food. The intestines had a diffused scarlet tint. The kidneys and liver were congested, but otherwise normal. The meninges of the brain were markedly congested. There was complete absence of any lesion to which death could be ascribed except the saturation of the blood with carbonic oxide.

Samples of the blood were taken in sealed tubes, and examined by the carmine method as soon as I had reached the laboratory. It was found to be 83 per cent. saturated with carbonic oxide. The conclusion was therefore perfectly clear that the patient had died from poisoning with carbonic oxide.

On January 4th, 1899, two fatal cases of gas poisoning occurred. A maidservant and two children had gone to bed in an attic room about 11 o'clock at night, and were discovered next morning shortly before 10 o'clock. The cubic capacity of the room was about 1,000 feet. There was a window, but no fireplace in the room. The floor and walls were imperfect in construction, and ventilation was probably fairly good. One child was discovered alive, and Dr. McHarry, by artificial respiration and other means,

succeeded in restoring it. The other child and the maidservant were dead.

Post-mortem Examination of M.A. (Maidservant).—The lips and conjunctivæ were pale. There was froth at the mouth and nose. There was a bluish-pink lividity on the dependent surfaces. *Post-mortem rigidity* was well marked. The body was scarcely cold twenty-four hours after its discovery. The muscles were red on incision. About 8 ounces of pink serous fluid was found in the left pleural cavity, and about 6 ounces in the right. About 2 ounces of a similar fluid was found in the pericardial sac. The lungs were pink in colour, and in both there was subpleural emphysema. There was a large amount of frothy mucus in the bronchi. The larynx and trachea were injected and pink in colour. There was no oedema or congestion of the lungs. The stomach and intestines were pale pink in colour. The stomach contained fluid food and its internal surface was pale. The œsophagus and spleen were blue. The liver and kidneys were redder than normal on their cut surface. The meninges of the brain were congested and bluish-red in colour, but otherwise normal. The brain substance was normal.

Samples of the blood were taken from the right auricle, and on examination by the carmine method were found to be 76.6 per cent. saturated with carbonic oxide.

Post-mortem Examination of M. C., aged 4 Years.—The appearances were similar to those of the case just described. There were some punctiform hemorrhages on the surfaces of both lungs, but no subpleural emphysema. There was, however, some vesicular emphysema in both lungs; otherwise the two cases were practically identical in their appearances.

The blood was examined by the carmine method, and was found in this case to be 69.5 per cent. saturated with carbonic oxide. The conclusion was, therefore, that in these two cases also death was due to carbon monoxide poisoning.

A point of interest in connection with these cases was that a young man who slept in an apartment, separated by a lath and plaster wall alone from that in which the escape of gas had occurred, also suffered from the effects of the gas. He was able to move about when he was discovered, and walked without assistance into the ambulance, which conveyed him to the Royal Hospital. Twenty-four hours later he had not completely recovered, and had no recollection of the occurrence, or of being brought to the hospital. It is probable, however, that the exertion he was allowed to make had a considerable effect in aggravating his condition.

On January 10th two cases of gas poisoning occurred. One of these was a woman aged 72, who had retired to bed about 11 P.M., and was found head at 8.45 next morning. The other was a young child sleeping in the same room, who was breathing when discovered, and was still alive when Dr. Tomb arrived. I am indebted to him for the following notes of the clinical condition of the case, and of the treatment he administered: "The child when I arrived was lying motionless and unconscious. The breathing was laboured, and there was froth exuding from the mouth. It had a cyanotic appearance, and the pulse was quick and thready. It was to all appearance in a dying condition. I wrapped the child in blankets, allowed free access of air to the room, and applied artificial respiration by Sylvester's method. I found a difficulty in this because when the arms were extended the muscles were thrown into a rigid condition from spasm. I administered coffee *per rectum*, and injected ether hypodermically, as the heart seemed to be in a weak, paralysed condition. The child remained motionless, but could be made to cry out when flapped with a wet towel. About three hours after I first saw it there occurred a fit of vomiting, when it died. There had been no sign of improvement during this time." I was requested by the coroner to obtain for analysis samples of blood from the bodies. There was no further *post-mortem* examination.

The blood when examined showed in the case of the woman the distinct pink colour of carboxy-hæmoglobin. It was found by the carmine method to be 57 per cent. saturated. The blood of the child, on the other hand, showed merely a recognisable trace of the pink colour and was not more than 5 per cent. saturated. As she had been exposed to at least as poisonous an atmosphere as the woman, there is no doubt her blood must have had at one time approximately the same saturation. The carbonic oxide had, however, disappeared from the blood presumably from the artificial respiration applied by Dr. Tomb.

The saturation in the cases was therefore as follows:

- No. 1.—83 per cent.
- No. 2.—76.6 per cent.
- No. 3.—69.5 per cent.
- No. 4.—57 per cent.
- No. 5.—5 per cent.

This series of cases affords an opportunity for raising some important questions in regard to the pathology and treatment of poisoning by coal gas. As is generally believed, and as was proved experimentally in an investigation which I recently carried out at the request of the Water Gas Committee, the poisonous effect of gas is due to the carbonic oxide and not to the other ingredients present, although there is no doubt that other poisonous bodies are present. The chief of these is probably benzene vapour; the quantity of this, however, which would be present in the atmosphere from accidental leakage would be harmless. Carbonic oxide