

Various writers have marked the prevalence of summer diarrhoea with a high ground air temperature; with this I agree, particularly when occasional showers alternate with prolonged high atmospheric temperature. We know that micro-organisms flourish best at the body temperature; this temperature is most nearly approached in both the ground air and atmosphere when infantile diarrhoea is most prevalent in any country. Moreover, micro-organisms want a certain amount of moisture present to flourish best as then they get a better nutritive medium. Now when we have occasional showers and a maintained high atmospheric temperature, and also high ground-air temperature, we have an ideal medium for micro-organismal growth. To my mind the organism of epidemic infantile diarrhoea is one which flourishes in such circumstances. Furthermore, investigation has shown that the fly requires neither too much nor too little moisture when depositing her eggs and that the sites she usually chooses are heaps of decaying vegetable matter or dung, which it is also well known are swarming hotbeds of micro-organismal life. I have pointed out that during the act of oviposition she remains motionless for seven and a half minutes. My theory is that whilst walking over the nauseous material in which she lays her eggs and during her act of oviposition multitudes of micro-organisms—and pre-eminent amongst them the *unknown one* of infantile summer diarrhoea, which seems to me to belong to the group of staphylococci in all probability—*adhere to* the moist sucker-like terminations of the hairs on the pulvilli of the fly's legs. Of course, any house fly, male or female, in its travels may pick up the organism as both sexes haunt masses of decaying vegetable and organic matter, but it is evidently the female that acts as the chief carrier.

The house fly loves warmth, and also the delicacies of our tables, so that we find it in abundance in our homes, frequenting the sugar bowl, the jam pot, and often falling into the milk jug. In this way it carries the contagion, which is left behind in our food, and when the fly falls into the milk the infective agent is washed off it and, unseen by us, floats in the milk. Babies often sleep with their mouths open and frequently also with milky saliva moistening their lips. As I mentioned previously, the fly seems to be attracted to an infant's mouth, and often will venture slightly inside it, the poison being washed off the insect's legs and infecting the child, which contracts summer diarrhoea even if breast fed. Of course, breast-fed cases of the disease are rarer than when the child is artificially fed, as it is not every infant who sleeps with open mouth. Moreover, the artificially fed child is constantly open to additional infection by virtue of its milk-supply, as also the sugar used to sweeten it. Usually the milk for the child is prepared twice daily and left uncovered either in the larder in better class houses, or amongst the poorer classes in the kitchen, so that the flies drop into it, and as the fly is removed and the milk not re-boiled but simply warmed before giving to the baby, the micro-organism remains alive, and later baby contracts the diarrhoea, especially among the poorer classes.

II.—*Methods of Prevention.*

The question now arises, How may epidemic infantile diarrhoea be prevented? During the years 1904, 1905, and 1906 I recommended patients of mine to keep a fine meshed muslin cover over the infant's face when sleeping. The milk should be prepared, diluted with water, barley water or lime water, and the requisite cream and sugar added, then boiled. It should then be placed in a vessel which is covered with a muslin cover and kept in a cool, well-ventilated place, for example, a safe or larder. A suitable covering is easily manufactured by twisting a piece of wire into a ring shape and sewing a muslin cover to it. The muslin should be renewed frequently. The milk should be prepared thus twice daily and a fresh supply used each time. All vessels used in preparing the milk should be scrupulously clean and scalded before use. The bottle used should be of the Lamb feeder type and should be cleaned after use, and then half a teaspoonful of boric acid should be placed in it, and the bottle filled up with hot water. The teat should be scrubbed and kept in a weak boric acid solution. Before again using both bottle and teat should be washed in warm water. I can safely say that where the above procedure has been adopted—and the cases are numerous—I have not known a case of epidemic infantile diarrhoea to occur. All vegetable and animal

refuse from the house, such as cabbage leaves, potato peelings, tea leaves, &c., should be burnt. No open ashpits should be allowed, but galvanised iron dust bins with a well-fitting lid should be insisted upon, and vegetable and animal refuse should be rigidly excluded from these. The bins should be emptied twice weekly. The dung from stables and shippens which are near dwelling houses ought to be frequently removed, and not allowed to accumulate as is usually the case. People should be instructed to keep their eatables in a safe outside the house, and such should be a necessary adjunct of every house, and should have a fine perforated metal door. Fly papers, of the sticky variety, should be at once used when flies appear, and if the pest gets numerous a little turpentine should be poured over some drying cinders on a shovel occasionally. I do not advise the wholesale extermination of the fly, for it has its place in nature. In fact, it is indispensable to our health and comfort, for if you turn over the body of some recently killed creature in the fields you will observe its frame is a seething mass of maggots. Thousands of these are the larvæ of the house fly. Returning in a few days you will find that they have devoured all the carcass save a little skin and the bones, the latter sinking into the ground and forming a valuable manure. So we see that the fly thus prevents decaying animal and vegetable matter decomposing into poisonous and ill-smelling compounds, which would breed pestilence. In turn, the larvæ, having developed into the fly, are preyed upon by birds, frogs, &c., which in turn are preyed upon by man and other animals, or are converted into constituents of the soil.

By the measures which I have recommended the occurrence of epidemic infantile diarrhoea can be effectually prevented. In large towns where there is already a system of dépôts where sterilised milk can be obtained it would only be necessary to go a step further and to print a leaflet of instructions and to provide the gauze articles at a low cost. Sanitary authorities could supervise the contents of the galvanised bins, &c.

Liverpool.

AN UNUSUAL CASE OF CHRONIC BI-NITROBENZENE POISONING.

By EDWARD WALKER, M.D. EDIN.,
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A YOUTH, aged 16 years, was admitted into the Huddersfield Infirmary on the evening of May 25th, 1907. In appearance he was pale and bluish, though the blueness did not amount to cyanosis. He was very weak and prostrate. His skin was a dirty yellow and his conjunctivæ were also yellow. He was restless and short of breath, rambling, excited, and sleepless. The temperature was normal; the pulse was 80, feeble, and of low tension. The tongue was dry and coated, the palate was yellowish, and the gums were blue. There was marked tenderness over the liver, stomach, and spleen. There was bilious vomiting and the bowels were constipated. The urine was dark brown in colour but clear and of specific gravity 1022. No bile was present. He was not in a condition to give an account of himself or to detail any history of his illness; but the condition of the urine, the colour of the lips and palate, the peculiar yellowness of the skin and conjunctivæ, the yellow pigmentation on the hands (so constantly found in workers amongst bi-nitro compounds), together with the tenderness over the liver, spleen, and stomach led us to make the diagnosis of chronic bi-nitrobenzene poisoning. We subsequently learnt that he had been working at some neighbouring chemical works amongst bi-nitro compounds from the previous January to April and that during that time he had on two occasions—once in February and once in March—suffered from slight attacks of acute bi-nitrobenzene poisoning; also that on each occasion he had been off work for two days. On April 29th he had another attack of a somewhat more severe character. He felt ill and weak, was dizzy, and had a staggering gait. He was breathless and his face was pale, and his lips and fingers were blue. He at this time left his employment because of his condition, but in a week's time was well enough to take some light open-air work. He,

however, never felt well. He was done up every night. He frequently had cramps in the legs and once or twice vomited dark green stuff. On May 19th he went home feeling very ill. He vomited and was very sick. He was dizzy, "light-headed and raving." He had pains in the head and limbs, and his urine was "like porter." His skin and eyes were much yellower than usual. This condition continued until he was sent into the infirmary on May 25th. After admission he showed no sign of improvement but each day the jaundice got deeper; the rambling and excitement were more marked. He became comatose and died on May 29th. The blood was examined by Dr. R. H. Walton, the senior house surgeon, who found the number of red cells and the amount of hæmoglobin normal.

Necropsy.—The post-mortem examination revealed several unusual features. The yellowness of the hands produced by contact with the bi-nitro compounds had not worn off, although the lad had been away from the work over four weeks. His gums were greenish-yellow and his palate had a yellow discolouration. On the lower costal cartilages on both sides were minute ecchymoses. The heart weighed 10 ounces and was pale and flabby. The colour of the blood was darker than usual. The liver weighed one and three-quarter pounds. It was rather soft in consistence and there was grey mottling on the upper surface of the right lobe. It was generally paler, especially the right lobe. On the under surface were several small paler areas, apparently fatty. On section, the whole of the interior had a peculiar yellowish-green mottling and appeared to be fatty. The liver substance was very friable, easily breaking down. The gall-bladder contained about six ounces of dark thick bile. The spleen weighed five and three-quarter ounces; it was more mottled in appearance than usual. The kidneys each weighed seven ounces; they were mottled, pale, and fatty. The mesenteric glands were enlarged and dark. On the mucous membrane of the intestines and the bladder were numerous minute ecchymoses. None of the organs were bile stained. Samples of the urine were sent to the Clinical Research Association who reported that they could find no trace of nitrobenzene nor could they find leucin or tyrosin or bile. The brown discolouration they attributed to the presence of a marked excess of indican. At the inquest evidence was given to show that the lad had worked amongst various bi- and tri-nitro compounds (bi-nitrobenzene, bi-nitrotoluol, and tri-nitrotoluol). Specimens of the liver and kidney and samples of the blood and urine were forwarded to Dr. W. Malden, Pathological Laboratory, Cambridge, who kindly sent the following report:—

Report on Specimens from the Patient.

Liver.—Deep yellow mottling round the bile-ducts; some large patches resembling acute yellow atrophy. Specimens too much macerated to cut sections from. Liver contains no excess of iron.

Blood.—Dark purple-brown colour. Spectroscopically gives band of met-hæmoglobin. Microscopically, differential leucocyte count per cent.: eosinophils, 1; polymorphonuclears, 50; large mononuclears, 5.5; lymphocytes, 43; mast, 0; nucleated red cells, 4 seen; myelocyte, 1. Great variations in size of red corpuscles. Some poikilocytes. A fair number of basophil reds and some polychromatophils.

Urine.—Rich brown, clear. Reaction faintly acid. Specific gravity, 1020 at 60° F.; albumin, distinct trace; sugar, none; bile, slight trace; free urobilin, considerable quantity chemically and spectroscopically; free di-nitrobenzol, none. Microscopically, much epithelial debris, flat, squamous, cylindrical, and goblet cells. Casts, a few epithelial casts. Blood and pus cells, a few.

Opinion.—In my opinion this case is undoubtedly one of chronic poisoning by some body of the nitrobenzene series. The clinical history is clearly in favour of this diagnosis. History of three months' work in chemical manufacture of di-nitrobenzol and other allied compounds. Twice off work during that time from symptoms of poisoning. State on admission to hospital: cyanosis, vomiting, jaundice, dark urine, dyspnoea.

Post-mortem appearances.—Liver resembling acute yellow atrophy. Heart pale and fatty. Intestines ecchymoses. Kidneys enlarged and fatty degeneration. The specimens I have examined confirm the diagnosis. The blood and urine are most characteristic. The only points which are not in favour of bi-nitrobenzol poisoning are these: 1. There

was no anæmia (red corpuscles counted normal). 2. There was no increase of iron in the liver. These, however, cannot outweigh the balance of probability in favour of bi-nitrobenzol poisoning as shown by all the other appearances.

WALTER MALDEN, M.D.

Remarks.—So far as I know, no case exactly resembling this has been described before, but there can, I think, be no doubt about the diagnosis. The only other thing that it could be is ordinary acute yellow atrophy. Probably the hepatic degeneration closely resembled that which takes place in this disease but that it was acute yellow atrophy and nothing more is rendered unlikely by the following facts. In acute yellow atrophy the gall-bladder is empty. In this case it contained six ounces of bile. In acute yellow atrophy many of the organs are bile-stained. There was no bile-staining at all. In acute yellow atrophy there is almost invariably leucin or tyrosin or both in the urine. In this case these were both absent. Besides, as Dr. Malden points out, the clinical history, the history of work amongst bi-nitro compounds, the fact that he had suffered on three previous occasions from acute bi-nitrobenzol poisoning all point to the probability of the case being one of chronic bi-nitrobenzene poisoning.

Huddersfield.

A CASE OF FOREIGN BODY IN THE LEFT BRONCHUS.

By H. A. MOFFAT, F.R.C.S. ENG.

THE patient in this case was a policeman, about 35 years of age, whose previous medical history was unimportant. I first saw him on May 29th, 1908, when he told me that six days previously he went to sleep with a piece of chewing gum in his mouth. He woke up with a fit of violent coughing and with some shortness of breath and he found that the gum was gone. For a day he thought nothing of it, but as the cough persisted he saw his medical attendant; though advised to go off duty he refused to do so. The cough persisted, there was wheezing in his chest, and he was short of breath. There was pain on the right side over the second intercostal space. On the next day he was shivering and feverish. A troublesome cough with expectoration of unpleasant tasting muco-pus persisted; he was short of breath. On the 29th he was sent to me.

The patient was a well-developed and well-nourished man. There was a strained, anxious look about his face, his breathing was slightly laboured, and his lips and ears had a bluish tint; he had a frequent clanging cough. There was audible wheezing. The temperature was normal. The larynx and vocal cords were normal. The right side of the chest moved a little less freely than the left; there was no loss of resonance; vocal fremitus was normal. The air entered well all over. A loud rhonchus was heard at the end of inspiration and of expiration all over the chest, but was loudest on the right side just above and inside the right nipple. The voice sounds were normal. I found that a piece of chewing gum like the one which he had lost gave no shadow with the x rays. The patient said that he had often chewed a piece for two days without any appreciable diminution. Apparently the composition is a little pepsin, sugar with peppermint, with guttapercha, which forms the great bulk of the gum.

I advised the patient to go into hospital, but it was not until the next day that arrangements were made. He was admitted just after a rigor and had a temperature of 103° F. Just after admission he vomited; in the vomit were about two ounces of thick muco-pus. He was put on expectorants and a kettle was kept going, as I decided to wait and to see if the temperature would go down. On the 31st the temperature had fallen and he felt better; the slight dyspnoea and cough were unchanged. The rhonchus was heard only occasionally, and then only on the right side below the right nipple; the percussion note in the right axilla was a little flat. On June 1st there was no change; the temperature ranged from 99° to 100°. I advised a tracheotomy and a search from the wound, but he wished to put this off, having an idea that the gum would disintegrate; he had coughed up a piece of about the size of a pin's head. In the afternoon he had an attack of dyspnoea,