glands, which might subsequently be aggravated by psychic trauma or conflict, or by other toxins. In dementia praecox there seemed to be both physical and psychic factors. If this idea of the origin were correct, we must look for prevention, not to the school doctor, but to the ante-natal clinic.

POISONING BY ARSINE.

BY

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ROYAL INFIRMARY.

IN THE BRITISH MEDICAL JOURNAL of June 19th, 1920, Dr. T. H. Wignall described a series of cases of non-fatal poisoning by arseniuretted hydrogen (AsH_3 ; arsine) occurring during the production of hydrogen gas and the reducing of certain benzene derivatives. Rambousek states that English factory inspectors reported in 1906 three cases occurring in an electrolytic process, and in 1907 two cases—one in a chemist who was separating bismuth from a solution of bismuth chloride in hydrochloric acid, and the other (fatal) in a man who had cleaned out a vitriol tank.

It may be interesting to record a further fatal case from inhalation of the same gas, produced during the manufacture of zinc chloride by treating in stone tanks or "becks," "zinc ashes" and "flux skimmings," residues in the zinc galvanizing process, with commercial hydrochloric acid. Gases are given off freely, and the men are instructed to go away from the beck as soon as the charge has been emptied into it; they are also provided with masks, but state that they do not find them of much use and rarely wear them. When the evolution of gas lessens, more material is thrown in, and towards the end of the process the becks are stirred with long iron bars until the neutralization is complete. In addition to the hydrogen, it is evident from the smell that sulphurous vapours come off, and the following case shows that arsine is also evolved in considerable quantity:

Case History.

J. C., aged 46, on the morning of May 11th, when shovelling zinc ashes into a large beck, complained that he did not feel well. He had been observed leaning on his spade near the beck instead of moving away at once. When he went home at 6.30 p.m. he complained of severe pain in the stomach and of having vomited. Dr. F. W. Taylor saw him late that night. He found marked cyanosis of the ears, lips, hands, and feet. The man complained of shortness of breath, which seemed to be due to pain in the upper abdomen. The next morning the skin was of a uniformly light brown colour and of a peculiar silky smoothness to the touch. The eyes were prominent and the conjunctivae swollen and intensely congested. He was slightly delirious; the pulse was of fair volume and not much quickened, and the temperature was normal. Respiration was quiet. The abdomen, especially the right hypochondrium, was tender, but not rigid; he vomited frequently during the day. No urine was available for analysis, for none had been passed since he came home, and on catheterization only a few drops of blood were obtained. On the 13th he was evidently much worse, being in a state of low delirium, with complete suppression of urine and obstinate constipation, though the vomiting had ceased and he was able to take fluids. I saw him with Dr. Taylor on the 14th. His appearance was as previously described; he was still delirious, taking no notice of anything unless spoken to; the temperature was 98.4° , the pulse 90, of fair volume. Heart sounds were clear, cardiac dullness was increased towards the left, respirations were not markedly quickened. The lungs showed no dullness on percussion, but mucous râles were heard on both sides. He died comatose at 12.45 a.m. on the 15th.

Post-mortem Examination.

On May 16th I made a post-mortem examination. The body was well nourished, the skin of a pale olive-brown colour and silky to feel. Lips, nails, and ears deeply cyanosed, eyes prominent, conjunctivae swollen and brownish. The trachea contained a frothy brown fluid, and its mucous membrane was congested; both lungs showed old pleuritic adhesions, and were congested and oedematous, the fluid being brownish in colour. The pericardial cavity contained $1\frac{1}{2}$ oz. of dark brown fluid. Heart enlarged (weight $21\frac{1}{2}$ oz.); its fat of deep orange yellow colour, muscle and valves apparently normal. The oesophageal mucosa was green, especially at its lower end; the fat of the omentum and mesentery was also of a peculiar green tinge. The exterior and the mucous lining of the stomach and intestines, which both contained a little thick brownish fluid, were of a uniformly slate-blue colour. The liver weighed $79\frac{1}{2}$ oz., and on section was of a greenish yellow-brown colour; the gall bladder was full of greenish-black bile. The pancreas and

adrenals were apparently normal; the bladder quite empty. The kidneys were enlarged (right $8\frac{1}{2}$ oz., left $7\frac{1}{2}$ oz.), and on section dark purple-brown. The perinephritic tissues were deeply congested. The spleen weighed $7\frac{1}{2}$ oz., and was of a deep red-brown colour. The meningeal vessels were rather full, and there was a little clear fluid under the membranes.

Chemical Analysis.

I sent portions of the stomach and its contents, liver and kidney, and some hair from the head, to the Manchester University Public Health Laboratory, and Dr. Henry, who represented the Home Office, obtained and caused to be examined samples of the materials used in the manufacture of zinc chloride. The report is as follows:

The hair contained 5,000 milligrammes of As_2O_3 per 100 grams; the kidney, 1,000; the stomach, 300; and the liver, 800. As the liver weighed 2,246.6 grams, it had an As_2O_3 content of 4.537 mmg. On analysing the materials used, it was found that the commercial hydrochloric acid contained 10,000 mmg. per 100 grams; the zinc ashes, 5,000; and the flux skimmings, 15,000. On heating the last named with the hydrochloric acid, a mixture of hydrogen, arsine and hydrogen sulphide was evolved; two cubic inches of arsine is stated to be sufficient to cause death.

The extremely dangerous character of the gas is shown by the fact that the man apparently absorbed a fatal dose before he felt any inconvenience, at any rate not enough to make him move away. Another point of interest is that up to this illness he was stated to have had excellent health, never losing a day's work, yet the heart, liver, and kidneys were all considerably over the normal weight. On section they had the appearance of having been in a hardening fluid, and no blood could be squeezed from them. The very striking light brown colour of the skin, so different from the ordinary jaundice, is also noteworthy.

This is the first fatal case at the chemical works at which this man was employed. I wish to express my indebtedness to my colleague, Dr. Taylor, for permitting me to publish this case, and to Mr. H. Heap, of the Manchester University Public Health Laboratory, for his careful analyses of the material.

A CASE OF ERYSIPELAS TERMINATING IN ACUTE THYROIDITIS.

BY

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ACUTE thyroiditis is a rare condition, but is sometimes associated with infectious diseases such as erysipelas and septicaemia, as explained by McCarrison in his book on *The Thyroid Gland*, in 1917. The details of the following case may therefore be of interest:

Mrs. S., aged about 46, married, with four children, had a swelling in the right lobe of the thyroid since the last confinement, about ten years ago. It had caused no inconvenience, but she had been attending a doctor for some time with beneficial results. On September 23rd, 1919, she suddenly developed erysipelas of the left side of face, scalp, and neck. She was seen by Dr. Duke on September 28th, and was transferred to me on September 30th. At that time the patient was slightly delirious; the temperature was 105° , the pulse 120, and there was marked erysipelas of the left side of the face and scalp and a brawny swelling of the neck. Dr. Beattie was called into consultation on October 1st, and it was decided to continue fomentations to the neck, ichthyol ointment to the scalp and face, and a mixture of quinine and arsenic by the mouth. Anti-streptococcal serum was not given.

The signs gradually subsided in six days, but immediately afterwards commenced on the right side, the temperature rising to 104° . The same treatment was carried out, and the temperature became normal on October 16th.

Three days afterwards small patches of erysipelas appeared on the right buttock, a hard painful, deep-seated swelling could be felt in the gluteal muscles, and the temperature rose to 103° . No fluctuation was obtained, and with fomentations the swelling disappeared, to be followed two days later by the same condition in the left buttock, the temperature rising to 102° ; it followed the same course, all signs disappearing by November 2nd.

On November 5th she complained of pain and stiffness in the right side of the neck, and the temperature rose to 101° . The following day the swelling in the thyroid was much larger and very tender and hard; it was adherent to the sterno-mastoid behind and overlapped the clavicle. There was pain on swallowing, and depression of the chin on the sternum on swallowing with marked limitation of chin elevation. On November 8th the patient was hoarse, the swelling was fixed to the skin, and fluctuation could be felt. Mr. Richardson was consulted, an exploring needle was introduced and pus drawn off. An incision was made and a cupful of pus evacuated, which on

examination proved to be a pure growth of streptococcus. The patient made a rapid recovery, but as the sinus would not close Mr. Richardson opened and scraped it, and it eventually healed on February 10th.

During this time the patient was showing increasing signs of myxoedema. The hair fell out so that a wig had to be worn, the body surface became dry, and she easily felt the cold; the pulse was slow and mental depression was present. These signs and the original swelling all disappeared when the sinus closed; marked drawing up of the scar on swallowing was noticed. There must have been a small focus left, for on July 26th she had a rise of temperature and complained of stiffness and swelling over the scar; two days after it was opened, the pus coming away still being pure streptococcus. A vaccine is now being used with the hope of increasing her resistance.

This case is clearly one which, commencing as an erysipelas, developed into a septicaemia and terminated as an acute thyroiditis. Each attack was milder than the preceding one, showing that some immunity was developing. However, I consider in the light of what occurred that had a blood examination been made in the early stages of the illness, an injection of antistreptococcic serum might have avoided the septicaemia and acute thyroiditis.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

HEREDITARY SPASTIC PARAPLEGIA WITH ATAXIA AND MENTAL DEFECT.

The following are some observations on a family presenting this condition.

The family consisted, in order of birth, of a daughter, M. A., a son, W., and two daughters, M. E. and E. A. There were no other children, and all four were affected. M. A. died in 1905, aged 22; W. died in 1915, aged 27. M. E. and E. A. are alive, aged 28 and 23 respectively. Both parents are alive, aged 60 years, and are unaffected. All the children were born seemingly healthy and intelligent, and went to school in due course, where they learned to read and write. The first sign of any abnormality appeared between the ages of 7 and 8 years, when they were noticed to stagger in walking. This defect progressed, and in a short time they were unable to attend school. The history is clear that in all the onset occurred in the same manner, and at approximately the same age. The two survivors, M. E. and E. A., present practically the same signs, so that one description will serve for both.

Mentality.—Expression vacant. Speech very slow, and articulation clumsy. Can answer simple questions, such as name and address, and can repeat alphabet, but cannot read. Able to form some of the letters in their names in slow, tremulous writing.

Eyes.—Double internal strabismus, lateral nystagmus; pupil reflexes normal.

Reflexes.—Greatly increased knee-jerks, ankle clonus, Achilles and triceps jerks. Babinski's extensor response in left foot of E. A.

Feet.—No marked deformity, slight tendency to pes cavus and talipes varus.

Gait.—On walking, the toes drag and the feet are spread out, but they cannot walk more than a yard without holding on. They often fall and hurt themselves, but there are no fits. With eyes open, they are unable to stand without swaying, and if the eyes are closed they tend to fall immediately.

Tremors.—Slight intentional tremors.

Menstruation.—Both commenced menstruation at 13, but after three "periods" the flow ceased completely, except in E. A., who had a slight "show" in April, 1919.

The palate in both is well formed, and there is no spinal curvature.

Although the condition itself is well known, and is believed to be allied to Friedreich's ataxia, yet its hereditary origin is difficult to elicit, and this case presents no exceptions. As stated above, both parents are alive and unaffected. On the mother's side, no abnormality can be traced. The father is the illegitimate son of a woman who was an inmate of Wigan Workhouse owing to blindness following an accident to her eyes at a mill, and of a man who was an "invalid" in the workhouse. The nature of this invalidity is not known, but it is known that he left Wigan and died in Preston Workhouse Infirmary, not long after this son was born. As this man died in 1860 or 1859, it is hardly likely that the records of either Wigan or Preston Workhouses would reveal much as to whether he suffered from a chronic degenerative disease of the central nervous system or not. What is certain is that this man suffered from some affection which made him a chronic invalid in a workhouse infirmary.

Now, the woman who gave birth to this illegitimate son

—the father of the persons affected and described in this note—was herself apparently free from any transmissible defect, because before becoming blind through an accident, she gave birth to another illegitimate son by a different father, and this son is still alive, and has a healthy family of four children. This reproductive test clears the woman of transmitting the defect, and leaves it to be inferred that the only traceable origin would presumably lie with the invalid grandfather, who through the medium of his unaffected son passed it on to his grandchildren.

Although the evidence as regards heredity is not quite clear, yet it would be still more fallacious to presume either syphilis or alcoholism in the parents, of which there is no evidence at all.

Warrington.

J. S. MANSON, M.D.

A CASE OF LOBAR PNEUMONIA ACCOMPANIED BY ACUTE APPENDICITIS.

THE following case shows that acute abdominal pain accompanying lobar pneumonia may sometimes be due to a simultaneous attack of acute appendicitis. The textbooks so often warn the student to distinguish carefully between the two diseases, that it is well to remember that they may in rare cases coexist:

M. S., aged 5 years, was under my care for an attack of bronchitis when on April 26th, 1920, I was called to see her owing to the onset of acute pain in the abdomen. On arrival I found her in great pain, having rigidity of both recti and being very intolerant to examination. Her temperature was 101.6° and her pulse 130; respirations were 36. Examination of the chest showed dullness on percussion at the base of the right lung, with inspiratory crepitations. Her face was symmetrically flushed and the tongue coated with a brownish fur. She had vomited once. On the following day there were unmistakable signs of a right basal lobar pneumonia, and the temperature chart for the next few days was typical of that disease. The abdominal pain gradually subsided, but a week later there were still localized pain and rigidity in the right iliac fossa. The temperature remained above normal, and became oscillating in character; the pulse varied from 90 to 100. She was removed to the Isle of Wight County Hospital for observation on May 5th. The temperature and pulse became normal, but even after all signs of consolidation of the lung had disappeared there remained still slight tenderness and resistance in the appendix region. On May 17th appendicectomy was performed by Dr. Turner, assisted by Dr. Pridmore. The appendix was found to be inflamed; the tip had sloughed off, and a concretion was found in the peritoneal cavity. Numerous adhesions in the appendicular and caecal regions made removal of the appendix difficult; no abscess existed round the concretion. The child made an uninterrupted recovery, and is now quite well.

It is remarkable that in spite of the acuteness of the appendicitis (as shown by necrosis of the tip of the organ) no abscess was found at operation. There was no history of a previous attack of abdominal pain. The question arises whether the two inflammatory processes arose simultaneously from a common septic focus, or whether one was secondary to the other. I am indebted to Dr. Turner for his courtesy in supplying me with details of the operation, and to Dr. Pridmore (in whose practice the case occurred) for his assistance and advice.

Ryde, I.W.

L. FIRMAN-EDWARDS, M.B., B.Ch.

INFLAMMABLE GAS AS A PRODUCT OF FER- MENTATION IN MALIGNANT DISEASE OF THE STOMACH.

As there are but few recorded cases of this nature, I think the following is worthy of being added to the list and may prove of interest to readers of the JOURNAL.

A middle-aged man had suffered for three weeks from symptoms of indigestion, and complained of foul eructations of wind, to which he was becoming very sensitive. He had not suffered from vomiting or pain, but had lost appetite and weight, and his bowels were confined. His abdomen was distended and "ballooned." I suspected the presence of tumour formation, but this was not palpable. One evening after supper when lighting his pipe he expelled a quantity of wind, which, much to his alarm, caught fire and singed his eyebrows and moustache. The eructed gas was not always inflammable, but on two occasions I was fortunate in seeing its inflammability demonstrated. The gas burned with a pale blue luminous flame and an explosive report; it was probably marsh gas. With the subsidence of fermentation under treatment a tumour of the stomach became palpable, from which he died a few months later.

Glasgow.

W. H. STIRLING ARMSTRONG, M.B., Ch.B.