4. Water from the same source allowed to stand for a few days or weeks in clean stoppered bottles constantly produced high fever after boiling. The amount of fever was always nearly proportionate to the volume injected. Several hundred observations were made. The total residue from 225 c.cm. of water that had stood for many weeks was 2.2 mg. Two different samples were plated out. (a) The water that had stood for two months gave 40 organisms per c.cm. (b) The water that stood for ten days gave 73,000 organisms per c.cm. The counts in all cases were made from agar plates grown for forty-eight hours at 37° C. Counts made from agar plates at 22° C. were taken on two occasions and gave very similar results. The water that contained 40 organisms per c.cm. gave a greater fever than that which gave 73,000 organisms. Both these waters were centrifugized for thirty minutes in Dr. Martin's turbine centrifuge. Small quantities of the upper layers immediately below the surface invariably gave rise to high fever. The invisible deposit when injected in 0.50 c.cm. of water gave rise to no disturbance of temperature.

5. Distilled water that had been allowed to stand gave rise to fever after filtration either through a Doulton filter or through cotton-wool. Two filters were used. One was new, the other had been used several times. In both cases after filtration the water showed on analysis considerable quantities of calcium. Before filtration the ammonium oxalate test gave no reaction. The same results were obtained with a Berkefeld filter.

REFERENCES.

- ¹ Grünbaudi, A. S., and H., *BRITISH MEDICAL JOURNAL*, November 11th, 1911. ² Bingel, A., *Arch. f. exp. Path. u. Pharm.*, 64, p. 1. ³ Baum, *Arch. f. exp. Path. u. Pharm.*, 1892. ⁴ Allbrecht, *Lubarsch-Ostertags Ergeb. der Path.*, 1907, Jg. xi, p. 1163. ⁵ Rössle, *Berl. klin. Woch.*, 1907, p. 1105. ⁶ Hössli, *Frankf. Zeitsch. f. Path.*, 2010, Bd. 4, p. 258. ⁷ Hort, E. C., *Proc. of the Roy. Soc.*, B. vol. LXXXII, 1910. ⁸ Wechselmann, *Deut. med. Woch.*, April 27th, 1911, N. 17, p. 779.

A REPORT OF SIX CASES OF ARSENICAL POISONING CAUSED BY FUMES FROM A COKE STOVE.

BY

J. GEORGE TAYLOR, M.A., M.D.CANTAB.,
M.R.C.P.LOND.,

HONORARY PHYSICIAN, GENERAL INFIRMARY, CHESTER,
AND

K. V. TRUBSHAW, M.B., B.S.LOND., M.R.C.S.,
L.R.C.P.,

HONORARY SURGEON, MOULD COTTAGE HOSPITAL.

THERE are many well-known ways by which arsenic can be slowly absorbed into the system, causing symptoms of poisoning, such as from wall-papers containing arsenic, but we do not remember having seen any record of arsenical poisoning being caused by fumes from a coal or coke stove. For this reason we think the following cases are of sufficient interest to be worth reporting.

On May 4th, 1911, one of us was called to see a girl working in a laundry who was suffering from vomiting and loss of power in her legs. As after a few days she did not improve, she was sent home, for there were no means of nursing her where she was working.

In the early part of June, 1911, two more girls were attacked with weakness in the legs and vomiting, followed in a few days by pain in the legs. When we together saw these two girls, on June 29th, 1911, one of them (Case II) was very ill indeed. Her face was cyanosed, and there was some puffiness of the eyelids. She seemed to be half-dazed, and lay in bed curled up. It was difficult to get an answer from her, and she gave the idea of being rather hysterical. She frequently vomited, but had no diarrhoea. She had diplopia, due to paresis of the external recti muscles; on making her look to either side, in addition to the eye not moving beyond the middle line, there was marked nystagmus of both eyes. The pupils were widely dilated, but reacted sluggishly to light. The legs were almost completely paralysed—they could not be raised from the bed or flexed at the knee by the patient, and her attempts to move them caused cramp. There was complete loss of sensation of touch below the knees, though sensations of heat and cold were normal. The

calf muscles were exquisitely tender, the patient crying out on the slightest squeeze. There were no knee-jerks or plantar reflex, and, owing to loss of power in the tibialis anticus, the feet lay in bed with the toes pointing downwards. The pulse was slightly irregular. There were no changes in the skin. The other case (Case III) was in practically the same condition, but less severe.

We both thought of arsenic as a possible cause, but the most thorough examination of the colouring on the walls and of the food failed to detect any anywhere. We searched the garden—a very large one, in which the workers were allowed to roam and pick leaves and flowers—for poisonous plants, but failed to find any cause here.

Two or three days later another girl fell down while standing at a wash-tub, her legs simply giving way under her.

On July 3rd we again visited the laundry to make a further examination. Inquiry elicited the fact that all the cases at the time they were attacked either actually were, or had just been, working in the ironing room. We thoroughly examined this room; it was large and lofty, there was no crowding or lack of air space, and we could not find anything even suspicious here. Leading out of the ironing room up a few steps was the room where the irons were heated; this was a small room, 15 ft. long by 7 ft. broad, and about 8 ft. high. There was no window, and only one door leading to the ironing room. It was lighted by two very small skylights, one of which was closed, and the other not half open. To open or close these lights some one had to climb on to the roof outside. There were two iron stoves in the room with the flues running straight up through the roof. The stoves were so placed that it was possible to walk completely round each one, and each stove had three tiers of shelves running quite round it, on which to stand the irons, a worker bringing her cold iron in, placing it on a shelf quite close to the stove to heat, and taking out a hot one. The stoves, which were cracked in places, and the flues being red-hot, we thought it just possible that the toxic agent might be carbonic oxide gas, for we found that coke, and not coal, was burnt in the stoves, as when coal was used the flues did not draw properly and the room became full of smoke; therefore, we thought that probably the air might be contaminated with CO gas both from the down-draught through the cracks, etc., and also possibly by the passage of the gas through the red-hot iron. We accordingly took samplings of the air in this room after having the stoves well stoked up, and the skylights and door closed for some time. We examined the blood of the two worst cases spectroscopically for carboxyhaemoglobin, but failed to detect it.

Mr. H. Lowe very kindly examined the air samples for us, and reported that there was no CO gas present in it, but a fair proportion of SO₂ gas. Mr. W. F. Lowe, A.R.S.M., F.I.C., suggested that as SO₂ was present in the air probably there was arsenic present in the coke used in the stoves, as "malt has frequently been contaminated with arsenic by the use of coal or coke in the kilns instead of anthracite." He most kindly undertook to examine for arsenic the coke used in the stoves. His report was as follows: "The total amount of arsenic (calculated as As₂O₃) in the coke is 0.001 per cent., or $\frac{1}{100}$ grain per lb. The fixed arsenic (as As₂O₃)—that is, the portion which remains in the ash when the coke is burnt—amounts to 0.0002 per cent. or $\frac{1}{5000}$ grain per lb., so that four-fifths of the total amount is volatile and passes off in fumes when the coke is burnt."

We have no doubt that the fumes from the coke escaping into the air of the stove-room were the cause of the poisoning in these cases and that the poisoning was arsenical, because we detected arsenic in the urine of the two worst cases admitted to Chester Infirmary, and the presence of this arsenic was verified for us by Mr. W. F. Lowe. Since attention was drawn to the danger from the stoves no more cases have occurred. Coal only is used in the stoves, so that any leak can be at once recognized by the smoke in the room, and a fresh stove has been installed in a bigger room.

CASE I.

F., aged 17 years. In February last this girl ran away from the laundry, and was out in the cold all night. Five days later she complained of pains in her legs. This continued until the end of April, when she began to vomit. There was "numbness

and tingling in her legs. She did not feel sure of her steps, and felt dazed."

When first seen, on May 4th, 1911, she could only walk with difficulty. There was much loss of power in the legs, especially of the extensor muscles. Knee-jerks and plantar reflex were absent. Sensation to touch was impaired, but not abolished. The upper extremities were not affected, and there was no diplopia or squint. She left the institution, and was subsequently treated in the Mill Road Infirmary, Liverpool. At the present time we hear she has practically recovered the use of her legs, and is able to walk slowly. Her general health is good. She had worked in the ironing room since the previous September.

CASE II.

F. G., aged 17 years, was seen by us together on June 29th, 1911, and admitted to Chester General Infirmary on June 30th. The patient stated that three weeks before admission her legs started "going from under her," and became painful. Everything she took "came back." She said she felt giddy, and if she looked long at one object she saw it double. She had worked from March to June in the ironing room.

The patient on admission is pale and anaemic-looking, with a cyanosed appearance, which becomes more marked on exertion. Her eyelids are puffy. Heart sounds are normal, but beat is irregular. Tongue clean. Bowels constipated.

The pupils are dilated, but react sluggishly to light. On fixing an object with the eyes there is convergent squint, the external recti muscles are both completely paralysed, and on looking at an object on either side of the field of vision there is marked nystagmus.

There is complete loss of power below the knees, and great wasting of the leg muscles; the muscles of the thigh act very slowly, but any attempt at movement brings on violent cramp in the legs. There are no knee-jerks or plantar reflexes, and on taking hold of the calf of the leg the patient cries out with pain. The tibialis anticus has completely lost its power, and the foot lies with the toes pointing downwards; on lifting the leg the foot is quite flail-like. There is complete loss of sensation of touch below the knee, but the sensations of heat and cold are not impaired. Electrically there is a complete reaction of degeneration. There is retention of urine. The ordinary routine examination of the urine on admission showed nothing abnormal. Later a more detailed examination revealed arsenic. For a week after admission a catheter had to be used.

She was treated with strychnine and dilute hydrochloric acid, and milk and barley water until the vomiting stopped. Massage to the legs was begun as soon as the tenderness was sufficiently diminished for the patient to bear it, that was in about nine weeks after admission. Supports were kept below the feet to keep the toes up in order to prevent any deformity occurring. The weakness of the external recti muscles slowly passed off, and now she is able to move her eyes perfectly, and there is no diplopia or nystagmus. Now, eighteen weeks since her admission, she is able to flex and extend her knee, but her leg muscles are still very weak, though the tenderness has completely disappeared. She is still improving under massage and electrical baths to her legs. Menstruation began again in October after four months' cessation.

CASE III.

A. D., aged 15 years, was admitted to the Chester General Infirmary at the same time as Case II. About the end of May she noticed her legs getting weak; about a week later her calves became painful, and have been getting worse since. At the same time there was giddiness and pain in her eyes; she says that she saw things "as through a mist." She had abdominal pain and vomited after food. She had been working in the ironing room for about two months.

On admission, this patient's condition was exactly the same as Case II, except that none of the symptoms were so severe. She had diplopia, but only slight paresis of the external recti. There was loss of power in the legs and tenderness of the muscles on gripping the calf, but very little if any wasting. There was no bladder trouble. There was arsenic found in the urine. This patient improved more rapidly than Case II, and in seven weeks' time was able to get up and in ten weeks could walk, but still had ankle-drop and difficulty in walking. In fifteen weeks she had lost all her symptoms and was apparently quite well.

CASE IV.

A. N., aged 16 years, seen by us at the laundry on July 3rd, 1911. The day before she had tumbled while standing at her work, her legs simply giving way beneath her. She had no vomiting, but was very anaemic looking, and had no other symptoms except the weakness in her legs. This patient was sent home at once.

CASE V.

M. C., aged 40 years, was admitted into Chester General Infirmary on July 11th, 1911. She was deaf and dumb, so that it was very difficult to obtain a history from her. Her legs were extremely painful on taking hold of them, but she was able to walk with help. There was no squint, and apparently she had not got diplopia. She improved rapidly under treatment, and was discharged quite cured on September 20th, 1911.

CASE VI.

G. M., aged 23 years, complained of pains in the abdomen and legs, with some loss of power. She was removed by her friends, and subsequently admitted into the Royal Hospital, Salford. Here she was diagnosed as a case of "peripheral neuritis of

unknown origin." This girl was engaged in the ironing room from April, 1910, to April, 1911. Her symptoms were much the same as in the other cases: Numbness and tingling in the legs and left hand; reflexes absent, loss of power; vomiting and giddiness. This patient has now returned to her work at the laundry. She has regained her reflexes, but still walks slowly, and does not seem quite well yet.

A NOTE ON PAROXYSMAL ATTACKS OF DYSPNOEA, OCCURRING AT NIGHT, IN HEART DISEASE.

By HUGH BARBER, M.D.LOND.,

ASSISTANT PHYSICIAN, DERBYSHIRE ROYAL INFIRMARY; LATE MEDICAL REGISTRAR, GUY'S HOSPITAL.

I AM tempted to send brief notes of four cases of heart disease, in which asthmatic attacks occurred at night, by a letter of Dr. James Mackenzie¹ in the BRITISH MEDICAL JOURNAL, which contains the suggestion that such attacks must be seen at the time, in the night, to be fully appreciated. After several years' post-graduate appointments in the medical wards of a large hospital, I had hardly realized that such attacks existed, but in private practice, in a few years, I have met with four cases in which very definite paroxysmal attacks of dyspnoea came on suddenly in the middle of the night as one of the earliest symptoms of a diseased heart.

I have been present during many attacks, and during at least one in each case. The patient has gone to bed apparently in his usual state of health, or, at any rate, not having complained of feeling ill; he has been seized with an attack, usually between 1 a.m. and 2 a.m., which has compelled him to sit up, and has appeared to be similar in every way to the spasmodic attacks occurring in asthma. The attack has passed off in time, varying according to its severity and the treatment adopted; and next morning, at any rate in the earlier attacks, there has been little or no distress on waking. The pulse has varied, being rapid in the most severe attacks; the veins have not been excessively full nor the cyanosis extreme. The patient usually has had the sensation of an obstruction to his breathing, which he locates as underneath the lower part of the sternum. Sometimes a little expectoration has occurred, and been accompanied by slight relief; there has not been much tendency to flatulence, and carminatives have not given relief. There has been no pain, nor have paroxysms of angina pectoris occurred at any other time in these cases.

Morphine, usually combined with atropine, given hypodermically, has brought relief in ten to fifteen minutes in most cases. Nitrites have proved disappointing, and so has morphine administered by the mouth if the attack is well established. To prevent recurrence rest has seemed to be the essential remedy, but the paroxysms seem to recur as soon as the reserve force of the heart is again exhausted.

The features, common to all the cases, are that they occurred in elderly men in whom the heart was enlarged. In no case was there valvular disease which appeared to be primary, nor signs of an aneurysm. In two the high blood pressure might suggest chronic interstitial nephritis as a primary disposing cause to the heart trouble, but in the absence of albuminuria, or other signs of kidney disease, the attacks would not appear to be uraemic. In the three fatal cases it has not been possible to obtain a *post-mortem* examination, but I imagine the morbid anatomy of the condition, in all, has been degeneration of the heart muscle.

CASE I.—A man, aged 72 years; had been a casual labourer. First seen at 2 a.m. in March, 1906, with a typical asthmatic attack; he had never had any attacks previously, nor any serious symptoms of heart disease. He felt quite well on going to bed before the attack, and was in no distress in the morning. The heart was enlarged with the impulse $\frac{1}{2}$ in. external to the nipple line in the fifth space; there was a mitral systolic bruit. He rested in bed a day or two. He died in the street about eight weeks later, having had no other symptoms of heart trouble.

CASE II.—A man, aged 75 years, who was known to have an enlarged heart, but was attending to the business of a large mill. On December 27th, 1906, he went to bed in his usual health. I was called up at 1 a.m. to an asthmatic attack, which lasted about half an hour, and then subsided. Examined later