solution of nitrate of silver was dropped in, and atropine again applied. Belladonna paste was applied externally. Henceforth chloroform was regularly administered morning and evening. Cremer's ointment was substituted for the nitrate of silver. By the sixth day, there was no swelling of the lid; no discharge. The pupil looked bright, and the ulcer was healing. On this day, a belladonna rash broke out on the head, neck, and back. The belladonna was withheld, and next day the rash was gone. By the twelfth day, the child was well.

I quote this case more particularly to show the value of chloroform in such cases. Chloroform or ether should be employed in all cases where, in consequence of photophobia and muscular spasm, a proper examination cannot otherwise be made. Chloroform acts readily on the pupil. At first, it causes contraction; later on, dilatation, by its paralyzing effects on the motor oculi. It has appeared to me that chloroform is not only useful in enabling one to carefully and easily examine the eye and to apply local remedies in a way that would in many cases be impossible without it, but that its sedative or anodyne action continues for some time, often many hours, after the general effects pass away. Ringer states that Sir James Simpson had observed that a few drops of chloroform put on the palm of the hand and held near a photophobic eye, so that the vapour entered the eye, would enable it to bear the light without pain.

CASE IV.—In December 1874, a young lady came to me from the neighbourhood of Swansea, suffering from phlyctenular ophthalmia. Her eyes had been bad off and on for three years. She was obliged to leave school, and has to wear blue glasses. There was extreme photophobia; several small ulcers were to be seen on the margin of the cornea, numerous vessels, and the conjunctival papillæ were red and prominent. There was a total absence of anything like scrofulosis. Atropine was fully applied; one-eighth of a grain of belladonna extract was ordered to be taken three times a day, and Cremer's pomade was applied night and morning. In a fortnight, the eyes were nearly well. Atropine was now used but once a day, though the belladonna was increased to half a grain. In February, she returned home, and was instructed to use weak atropine solution and Cremer's ointment, and to take quarter-grain doses of belladonna. Wishing to prevent a recurrence of the disease, I advised her to use the remedies for some months. In June, the belladonna was reduced to one-eighth of a grain. The biniodide of mercury ointment was regularly applied until the end of June. The eyes were then quite well, and the conjunctival papillæ were no longer visible. From that day to this, now two years, she has had no further trouble.

CASE V.—The next case is a good example of the so-called scrofulous ophthalmia. The girl, ten years old, presented the general aspect of scrofulosis. She had been suffering from bad eyes for more than a year. The mother told me that she had consulted several doctors in this county and in Glamorganshire; that all sorts of remedies had been tried; that so many painful things had been done to the child that she quite dreaded coming to me. On inquiry, I found that nearly all the local applications that had been used had given her pain; also that she had been freely and repeatedly leeches and blistered. The large scars behind the ears, extending down the neck, showed how severely she had been treated. In fact, she was permanently disfigured.

I noted that there were extreme photophobia, a little muco-purulent discharge, ulcers on the cornea, and granular lids. Atropine was instilled; belladonna paste was ordered to be applied around the orbit, and belladonna and iodide of potassium were given internally. The lids were touched with lapis divinus, which is a handy and useful remedy composed of equal parts of sulphate of copper, nitrate of potash, and alum, moulded into sticks. Cod-liver oil and syrup of iodide of iron were also prescribed. This was on November 15th; on the 17th, *i.e.*, in two days, there was a marked improvement, and in six weeks, under this treatment, she perfectly recovered.

CASE VI.—Some time ago, a young man consulted me who, for some months, had been suffering from phlyctenular ophthalmia. He was getting no better, worse in fact, under medical treatment, and he was informed by his doctor that nothing further could be done. He inquired if it would be any use consulting an oculist; he was told that no oculist could do him any further good. A friend of mine happened to see him and sent him to me. On examination, several minute ulcers and small pustules were seen on the cornea, with a cloudy and dull pupil. There was photophobia and ciliary neuralgia. I told him that, if he would but promise to come and see me regularly, and would do exactly all that he was ordered, he would probably be well in three weeks; but that he must expect to have some corneal opacity left.

He promised, and he was most exact in performance. I applied my usual remedies, *i.e.*, atropine, Cremer's pomade, and a compressive

bandage, and gave him iron. At the end of a fortnight, he was almost well, and, before the end of the three weeks, was quite free from trouble. He was then told to discontinue all the remedies except the ointment, and this he was advised to use occasionally for some months. A faint cloudy opacity of the cornea remained, as was prognosticated.

In the treatment of most of the commoner forms of disease of the conjunctiva, cornea, and iris, the most successful practitioner will be he who, in addition to making a wise use of the neurotic and constitutional remedies of which we have spoken, also knows best how to apply local remedies. Let me briefly say that, where the conjunctiva is inflamed, metallic astringents are indicated, and that the best are nitrate of silver and acetate of lead.

When the cornea is inflamed or ulcerated, there is no local remedy equal to atropine; and metallic astringents must be avoided, though, in certain stages and under certain conditions, a weak red precipitate ointment is often of service; and, when this is indicated, there is no preparation, to my mind, so convenient, so useful, and so handy as Cremer's pomade.

When the iris is inflamed, atropine must be freely instilled, and, for this disease, it is the local remedy. When both conjunctiva and cornea are inflamed, the practitioner must learn how best to apply astringents to the mucous membrane and yet avoid irritating the cornea.

In conclusion, gentlemen, let me say that I shall have gained my object if the observations which have been addressed to you will induce the members of this Association to pay more attention to the diagnosis and proper local treatment of the commoner diseases of the eye; if it will but induce you to give up blisters, setons, leeches, and painful astringent lotions in but one disease, *viz.*, phlyctenular ophthalmia, and make you trust more to atropine, I shall be indeed amply repaid.

THE PATHOLOGY OF URÆMIA AND THE SO-CALLED URÆMIC CONVULSIONS.

By T. J. MACLAGAN, M.D., Dundee.

DR. MAHOMED'S interesting communication on the above subject is a notable addition to the literature of a vexed question. While recognising the agency to which he ascribes them as a possible cause of convulsions, it may be well to note one or two objections, besides those which he has indicated, which militate against his opinion that these convulsions are caused by minute capillary hæmorrhages. These objections are general and special.

General.—The general objections are these.

1. To separate convulsions associated with renal disease from convulsions not so associated, and to study their pathology apart from that of convulsions in general, is a mode of treating the subject which is calculated to exclude much valuable information regarding the general question, and to lead to narrow, and therefore probably erroneous, generalisations regarding the special subject of uræmic convulsions.

2. To separate convulsions from all the other nervous symptoms associated with uræmia, and to regard them as pathologically distinct, is a mode of treating the question which can scarcely commend itself to physicians or pathologists, unless a better reason be advanced for so doing than any which Dr. Mahomed has given us.

In connection with this point, I would note, what Dr. Mahomed does not seem to be aware of, that Traube years ago advanced the theory that the nervous symptoms of Bright's disease (convulsions among the rest) were indirectly caused by cerebral oedema. His idea was, that the impoverished state of the blood, and the coincident occurrence of cardiac hypertrophy, led to the exudation of water through the coats of the minute vessels; that there was thus produced anæmia of the cerebral substance; that the capillaries and veins were pressed upon by this effusion; and that this pressure gave rise to anæmia of the brain. To this anæmic condition of the brain he ascribed the nervous symptoms. To Dr. Mahomed's, even more than to Traube's, view of the part played by oedema in the production of the nervous symptoms of Bright's disease, there is the very grave objection that such symptoms are most common in that form of renal disease in which oedema is least apt to occur—the contracting granular kidney. In attributing the nervous symptoms only indirectly to oedema, and directly to anæmia, Traube greatly strengthened his theory; for, of all the causes of nervous symptoms, anæmia of the brain, or, to be more accurate, malnutrition of the nervous centres, is the most potent.

On this part of the general question, space forbids me to enter. I would, therefore, simply refer Dr. Mahomed and those interested in the question to Chapter XIV of my work on the *Germ-Theory of Dis-*

case. In this chapter (on the nervous symptoms of fever), the whole question of uræmia is fully discussed.

Special.—The special objections to Dr. Mahomed's theory may be noted as follows.

Granting the existence of minute capillary hæmorrhages (which has yet to be demonstrated), and granting their competence to produce convulsions, how do they act? There are only two possible ways: (a) by loss of blood, and (b) by physical pressure on the cerebral substance (for such minute hæmorrhages could not lacerate the brain-tissue as do larger ones). But the hæmorrhages, as described by Dr. Mahomed, are so minute that the loss of blood cannot be felt by the brain. They must, therefore, act by compressing the brain-tissue. Now, if one or two minute hæmorrhagic points can produce convulsions in this way, we ought frequently to find such symptoms in those cases, first described by Charcot and Bouchard, in which minute capillary aneurisms exist in large numbers in the brain, producing, in virtue of their number, greater pressure than could be caused by a few hæmorrhages not exceeding in size the individual aneurisms. But such is not the case. If Dr. Mahomed will refer to the published observations of the French pathologists, he will find that miliary aneurisms, which differ from his hæmorrhages only in having the blood surrounded by the aneurismal covering, may exist in the brain in dozens, and even in hundreds, without causing any such symptoms as those which he attributes to minute capillary hæmorrhage. A certain allowance, I am aware, must be made for the more sudden occurrence of the hæmorrhagic pressure; but this I believe to be more than counterbalanced by the large number of the aneurisms. These aneurisms, too, occur in all parts of the brain: the optic thalami, the corpora striata, the convolutions, the pons Varolii, the cerebellum, the centrum ovale, the middle peduncles of the cerebellum, the peduncles of the brain, and the bulb.

To sum up: the objections to Dr. Mahomed's views are, first, that they lay aside as inadequate an agency which is at work in every case of uræmia, and which has been proved to be a potent cause of convulsions—viz., malnutrition of the nervous centres; and, second, that they substitute for this an agency whose existence has not been proved, and whose competence to produce convulsions is, at the least, doubtful.

ON A CASE OF DOUBLE HEMIPLEGIA, WITH CEREBRAL SYMMETRICAL LESIONS.

By THOMAS BARLOW, M.D., B.S.,

Assistant-Physician to Charing Cross Hospital, and to the Children's Hospital, Great Ormond Street.

W. S., A BOY aged 10, was admitted into the Hospital for Sick Children, Great Ormond Street, under the care of Dr. Dickinson, on December 16th, 1875. There was no history of rheumatism, and no suspicion on the part of his mother that the boy had heart-disease.

Four months before admission, according to the statements given, he must have had an attack of right hemiplegia. Whilst holding the baby, he suddenly burst out crying, his face was drawn, and he fell down. But he did not lose his senses, and within a quarter of an hour was able to walk. He was put to bed, and at that time he talked naturally and swallowed without trouble. But next morning, he had lost his speech; he could only say "Haw-haw"; he had also difficulty in swallowing. His face was drawn. He could not grasp with the right hand, and he dragged the right leg when put on his feet. In ten days, he was greatly improved. The leg improved before the arm. The speech had returned on the tenth day; but occasionally he made a mistake, gave the wrong name for a boy, and did not seem always quite to understand what was said to him.

In a month's time, he seems to have quite recovered. He was then able to go errands for his mother, and he discharged them correctly. He was very pale. Six days before admission, he got an attack of left hemiplegia. It was noticed that he was very irritable, and could not bear the noise made by the other children; he cried and sobbed a great deal. Next morning, he could not use the left hand, had difficulty in swallowing, and had quite lost his speech. His mother does not know whether the face was drawn or not. No notice was taken of his legs till five days afterwards, and then it was found that he could stand without support. When brought to the hospital, he cried just like a patient with senile softening, directly he was approached. But after a few days, he was amenable to investigation. As he changed very little, I may summarise his condition whilst in the hospital.

The only approach he could make to a voluntary articulate sound

was "Ah". He could cry vigorously; there was no lack of voice. From his admission, he appeared to understand all that was said to him. When asked his age, he counted ten on the questioner's fingers; counted four, when asked how many brothers he had; and so forth. He made signs for what he wanted, and the signs were correct.

From the first, he was able to write his name when asked, and after a few weeks would answer in writing any question that was put to him.

He could not be induced to show his upper teeth, nor to protrude his lips, nor to smile when he was told. But his face was not expressionless. He occasionally smiled involuntarily. There was at first the slightest possible flattening of the left naso-oral ridge, but this soon passed off, and there was no loss of symmetry.

He could not protrude his tongue at all. There was no wasting of the tongue. Both sides responded to Faradism. There was no deviation of the uvula; no dropping of either palatine arch; no regurgitation of fluids. The palate was sensitive. His swallowing of solids was very peculiar. He was fed best when lying on his back. A large morsel succeeded better than a small one. It seemed very gradually to find its way to the fauces, but without any arching up of the tongue. When once past the arch of the palate, there was at no time any difficulty. There was no choking nor regurgitation; nor did the boy push the morsel down to the back of the pharynx, as is seen sometimes in cases of labio-glosso-pharyngeal paralysis.

He never made any attempt to chew. He did not use his pterygoids at all; but on one occasion, after much persuasion, he was induced to give my finger a bite between his molars, and the masseters were felt to contract. The sensory division of the fifth nerve was quite natural. There was no ocular palsy; the optic discs were natural. The sense of taste and the sense of smell were natural.

To sum up the cerebral condition, there appeared to be loss of voluntary motor power over the muscles concerned in articulation and the first part of deglutition.

As to the limbs, there was at first weak grasp with both hands, the left the weaker. There was slight wasting of both upper limbs. But all this greatly improved in three weeks' time. At no time was any rigidity elicited by flexion. Both arms responded well to Faradism. At the time of his admission, he was able to draw up his lower limbs equally; and in a month, when he was allowed to walk, he did so without dragging. There was no diminution of irritability to the Faradic current.

There was no perversion of sensation. For a couple of days after admission, he had retention of urine, but this did not recur. There was no trouble with the sphincter ani. The cause of his hemiplegia was not far to seek. He had a double aortic murmur, with considerable hypertrophy and dilatation of the heart.

He was in the hospital less than two months. His condition as to speech and swallowing did not alter in the least, though his limbs improved. No fresh nervous signs appeared.

He died from the results of his aortic regurgitation. At the *post mortem* examination, the right and left aortic valves together measured the same width as the posterior aortic valve. They were partly united along one edge, and there was a frænum uniting two contiguous cusps. This was probably a congenital condition. Besides this, there were vegetations partly calcified at the free margins of all three valves, and there were a few recent vegetations at the base of the aortic curtain of the mitral. There was dilatation and hypertrophy; and there were some oldish thrombi in both auricles. As to the other viscera, there was some induration with old infarcts in the lungs, a nutmeg liver, infarcts in the spleen and kidneys, and some effusion, probably passive, in the peritoneum.

As to the brain, no disease of the vessels forming the circle of Willis could be detected; but in the left middle cerebral, an inch and a quarter beyond its origin, three calcified nodules, each rather less than a millet-seed, were found. Two branches, traced from this vessel, were also found to present calcified spots. There was no actual occlusion, but the calibre was obviously diminished by the above condition. No aneurism was detected and no plug of fibrine found. In an identically corresponding position on the right side some similar calcification was found, though not quite so much in amount. These vessels were traced into small oval regions of softening, each of which might be covered with a shilling. On each side, these regions consisted of the lower end of the ascending frontal and the hinder end of the middle and inferior frontal convolutions. These areas were pale, buff-coloured, slightly depressed, slightly softer than the surrounding brain-substance. Reckoning from the surface, they were less than a quarter of an inch deep, *i.e.*, they involved the cortical and a little of the subjacent white substance.

The rest of the brain, so far as could be made out by naked-eye inspection, was natural.