Cystotomy was neither difficult, if performed within two to three weeks, when the average patient was quiet, or dangerous if aqueous was not lost, thus preventing the prolapse of vitreous into the anterior chamber. A simple extraction, or one with peripheral iridotomy, was to his mind the ideal operation. By causing adhesion of the anterior and posterior parts of the lens capsule it prevented capsule in the wound, and, even if iris prolapse occurred twenty-four hours afterwards, and this adhesion had taken place, this, the most serious complication of the operation, was avoided.

PARESIS OF THE ACCOMMODATION AS A LATE SEQUELA OF ENCEPHALITIS LETHARGICA.

BY

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MILD attacks of encephalitis lethargica are probably much more frequent than is generally believed. The symptoms are very similar to those of typical encephalitis, but are transient and leave no permanent ill effects. They are often incorrectly diagnosed as influenza, but the ocular symptoms should indicate the true diagnosis. Diplopia, often very transient, and a slight paresis of certain extra- and intraocular muscles will always be found.

In one case under my observation in a child double vision was not complained of, but the parents noticed that the boy constantly misjudged distances and had difficulty in directing food to his mouth. Affections of the ocular muscles are probably the most common of all the symptoms of this disease in the acute stage of both severe and mild attacks. As a final result or sequela they are less frequently present.

Many cases have been recorded in which paralysis or paresis of the accommodation has been found in association with palsies of the external ocular muscles. As an isolated symptom it is much less frequent, and a series of such cases having recently come under my observation, it seemed advisable to place them on record. Through the courtesy of Dr. Johnstone Jervis, the medical officer of health for Leeds, I have been able to obtain particulars of twenty-four cases of encephalitis lethargica occurring in the Leeds area in the first six months of this year. Ocular symptoms were present in all in the early stages, and in some persisted for long periods. Most of the cases were serious, and the diagnosis was made without much difficulty. In the case of four patients who came under my care complaining of defective vision, the original illness had been mild, followed by a more or less complete return to good general health; in only one had a diagnosis of encephalitis been made.

The brief notes of these cases are as follows:

T. R. B., male, aged 28, was seen first on March 10th, 1924. Six weeks previously he had been ill for a few days with severe headache and dilatation of the left pupil; on recovering he found that he was unable to read ordinary print but his distant vision was unaffected. The dilatation of the left pupil gradually passed off and the pupils were now equal. Vision was 6/6 in each eye with normal refraction; he could read J.1 easily at 14 inches with +2.5 D. sph. for each eye. The condition had improved in June, so that he was able to read J.1 with +2.0 D. sph.

D. B., female, aged 16, seen on March 24th, 1924. There had been a sudden illness seven days previously, with severe headache and diplopia, lasting five days; she was now unable to read. The vision was 6/6 in each eye, refraction normal. Paresis of accommodation was present; she could read J.1 with +3 D. sph. On June 12th she could read J.1 with +2.5 D. sph.

E. M., male, aged 32, seen on April 28th, 1924. Four weeks previously he had had a severe illness, with headaches and diplopia, for about a week, and encephalitis lethargica was diagnosed. Recovery was gradual, and he now feels perfectly well, but is unable to read. The pupils are rather large, but reacting and equal. There is a well marked paresis of accommodation, corrected by +2.5 D. sph. The right eye is hypermetropic, vision 6/18; the left is emmetropic, vision 6/6. On June 20th the condition was unaltered, but on July 14th he was able to read J.1 with +2.5 D. sph.

W. F. T., male, aged 12, seen on May 5th, 1924. About a month previously he became drowsy and had diplopia. Subsequently he was heavy and very slow mentally, and was inclined to sleep frequently during the day. He complained of inability to read any

except very large print. The vision was 6/6 in each eye, refraction normal; he required +1.5 D. sph. to read J.1. On June 19th he was much brighter, and read J.1 with +1.0 D. sph.

There has been progressive but slow improvement in these cases.

Paresis of the accommodation is apparently most common in the mild cases of encephalitis lethargica, and it has not been observed as a sequela in any of the severe cases occurring in this district, except in association with such other ocular symptoms as inequality of the pupils, defect in convergence, or a complete third nerve palsy. It bears some resemblance to the paralysis of the accommodation which occurs in diphtheria epidemics, in that it appears in mild and somewhat atypical cases. It is well known to ophthalmic surgeons that a definite history of diphtheria is often very difficult to obtain in post-diphtheritic cycloplegia—often the only complaint is of a mild transient sore throat during a diphtheria epidemic—or where diphtheria has been present in the same house or district. In three of my cases the original attack was so slight as not to direct attention to the real nature of the illness. It is on account of the diagnostic significance of this partial cycloplegia in association with the antecedent history that I have brought these cases to your notice.

Mr. HARRISON BUTLER (Leamington) had seen several cases of encephalitis during the previous six months; in fact, at one time several cases a day came to the Birmingham Eye Hospital. In all there was a history of "influenza," followed by the paralysis of an eye muscle. Unfortunately he had seen four cases in which the optic nerve was affected—three of retrobulbar neuritis and one of optic neuritis. Two were followed by almost total blindness and two made a complete recovery.

RELATION OF THE BLOOD UREA TO DIABETIC COMA AND TO THE DURATION OF DIABETES MELLITUS.*

BY

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As the blood urea concentration in cases of eclampsia and of diabetic coma is at present under discussion¹ it may be of interest to publish some results which were obtained in a recent investigation on certain blood constituents in diabetic patients under treatment by insulin. Two cases of coma are described, and a table is given showing the blood sugar and urea content of fourteen patients, in whom the duration of the diabetes mellitus was known.

The estimations of blood sugar were made by Wallis and Gallagher's modification² of the Folin-Wu method; those of blood urea by the method of Twort and Archer.³

CASE I.

A married woman aged 56. Her aunt had suffered from diabetes mellitus, and she had noticed the symptoms in herself six years ago. She had been treated for four years. On November 17th, 1923, she had pain in her knee; on November 19th vomiting and loss of appetite. On the night of November 22nd she lost consciousness, and was admitted on the afternoon of November 23rd semi-conscious, but unable to speak. The temperature was 97.6° F., and the urine contained much sugar and acetone.

One hour after admission, having had nothing but water during the previous twenty-four hours, her blood contained 466 mg. of sugar and 84.2 mg. of urea per 100 c.cm. On the following morning, after a diet of water only and 90 units of insulin, the last 20 units having been given four hours previously, the figures were respectively 268 mg. and 44.7 mg. per 100 c.cm.

On November 27th, on a diet containing 24 grams of protein and 110 units of insulin in the twenty-four hours, the figures were 300 mg. and 30.9 mg.

On December 11th, on a diet containing 51 grams of protein and 40 units of insulin, the figures were 168 mg. and 24.1 mg.

On January 5th, 1924, on a diet containing 63.5 grams of protein and 40 units of insulin, the figures were 215 mg. and 21.9 mg. per 100 c.cm. of blood.

It will be seen, then, that in this case the initial high blood urea fell more or less concurrently with the blood sugar as the result of treating the diabetes.

* Extract from a report to the Science Committee of the British Medical Association.

CASE II.

An unmarried woman, aged 20, stated that appetite and thirst had for several months been unusually great. One month previously she had noticed loss of weight. For twelve days she had ceased to work owing to weakness. On January 2nd, 1924, she felt definitely unwell. On January 4th she "fainted" and was admitted to hospital sufficiently conscious to attempt, unsuccessfully, to answer questions. The knee-jerks and abdominal reflexes were then absent, but returned before the second examination of the blood was made. One hour after admission, having had an almost protein-free diet during the previous twenty-four hours, the concentrations of sugar and of urea in the blood were respectively 620 mg. and 27 mg. per 100 c.cm. On the following day, after an enema of olive and castor oils, the administration by the rectum of half a pint of water every hour, and a total of 40 units of insulin, the figures were 250 mg. and 41.4 mg.

Thus, in the second case the concentration of blood urea was within the normal limits and actually rose as the blood sugar concentration decreased and the clinical signs of coma disappeared.

Since the blood urea concentration is not necessarily increased in diabetic coma, it follows that the kidney threshold for urea is not necessarily raised. One or two estimations done some time ago on the blood of eclamptic patients showed a normal concentration of urea. These results and those of De Wesselow,⁴ who studied the coma of nephritis, suggest that coma is not due to hyperuraemia. Among the possible causes are an increase in the inorganic phosphates, a relative alteration in the amounts of the inorganic constituents, and an alteration in the hydrogen-ion concentration of the blood. I am inclined to suspect some decomposition product of urea, but the estimation of such products is difficult.

The table shows the blood urea concentrations of patients who had suffered from diabetes mellitus for various periods ranging from three months to seventeen years. Only those patients are included whose histories were definite and intelligent. Case 13 is the first case of coma described above, and the figures given are, as in all the other cases, those obtained on the first examination after admission. Case 14 is the second case described. In all the other cases the blood urea remained constant under diabetic treatment in hospital.

TABLE.

Case.	Sex.	Age (years).	Duration (years).	Blood Sugar (grams per 100 c.cm.).	Blood Urea (milligrams per 100 c.cm.).
1	M.	27	1/2	0.098	28.1
2	M.	28	1/4	0.088	25.0
3	M.	40	2	0.137	31.5
4	M.	63	17	0.113	25.6
5	F.	16	2	0.207	23.5
6	M.	66	1/4	0.173	29.9
7	F.	23	1 1/2	0.336	34.7
8	F.	43	2 1/2	0.137	31.5
9	M.	46	3	0.138	48.7
10	F.	52	3 1/2	0.242	28.0
11	F.	20	4	0.250	41.4
12	F.	20	1	0.108	24.5
13	F.	56	6	0.466	82.2
14	F.	20	1/4	0.620	27.0

It is of interest to note that the patient Case 4 in this table had a systolic pressure of 160 mm. Hg taken over the brachial artery by the auscultatory method, but no arterial thickening or proteinuria.

It seems certain that the duration of diabetes mellitus does not necessarily raise the concentration of blood urea. From this it may be inferred that the disease does not lessen the excretory power of the kidneys for urea.

The research on which this note is based was carried out at St. Bartholomew's Hospital. The author is deeply

indebted to Sir Frederick Andrewes, F.R.S., to Dr. Mackenzie Wallis, and to the members of the staff who allowed access to their patients. Towards the expenses of the research a grant was made by the British Medical Association.

Summary.

1. The concentration of blood urea in diabetic coma is not necessarily proportional either to the depth of the coma or to the concentration of blood sugar. Low concentrations have been found in the coma of eclampsia.

2. The concentration of blood urea does not necessarily rise in chronic diabetes mellitus. Diabetes mellitus does not raise the kidney threshold for urea.

REFERENCES.

- ¹ BRITISH MEDICAL JOURNAL, 1923, i, 277. ² *Lancet*, 1920, ii, 784. ³ *Ibid.*, 1923, i, 1102. ⁴ *Quart. Journ. Med.*, 1923, 16, 341.

ACUTE PNEUMONIC PULMONARY TUBERCULOSIS
TREATED BY ARTIFICIAL PNEUMOTHORAX.

NOTES ON TWO CASES.

BY

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It is a matter of general experience that in a large proportion of cases of acute caseous tuberculosis (lobar pneumonic type) there is a fatal termination within three months of the onset of illness, while nearly all the remainder, who have succeeded in overcoming the initial toxæmia, succumb to fibro-caseous disease and broncho-pneumonic extension within eighteen months. The very few who are alive at the end of two years tend to be hopelessly incapacitated by reason of extensive fibro-caseous and fibroid pulmonary disease. With the exception of the first case recorded below we do not know of one person, undoubtedly afflicted with this form of pulmonary tuberculosis, who is in good health and able to do Grade III manual work more than two years after the onset of his disease.

Radiographic examination of this type of case shows that, in addition to the primary "pneumonic" focus, there is, within a few weeks, considerable secondary bronchopneumonic involvement of the remainder of the same lung, and also similar implication, to a variable extent, of the opposite lung, usually about its middle zone.

It is reasonable to suppose that toxæmia will be limited, and further spread of disease to the opposite lung be checked, so soon as the primary lung can be collapsed by artificial pneumothorax.

Just as in the treatment by artificial pneumothorax of ordinary fibro-caseous tuberculosis the presence of definite (even considerable) disease in the "sound" lung need be no deterrent, if for other reasons the treatment is deemed advisable, so in the acute caseous form artificial pneumothorax should be attempted provided there be only a small or moderate (up to one-third) involvement of the "healthy" lung.

This almost inevitable secondary implication of the "healthy" lung is, however, one very cogent reason for beginning the treatment by artificial pneumothorax as early as possible, and another reason, equally cogent, is the fact that if treatment be delayed more than a few weeks the formation of tough pleural adhesions may prevent satisfactory collapse of the affected lung, and the efficacy of the treatment may be correspondingly impaired. It is not usually possible to diagnose this form of tuberculosis with certainty till the second or third week, since it is likely to be regarded as ordinary pneumonia till about the fourteenth day, when the absence of crisis, the swinging