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.

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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 bronchopneumonic 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

temperature, and the evidence of softening may lead to a correct diagnosis; the discovery of tubercle bacilli in the sputum may be expected towards the end of the second week, but their appearance may be delayed for some time further. In any event we consider that the diagnosis should be established and treatment commenced in nearly every case by the end of the fourth week, and in some cases quite a week earlier.

At the present time there has not been recorded a sufficient number of cases of acute caseous tuberculosis treated by artificial pneumothorax to justify an opinion as to what percentage can be restored to health by this means, but we venture to publish a short account of our two first cases in the belief that the present very high death rate in this class of case will be reduced very materially, provided artificial pneumothorax treatment can be employed sufficiently early in the disease.

CASE I.

A. G., a schoolboy, aged 16 years, was always quite healthy till January 20th, 1922, when he contracted "influenza." He was treated by Dr. Milne of Streatham, who kept him in bed for two weeks; after a further week's convalescence the boy felt perfectly well and returned to school. The following day, February 11th, he shivered and had three haemoptyses, totalling about twelve ounces of blood. He was put to bed and Dr. Milne noted the rapid development of signs of consolidation in the upper half of the right lung, and, later on, increasing infiltration of the right base. At first the clinical picture resembled lobar pneumonia, but no crisis occurred, the temperature began to swing widely, and by the middle of March, in spite of the fact that no tubercle bacilli were found in the sputum on three occasions, Dr. Milne was satisfied that the case was one of acute pneumonic phthisis. On March 23rd the late Dr. Arthur Latham was asked to see the boy with a view to deciding whether artificial pneumothorax should be attempted. Dr. Latham agreed that this treatment would afford the only possible hope of recovery, and on March 24th the boy was admitted to St. George's Hospital, x-ray photographs being taken on his way from the receiving room to the ward. A few weeks later the onset of Dr. Latham's fatal illness precluded his further attendance at the hospital, and the subsequent treatment of the case devolved on the present writers.

Condition on admittance (March 24th).—A pale, wasted boy, with hectic flush, dirty tongue, injected fauces, and frequent cough; he was obviously very ill indeed. Temperature 102° , pulse 120, respirations 28. The right lung showed diminished expansion, especially of the upper half, with increased tactile vocal fremitus; solid dullness to percussion over the upper half, back and front; vocal resonance greatly increased, some aegophony. The breath sounds were tubular with many hard crackles and pleural creaks over the upper and middle lobes. The lower lobe showed impaired resonance and many medium and fine crepitations. The left lung was hyper-resonant and emphysematous, with a patch of fine crepitations in front of the axilla and a band of hard crepitations to the left of the vertebral column (? conducted from the other side). The heart, which was dilated, gave sounds of poor quality; there were no murmurs.

A skiagram showed much fibrosis with excavation of the upper and middle lobes of the right lung, with pleural involvement, and fine mottling all over the lower lobe. There was emphysema and general tuberculous infiltration of the left lung, chiefly in the lower lobe, and most marked just external to the cardiac impulse.

Subsequent Course of Case.—It was considered advisable to collapse the right lung fairly quickly. The first injection was given on March 25th; no difficulty was met with, and 320 c.cm. of oxygen were introduced. The initial manometer readings were -4, -8 cm., and the final -3, -6 cm. By the end of a fortnight five injections had been given, nitrogen being used after the first day, and satisfactory collapse had been achieved, the final pressure after treatment was maintained at roughly -1, +3 cm. A persistent adhesion from the apex to the first rib did not interfere with the collapse. The sputum, which after the first injection of gas was copious and loaded with tubercle bacilli, ceased completely when collapse had been achieved, and there has been none since April, 1922. Constitutional improvement was slow but steady; the temperature did not reach the normal for five months and was not stable for seven months; the pulse remained high for several months, but was down to 70-90 a minute by March, 1923. The weight rose in eighteen months from 99 lb. to 121 lb., but has fallen a little since manual work has been undertaken.

After nine weeks of treatment, pleurisy with effusion developed on the right side, with severe constitutional disturbance for several weeks. This necessitated gas replacement of the fluid, which took several weeks to re-form in any amount after the chest had been completely emptied. Finally, the fluid, though sterile, became purulent, and in July, 1923, was so thick that it could not be withdrawn through any ordinary aspirating apparatus. Accordingly 5 c.cm. of Gauvain's fluid* was injected to liquefy the effusion, and a few days later 18 oz. were removed with success. Since September, 1923, the boy has been at a sanatorium, where, we are informed, it was once necessary to liquefy and remove a thick effusion as described above, but since then no fluid has formed and ordinary gas refills are performed every fortnight or

so. He is now doing Grade III work (pick and shovel and drawing a truck), is afebrile, and without cough; the signs in the left lung have long since disappeared and he looks and feels perfectly well.

CASE II.

W. H., a schoolboy, aged 15, was admitted to St. George's Hospital on January 1st, 1923, with a history of the sudden onset of illness with shivering, pain in the right chest, cough and expectoration, ten weeks before. Physical examination, as well as x-ray photographs, showed an almost identical condition to that described in Case I, except that there was rather more implication of the left lung. The same treatment was adopted, but owing to the existence of numerous tough adhesions only partial collapse of the right lung was obtained, in spite of the fact that the intra-pleural pressure was raised at each treatment to +30 and +40 cm. Nevertheless progress has been, on the whole, satisfactory: the fever has disappeared, there is neither cough nor sputum, and moderate walking exercise has been permitted from time to time without obvious ill effect other than an increase in respiration rate. By reason of the pleural adhesions it is now (sixteen months after the beginning of treatment) impossible to introduce any more gas, and the lung has partly re-expanded, though with a very thickened pleura and marked falling in of the chest wall.

We do not maintain that this case is so satisfactory as the first one, and it is too early yet to say what may be his ultimate capacity for work or exercise; but we think that if he fails to make a good recovery it will be because he was not got at early enough to achieve full collapse of the damaged lung before the presence of multiple adhesions rendered this impossible.

A CASE OF STREPTOCOCCAL MENINGITIS TREATED WITH SERUM: RECOVERY.

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RECOVERY from meningitis due to a haemolytic streptococcus is sufficiently rare to warrant placing the following case on record.

A girl, aged 10, of delicate constitution, had been subject to mucous catarrh of the bowel and mild attacks of acidosis associated with an irritable patchy dermatitis which were quickly relieved by treatment. Adenoids, but not the tonsils, were removed during 1922.

On March 14th, 1924, she was sent home from school with mild tonsillitis and a temperature of 99° . On March 18th she complained of earache; the tympanic membrane was hyperaemic but not bulging; on March 19th the temperature was 100° and the pain in the ear continued. On March 22nd the dermatitis was very troublesome, but the temperature was 98° F. On March 24th she had pain in the right ear and the breath smelt of acetone. Vomiting occurred, but was relieved by alkali. On March 25th the pain in the ear ceased and she was better until March 30th, when persistent vomiting recurred and the vomit was thought to contain pus. Mr. E. S. Steward of Harrogate examined the ear, but did not consider there was any indication for surgical treatment. There was slight hyperaemia of the tympanic membrane, but no swelling or vertigo; the ear appeared to be settling down. A small amount of pus was squeezed from the right tonsil.

On March 31st she complained of severe frontal headache, said she saw two of everything, and was confused and apathetic. The temperature was 104° F. and she was seriously ill; the vomiting continued. The neck was stiff, with pain when the head was bent forwards. The knee-jerks were not obtained and Kernig's sign was present. Exploratory lumbar puncture yielded 20 c.cm. of turbid fluid under increased pressure; polymorphs 60 per cent., lymphocytes 40 per cent. No organisms were seen in direct smears. After the puncture the headache was relieved and the temperature dropped to 99.8° .

The headache and general symptoms returned next day and the temperature was 103° F. After the removal of 20 c.cm. of fluid 15 c.cm. of antimeningococcal serum were given intrathecally and 10 c.cm. subcutaneously. This was followed by great restlessness, much generalized pain, and considerable increase of the headache. Towards the end of the next day (April 2nd) there was distinct improvement and the temperature dropped to 100° . The pathological report upon the fluid was: "Fluid turbid, the turbidity being due to pus cells composed of polymorphs 64 per cent., lymphocytes 36 per cent. No blood present. Gold sol reaction nil. Globulin increased. Culture on solid media gave a moderate growth of a short chained streptococcus which was markedly haemolytic in character." Lumbar puncture was again performed;

*Camphor 1 part, thymol 2 parts, ether 3 parts.