These are generally large, situated in the neck, and on the posterior aspect of the tube. Those occurring at the tracheal bifurcation are smaller and on the anterior aspect, as in Dr. Norman Moore's case quoted above.

Beyond the causation of a far-spreading abdominal tuberculosis, perforation of the oesophagus by a caseous gland seems to give rise to no symptoms and its presence cannot be diagnosed during life.

AN IMPROVED HYPOBROMITE PROCESS FOR THE ESTIMATION OF UREA.

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DURING a recent research I have had occasion to make numerous estimations of urea. I employed the hypobromite process with Dupré's apparatus, but with the necessary modifications; the method to be described below is applicable to any form of apparatus in which this process is employed.

Whilst examining the literature upon this subject I found the following statement in Halliburton's textbook of *Physiological Chemistry*, 1891, p. 839:

From 1 gram of urea 0.46 gram (=372.7 c.cm.) of nitrogen is obtained. In practice, however, it is found that only 254.3 c.cm. are obtained, except in diabetic urine, in which the urea yields nearly the normal amount of nitrogen.

This suggested that possibly an improvement might be made in the ordinary method of estimation for clinical purposes by converting the urine to be examined into an artificial "diabetic" urine. This was done by the addition of 2 c.cm. of a 25 per cent. solution of glucose to the 5 c.cm. of urine used in Dupré's method, which produces a urine containing about 7 per cent. of sugar. The glucose was of the ordinary commercial type, and was tested by the hypobromite method, but no gas was evolved which could invalidate the process.

By comparing the readings thus obtained with those recorded by the same urine without the addition of sugar, a practically constant excess of one-eleventh (9 per cent.) was noticed. Details of a few actual observations are given below. Control estimations were also made with a solution of chemically pure urea of known strength, but the evolution of nitrogen in this case is unaffected by the addition of glucose. This fact indicates that the explanation of the process in the case of urine is by no means simple.

In the table given it will be noted that when any specimen has been tested more than once (*a, b, c, d*) and varying readings have been obtained, the lowest of the "glucose" readings is always higher than the highest of the "normal" readings.

No. of Case.	I.	II.	III.	IVa.	b.	c.	d.	Va.	b.	VI.	VII.	VIII.	IX.
"Normal" urine ...	0.75	0.85	1.5	1.55	1.6	1.6	1.55	1.8	1.8	2.4	2.85	1.9	2.3
"Glucose" urine ...	0.83	0.9	1.6	1.7	1.75	1.7	1.7	1.95	1.9	2.72	3.15	2.03	2.5

This procedure is so simple, and follows so obviously on the statement already quoted from Halliburton's work, that it may not be new. I can, however, find no evidence of its previous publication.

I can give no reason for the difference in the evolution of nitrogen of urea in watery solution and in urine, nor for the reason why glucose increases the nitrogen output. Possibly its action may have a parallel in that of manganese dioxide in the process of making oxygen from potassium chlorate. Undoubtedly nitrogen is evolved from a "glucose" urine at a lower temperature than from a "normal" urine, as the level in the calibrated tube is much sooner established in the former than in the latter case. The method may in practice be further simplified as follows:—Place in the urine tube of the apparatus a piece of glucose as large as a haricot bean and about 2 c.cm. of water; dissolve the glucose by gentle heat, cool, add 5 c.cm. of the urine to be tested, and proceed in the usual manner.

I am indebted to Dr. W. S. Lazarus-Barlow, Director of the Westminster Hospital Pathological Laboratory, for much help and advice in the prosecution of the above research.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, ETC.

CASE OF ASCITES DUE TO HYPERTROPHIC CIRRHOSIS OF THE LIVER CURED BY OPERATION.

K. W., aged 53, married, was admitted to the Perth Public Hospital in April, 1901. The patient, who had borne ten children, rearing six, and who had had five miscarriages, had enjoyed good health until two years before, when she had an attack which was believed to be due to impacted gall stones. The jaundice and symptoms persisted for twelve months. She was then residing in Melbourne, Victoria, and was treated at home and at the Women's Hospital, Melbourne. Twelve months after the onset of her illness ascites and hæmatemesis occurred, and she was tapped at the Women's Hospital, Melbourne, 14 pints being drawn off. Six months afterwards she came to West Australia for a change. After coming to Perth she was tapped four times, at weekly intervals, by a doctor outside the hospital.

During her stay in the Perth Hospital she was tapped ten times, a large quantity of fluid being withdrawn each time. The fluid rapidly reaccumulated, and she required retapping sometimes in a week, sometimes in a fortnight. After the tappings the hard, rounded edge of a hypertrophic cirrhotic liver could be felt. There was no evidence of cardiac or renal disease. She denied all history of alcoholism. There was no evidence of syphilis.

She was transferred to the surgical side on October 2nd, 1901, and on October 4th her abdomen was opened in the mid-line above umbilicus, and ascitic fluid evacuated. The liver edge extended below the costal margin, and on examination the liver was found to present the appearance and consistency of an ordinary hypertrophic cirrhosis.

The surface of the liver was firmly scrubbed with a sponge, also that of the adjacent parietal peritoneum. The omentum was sutured in six places to the parietal peritoneum around the abdominal incision. An incision was made above the pubes, and a glass drainage tube inserted, which was kept in for several days. When the abdomen was opened it was found that adhesions had formed at several sites where former tappings had been made.

The after-history was uneventful. The patient never had a bad symptom after the operation; the fluid showed signs of reaccumulating for a few weeks, but was then absorbed. She left the hospital about a month afterwards apparently in good health, and has presented herself for examination several times since. The last time she was seen was nine months after the operation. She then stated that she had not felt so well for years. There was no ascites. The hard edge of the liver could still be felt just below the ribs; she was otherwise apparently in perfect health, and was able to attend to her domestic duties.

After the ascites had disappeared there was nothing in her appearance to suggest alcoholism, and I am inclined to think that the cirrhosis of the liver was due to a long-standing impacted gall stone which was passed some eighteen months before operation. I think that the length of time—nine months—since operation justifies one in regarding this as a cure.

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HYDATID OF THE LIVER.

On the morning of August 30th, 1902, I was asked by Miss McNeil, M.D., of the American Mission of this station to see a case in her hospital of obscure abdominal tumour which she had had under observation for two days previously.

A well-nourished, healthy-looking man, aged 50, had come to the Mission Hospital on August 27th complaining of a swelling in the belly. He stated that two and a half years before he had suffered from fever for four months, for which he was treated by native remedies and got relief. He then had a pain in the epigastrium, which continued for about two months, when he was again relieved by native remedies. After this his belly began to swell, and has continued to do so for the past two years.

An examination on August 30th, 1902, showed an oval painless swelling, moving with respiration in the epigastric and umbilical regions of the abdomen. This swelling was roughly 7 in. in the vertical and 6 in. in the trans-