CASE II.—T. G., aged 59. Both eyes affected: the right began at the age of 46, and the left at 49. The right lens is quite ripe and pearly white in colour. I removed the right lens on December 18th; there were some adhesions of the iris to the anterior capsule, which was very dense, so I delivered the lens in its capsule complete, after performing an iridectomy. A small bead of vitreous escaped as the lids were being closed. The anterior capsule of the lens was dense and pearly white and about the thickness of tissue paper; the posterior capsule was much thinner and more translucent but whitish. The result of the operation is excellent. The left lens was removed by me on October 4th, 1902, and with + 10 D he sees $\frac{1}{2}$. There is no posterior mischief.

CASE III.—J. B., aged 59. Both eyes affected; the right began at the age of 55, in January, 1898, and the left shortly after. I removed the left lens on June 18th, 1901; he had had an injury to this eye many years before which had set up iritis and resulted in adhesions of the iris to the lens, necessitating the removal of the lens by the vectis. Some vitreous escaped, and his vision now in this eye is $\frac{2}{3}$, and there is some displacement of the pupil. The right lens I removed on September 26th, 1902; six weeks afterwards with + 11 D he read $\frac{1}{2}$. He has no fundus mischief.

CASE IV.—J. S., aged 53; both eyes affected: left began at age of 48, and right at 51. I removed the left lens in March, 1901. Immediately after the section some vitreous presented and I removed the lens with the vectis in its capsule. He has a beautifully clear pupil, and with a + glass he saw $\frac{1}{2}$ until May, 1902, when the vision began to fail, and now with + 6 he sees only $\frac{1}{3}$. There has developed recently a separation of the retina at the lower half of the fundus and a patch of choroidal atrophy. On September 23rd last I removed his right lens, and on November 8th he read $\frac{1}{2}$ with + 6 D. He has a good pupil and no posterior mischief.

CASE V.—J. W., aged 40, presents in both eyes the appearance of early posterior cortical cataract described above. The defect in his sight he first noticed at the age of 38. He is still at work. The fundi are normal.

CASE VI.—D. McL., aged 55. Early appearances of posterior polar cataract in both lenses; left began at 50, and right at 51 (?); in each lens is a central well-defined dot, a central congenital cataract, which shows no tendency to increase, and by looking well from the side with light reflected from the mirror, clear lens tissue can be readily seen between the central and posterior cortical cataracts in each eye. He is about to give up work owing to increasing blindness. His fundi are normal and "tiger-striped." He has astigmatism.

Cases I, II, and IV were shown at the meeting of the North-umbrian and Durham Medical Society on November 13th last. I have gone into this matter in some detail because, so far as I know, there is no literature at all on the subject, except a short paragraph in Berry's *Diseases of the Eye* (page 177 of second edition), in which it is stated that glass-blowers are subject to cataract, which generally affects the left eye first or altogether, owing to the left eye being nearer the glowing glass.

In this investigation I have been much helped by our house-surgeon, Dr. C. A. S. Dalglish, especially in the observation of cases.

PERFORATION OF THE OESOPHAGUS BY TUBERCULOUS GLANDS.

By CLIVE RIVIERE, M.D. LOND., M.R.C.P.,

Casualty Physician, St. Bartholomew's Hospital; Assistant Physician, East London Children's Hospital, Shadwell.

INSTANCES of tuberculous glands opening into the oesophagus are not often met with, and the following three examples seem, therefore, worthy of record. They occurred at the East London Children's Hospital, Shadwell, and I am indebted to Dr. Eustace Smith and Dr. Coutts for permission to publish the notes. The necropsy in each case was performed by myself.

CASE I.—W. C., a male, aged 2 years, was admitted on November 26th, 1900, with a history of three weeks' wasting and loss of appetite. There had been no diarrhoea or vomiting but cough had been troublesome. There was no family history of tuberculosis.

Condition on Admission.—The child was delicate-looking and showed well-marked rickets. The temperature was 102°; there were scattered rhonchi in the lungs; the liver was found to be enlarged and the spleen reached to the umbilicus.

After-History.—On December 7th it was noted that the abdomen was much distended and the temperature still remained high. On February 1st dullness over the upper sternum and on either side of it was recorded. The temperature kept high throughout the three months' illness, with fluctuations of 2° or 3°. Diarrhoea, which had begun on December 21st, continued throughout with three or four stools daily, and there was occasional vomiting, seven times in all. The child died on February 17th, 1901.

Necropsy.—At the necropsy a mass of caseous glands the size of a tangerine orange was found behind the manubrium sterni. The trachea lay behind it, and an offshoot of the mass surrounded and narrowed the left bronchus. The oesophagus was deflected to the left, and in its front wall, just below the bifurcation of the trachea, was a large opening where a caseous gland had become adherent and had ulcerated through. This was the bifurcation gland, and the opening led into a cavity in its interior containing foul pus and debris. In the stomach were three small ulcers in the mucous coat formed by the softening of caseous tubercles. The intestine contained only two small tubercles in the mucous membrane of the ileum. Some of the mesenteric glands were caseous, and the retroperitoneal glands formed a large caseous mass. There were besides tubercles in most of the other organs of the body.

CASE II.—L. K., a male, aged 10 months, was admitted on April 1st, 1902, with a history of three months' cough and wasting. There had been much vomiting, but the bowels had been regular. The parents were healthy and there was no tuberculous family history.

Condition on Admission.—A pale, wasted child, with yellow-tinted skin. In the lungs were found dullness and feeble breath sounds over the whole left side, and the heart was best heard to the right of the sternum. The abdomen was somewhat distended; the spleen was palpable and firm.

Operation: After-history.—On April 8th an incision was made into the left pleural cavity and 8 oz. of pus evacuated. Thereafter the child steadily wasted, vomiting occasionally (seven times during the two weeks' illness), but having no diarrhoea, and died on April 24th.

Necropsy.—At the necropsy a mass of caseous right tracheal glands, caseous bronchial and pulmonary glands and a limited lung tuberculosis were found. In the oesophagus, below the bifurcation of the trachea, opposite the bifurcation gland, was an opening through the anterior wall large enough to admit the little finger, and leading into a cavity the size of the end of a thumb. This was formed by softening of the bifurcation gland and was lined by a mucous coat over a fibrous wall. It extended upwards in front of the right bronchus to some caseous right tracheal glands. No caseous contents were left. There were no tubercles or ulcers in the stomach, but the intestine was lined with tuberculous ulcers from end to end and the mesenteric glands were caseous.

CASE III.—G., a male, aged 1 year 11 months, was admitted on April 18th, 1902. There was a history of five months' diarrhoea and vomiting, accompanied by wasting. He had had a discharge from the left ear for the past four weeks, and a left-sided fit three weeks before admission. There was no tuberculous family history.

Condition on Admission.—A pale, wasted child, with a profuse purulent discharge from the left ear, and a complete left facial paralysis. In the lungs a few crepitations were heard at the bases. The abdomen was somewhat distended, and the liver and spleen slightly enlarged.

After-history.—The child had a high, fluctuating temperature and much diarrhoea, the bowels acting three to seven times daily. He progressively wasted, and died on April 24th.

Necropsy.—At the necropsy performed next day was found an enormous mass of caseous glands surrounding the trachea. They were largest on the right side. There were racemose tubercles and "caseous blocks" in the lungs, and scattered tubercles in the abdominal organs. The bifurcation gland had completely emptied its contents into the oesophagus, leaving a smooth-walled sac opening through the anterior oesophageal wall by an aperture $\frac{1}{2}$ in. in diameter. There were no tubercles in the stomach. The intestine contained small ulcers and tuberculous nodules throughout its extent. The mesenteric glands formed a caseous mass the size of a turkey's egg, one individual gland being as large as a pigeon's egg. The retroperitoneal glands were caseous, but not very large.

In these 3 cases it was the gland (or glands) situated below the bifurcation of the trachea which caused the trouble, and this seems to be generally the case. I can find but few instances recorded in the literature. In 1890 Dr. Voelcker exhibited a specimen to the Pathological Society from a boy aged 9 years, in whom the bifurcation gland had opened into the oesophagus, and discharged part of its contents, leaving some caseous material still present in the cavity. There was no history of oesophageal trouble, nor is the presence of any tubercle elsewhere in the body recorded. The boy died of quite other disease.

The absence of abdominal tuberculosis in such a case, if the omission of its mention really means its absence, seems to me very extraordinary. In the 3 cases described above it will be noticed that the amount of abdominal tuberculosis is, all through, as we should expect, a striking feature. In one there were tuberculous ulcers in the stomach, a very rare condition; in all 3 there was intestinal tuberculous ulceration and caseous mesenteric glands, and in 2 the retroperitoneal glands were also caseous, a condition which in my experience is by no means common. Dr. Voelcker finds only 3 other cases recorded among 2,504 necropsies made during thirty years at Great Ormond Street Hospital, and in 2 of these 3 the air passages were also involved. This we should expect to occur in many cases, the gland becoming adherent to the oesophagus on one side, and to a bronchus (generally the right bronchus) on the other side, ulcerating into both, and thus forming a passage between the two which finally may become lined by mucous membrane.

In two of the cases mentioned above the gland had completely emptied its caseous contents, its cavity was smooth-walled and appeared to possess a mucous lining.

In March, 1901, Dr. Norman Moore showed at the Pathological Society a case of an oesophageal diverticulum the size of a pea occurring in the anterior wall just below the end of the trachea. The patient was a man, aged 49, who died of bronchitis, and the diverticulum was regarded by Dr. Norman Moore as a congenital abnormality. May not such a diverticulum have originated in the same manner as in the cases described above? The position is at the seat of the bifurcation gland in the anterior wall, and, moreover, the absence of any mention of tubercle in the body does not militate against this suggestion.

In children the bifurcation gland is often, or even generally, the first visible seat of tubercle in the body, and one may suppose that in some cases the disease goes no further. I am inclined to think that many cases of oesophageal diverticulum occurring in this situation are due to this cause. This would not include those instances of "pulsion diverticulum" which have caused so much surgical interest of late years.

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.

By H. J. PECHELL, M.B., C.M.,

Late House-Physician and Anaesthetist, Derbyshire Infirmary.

(From the Pathological Laboratory of the Westminster Hospital.)

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.

ATHELSTAN SAW, M.D. Cantab.,

Honorary Surgeon, Perth Public Hospital, West Australia.

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-