DRUG ERUPTIONS.

These were chiefly discussed as regards bromide and iodide eruptions, which are the only ones which are distinctive, other drug eruptions not being recognized as such unless it was known that one of the possibly eruption-exciting drugs had been given. Bromide and iodide agminated eruptions could always be recognized, and it was pointed out that while in infants they might occur from very small doses even through suckling when the mother was taking the drug, in adults if an eruption occurred from a moderate dose, it was an indication of defective elimination due to either heart or kidney disease or both. It was also pointed out that both bromide and iodide eruptions often attacked scar tissue and the fact that their diagnosis was missed sometimes because they often did not come out one or two weeks or more after the drugs had been stopped. This was explained by their having a diuretic action which ceased soon after the drug was stopped, and then the salt which was left in the tissues produced the eruption.

THE INFLUENCE OF GOUT.

The lecturer said he had left this to the last because there was no need to emphasize a factor which in the past had been overrated both by the doctor and the public. But while he did not doubt that gout was a strongly predisposing cause of inflammation of the skin, it was not possible to diagnose gout by the skin lesion alone; moreover, experience often showed that frequently when we had done our best to neutralize the supposed uric acid element by alkalies and other antigouty remedies, the eruption was still too often recalcitrant, and he believed that in a large proportion of cases it was again another example of toxin absorption from the intestine. The tendency of gouty persons to chronic intestinal catarrh, and the readiness with which intestinal fermentation was set up being the real factors requiring attention, intestinal disinfectants of the salol class, and a dietary which would not be likely to disagree and produce further intestinal fermentation were the real indications.

CASE OF APPARENT SECONDARY ABDOMINAL PREGNANCY IN A RABBIT AFTER PRIMARY RUPTURE OF THE UTERUS.

By KURT KAMANN, M.D.,
Greifswald, Germany.

At the necropsy on a rabbit, which had been used for experimental purposes, a complete fetal sac was found in the abdominal cavity containing a full-time fetus which had only recently died. The sac was attached to a very vascular portion of the great omentum which had been drawn out in the form of a pedicle.

The placenta was situated on the wall of the sac opposite its point of attachment to the omentum, which was the only means of connexion between the fetal sac and the mother. The internal genital organs lay at some distance from the sac, and appeared to me to be free of any marked changes. At first I considered it to be a case of primary abdominal pregnancy.

In the course of further investigation, however, this hypothesis appeared to me to be untenable. On examining the uterus by means of sections (the examination being carried out in the laboratory of Professor Martin) I found that the left uterine horn, which macroscopically appeared quite smooth, on microscopical examination presented a tear in its wall, which was filled with fresh granulation tissue.

The discovery of a recent cicatrix in the uterine wall proved conclusively that the fetus had not developed primarily in the abdominal cavity, but in the uterus, and that the full-time fetal sac, together with the placenta, had passed out of the uterus through the tear in its wall into the abdominal cavity.

I believe that its secondary attachment to the omentum did not occur suddenly after a complete expulsion through an extensive uterine rupture, but that it took place during the course of a gradual protracted rupture of the uterus (*protrahierten Uterusruptur*), so that the fetal sac, becoming adherent to the omentum, received its blood supply from it before being completely expelled from the uterus.

Although one must be careful in drawing comparisons between corresponding conditions in men and animals, yet I think I shall not be wrong in comparing the uterus of the rabbit with the Fallopian tube of the human female in this case. With this proviso, I believe the events which occur in most of those cases of so-called abdominal pregnancy in the

human female may be described as a gradual rupture of the Fallopian tube and the adhesion of the fetal sac, which is slowly expelled, to the neighbouring abdominal organs.

The microscopical sections also showed an interesting condition. On the internal surface of the uterine cicatrix I found a peculiar body, which was in all probability a fertilized ovum that had failed to develop. If this were in reality an ovum, it would be justifiable to suppose that the expulsion of the fetal sac into the abdominal cavity had brought the pregnancy to an end, and that the rut in the animal had returned. This supposition was supported by the presence of comparatively recent corpora lutea in both ovaries and a condition of evolution of the uterine mucous membrane.

A complete report, extensively illustrated, of this case will appear shortly in the *Monatschrift für Geburtshilfe und Gynaecologie*. The above presents in an abbreviated form the chief points of interest in this case.

CASE OF RHEUMATIC FEVER COMPLICATED BY CHOREA, IRITIS, AND ENDOCARDITIS: RECOVERY.

By FRED. C. FORSTER, M.R.C.S.ENG., L.R.C.P.LOND.,
Lowestoft.

A BRIGHT young girl, aged 12½, was first seen by me in the summer of 1901 complaining of sore throat and slight aching pains in the right knee and along the right tibia. The knee was not swollen, nor was there any sign of inflammation in the joint. The tonsils were inflamed, the fauces congested, and the tongue furred. The pulse was 90 and the temperature 99° F.

I was told that three days previously she had been caught in a heavy shower coming home from school. Suspecting the tonsillitis to be rheumatic in nature, I at once advised the child being put to bed, to be kept on a strictly milk diet, ordering a saline purge and sodium salicylate mixture in gr.x doses four-hourly.

In spite of thus trying to anticipate the disease, I found next morning the right knee joint much swollen and inflamed and also the right elbow and left ankle, whilst the temperature had risen to 101° F., and the pulse-rate to 120. For the next fortnight the case was a fairly typical one of rheumatic fever, the temperature rising on two occasions to 104° F., and both shoulder-joints becoming affected.

At the end of the fortnight the temperature had fallen to normal, and the inflammation in the attacked joints had quite subsided. The cardiac condition at this time was satisfactory, the pulse was 80, soft and regular and at no time in the illness had there been any evidence of commencing dilatation.

Three days later, whilst looking at the child's tongue, I noticed that it was tremulous: on being asked to hold out her hands above the bed-clothes and to extend her fingers, slight, purposeless, jerky movements were noted—so suggestive of chorea. The child was still on a careful diet and was taking 5-gr. doses of sodium salicylate with mxx of aromatic spirits of ammonia thrice daily.

During the next four weeks the choreic movements became more developed and continued extremely typical, involving the muscles of the face and upper extremities, but not the trunk nor lower limbs.

Whilst the movements were slowly decreasing and the child making a steady convalescence she one day complained of pain "like a needle" in her right eye and inability to see clearly. This was the starting point of an attack of acute iritis which was presumably "rheumatic" in origin, and clinically resembled that variety in the severity of the congestion, pain, and photophobia.

For some time the patient, who had borne up with wonderful cheerfulness, was very ill, and my anxiety was increased by the insidious onset of a cardiac lesion. This was first noted shortly after the onset of the iritis, in the shape of a blurring of the first sound at the apex, with an increased and irregular pulse. Later there developed a loud grating systolic *bruit* audible all over the left axilla and back, whilst the heart's impulse could be felt 1 in. outside the vertical nipple line. Cyanosis and oedema accompanied the dilatation, and for several days the issue was doubtful, but I am pleased to report that she successfully pulled through, owing not a little to her excellent nursing and her own splendid pluck.

After spending the winter in the South of England she went abroad with her mother in the spring, and I have recently heard that she has "had no return of her rheumatism and

can take long walks and cycle rides without getting blown," which speaks well for her cardiac condition. I have omitted the details of her treatment as the chief interest of the case, and its claim to be recorded lies in the unusual severity and persistence of the rheumatic invasion as shown by the sequelae. The associations between rheumatic fever, chorea, iritis, and endocarditis are well recognized, but it is rare for these complications to occur consecutively in one patient. I may add that the child had never previously suffered from rheumatism in any of its manifestations, and the only point of note in her family history was that a maternal aunt had had chorea as a child.

BRIGHT'S DISEASE: ANURIA: INTESTINAL HAEMORRHAGE: DEATH.

By Lyster Cole-Baker, M.D.,
Assistant Physician, Royal Portsmouth Hospital.

THE following case is of interest from its suddenness, the difficulty of diagnosis, and the confusing symptoms.

Miss B., aged 43, unmarried, had previously enjoyed good health, except that she occasionally, perhaps, had felt more tired than could be accounted for by the amount of exercise taken, and except for an attack of meningitis six years previously. On November 1st, 1901, she went to see H.M.S. *Ophir* come in, standing in an exposed position on the top of a building in the dockyard for some three hours. Next day she went for a bicycle ride of about twelve miles, and after a rather heavy meal went to bed at 11 p.m. At 12.30 a.m. she woke up in great pain in the abdomen, and vomited two or three times. I was sent for, and saw her at 4 a.m. She then complained of great pain in the epigastrium, just beneath the xiphoid cartilage, and on palpation there was distinct tenderness. Her pulse was normal, and temperature slightly subnormal—98°. In a basin beside the bed was some mucus tinged with bile which she had brought up. I injected morph. hydrochlor. gr. $\frac{1}{2}$ hypodermically, and, as her condition improved, left her. During November 3rd she was still in pain, though not so severe, located in the same place; the vomiting had ceased. She had a feeling of nausea, so was kept on water and ice all day. The pulse and temperature were as on the previous night. Calomel gr. v was given twice at intervals of six hours.

On November 4th I saw her at 10 a.m., when the pain had shifted, and was referred to the hypogastric region between the pubes and umbilicus. I was told she had not passed urine for twenty-four hours, and on palpation a distinct tumour could be felt, pressure causing great tenderness and pain. On percussion this tumour was dull, with very pronounced resonance above and in both flanks, presenting in a most remarkable manner all the symptoms and signs of a full bladder. On passing a catheter, however, no urine was obtained, although four different ones were tried. Per vaginam the bladder was felt to be empty, the uterus and tubes and ovaries normal. Therefore one had to come to the conclusion that the kidneys had ceased to secrete. A hot bath was ordered. I saw her almost immediately after it, and then her condition had changed very seriously. She was very cold; the lips, nose, and finger tips were blue or livid. Her breathing was very rapid, 46; her pulse could not be felt at the wrist. In fact she was moribund, and died an hour afterwards. Ether and strychnine had been injected hypodermically, champagne administered by the mouth, and she had been entirely enveloped in blankets and surrounded with hot bottles.

Necropsy.—On opening the abdomen in the hypogastric region there was a coil of intestine, livid or plum-coloured, 4 ft. in length. This was found filled with dark-coloured blood, 2 pints in quantity. The mesentery for the whole length of this part of the intestine was of the same colour as the intestine, swollen to a thickness of 1 to 1½ in. On cutting into the mesentery, it was found to have all its meshes filled with blood clot; there was about 1½ pint of bloody fluid in the peritoneal cavity. The kidneys were enlarged and heavy, presenting the typical characteristics of large white kidney. All the rest of the abdominal organs were normal and healthy, and all the rest of the intestine was of a pale pink colour. There was no strangulation or twisting of the intestine, and no point could be found from which the bleeding had occurred.

Among other points of interest are the following:

1. Death occurred forty hours after the onset of pain.
2. The pain in the epigastric region was the only prominent symptom for about thirty hours. Pain in this region is so very common, no matter where the lesion, that its occurrence is of little help in diagnosis. I have myself noticed that pain has been located there on the onset of perforating gastric ulcer, perforation of the appendix, simple appendicitis, and hepatitis; in fact, so common is it that one is tempted to neglect it entirely in trying to arrive at a diagnosis.
3. The patient had passed water on November 3rd. On November 4th she complained that she had not passed any for twenty-four hours and was suffering from pain in the region of the bladder. The coil of intestine filled with blood lying in the hypogastric region gave all the subjective and objective symptoms of a full bladder; indeed, I venture to say that, had such a case occurred in a male subject into

whose bladder there was difficulty in passing a catheter from stricture or false passage, one would have been sorely tempted to try suprapubic aspiration.

My interpretation of this case is that the severe cold on November 1st, combined with the bicycle ride on November 2nd, caused the kidneys to cease secreting, or almost so, and that the increased blood pressure in consequence caused rupture of a small intramesenteric vessel, which blood slowly oozed and clotted as it flowed, till the bath on November 4th, when the collapse set in from excessive haemorrhage. The urine voided on November 3rd was, I think, secreted some time on November 2nd, previous to the rupture of the vessel.

4. It was impossible to diagnose the anuria till six hours before death, and when diagnosed there was no urgency or gravity in the case.

5. Remedial measures for the anuria only aggravated the bleeding and precipitated the end.

DIABETIC COMA TREATED BY TRANSFUSION.

By David Young, M.B. Edin.,
Late House-Physician, Edinburgh Royal Infirmary.

SINCE Studelmann recommended the treatment of diabetic coma by alkalies administered intravenously, several recoveries have been recorded. In the case to which I refer the temporary improvement after transfusion was very marked, and it appeared as if the patient would be tided over his attack of coma, till pericarditis supervened, and apparently closed the scene.

History.

J. McW., aged 39, was admitted to the Edinburgh Royal Infirmary on June 11th, 1902, suffering from diabetes of nearly three years' duration. He had been treated in Glasgow Royal Infirmary in 1899, and again in 1901. On each occasion he improved, but relapsed after leaving the institution. Three weeks before admission to the infirmary he restarted work as an ironmonger, and this appeared to lead to his symptoms becoming aggravated.

Condition on Admission.

On admission, patient was put on ordinary diet, and no drug administered, in order that his condition might be observed before active treatment was commenced. The day after admission he passed 136 fluid ounces of urine containing 310 grams of sugar. No diacetic acid was present.

Treatment.

On the morning of June 13th he complained of pain in the abdomen relieved by fomentations, and headache. He was sick after dinner, and at 7 p.m. complained of lightness in the head. An enema and purgative were ordered, also sod. bicarb. 5j every three hours, and diuretin gr. xv. four times daily. At 9 p.m. he was becoming drowsy, and had vomited twice. Breathing was noisy, and the breath had a sweetish odour. The urine contained diacetic acid and had fallen for the day to 31xxx, and the sugar to 200 grams. The case at this time appeared to be one of diabetic coma, progressing rapidly.

On June 14th, at 1 a.m., he was very drowsy, and could be roused only with difficulty. The pulse was 130 and the respirations 26. I then injected 3xxx normal saline solution intravenously after withdrawing 3viii blood from the same vein. Two hours later he had revived to a remarkable extent but said his head felt light. He was able to answer questions intelligently but did not remember anything about the transfusion: the pulse 102 and the respirations 22. At 11 a.m. he felt better and had taken a fair breakfast. During the day he was seen by Dr. Byrom Bramwell, in whose ward he was, and to whose kindness I am indebted for permission to publish these notes. Dr. Bramwell confirmed the diagnosis and approved of the treatment. At 4 p.m. he was again tending to become drowsy. He bled to 3v, and 3xxx saline solution again injected. At 10 p.m. he was quite conscious.

On June 15th, in the morning, he looked better than on the previous day. The pulse was 93 and the respirations 20. At 5 p.m. the pulse was feeble and he was again transfused with 3xx of sterile fluid made to the following formula (from *Tirard's Medical Treatment*): Sodium chloride, 93 gr.; liquor sodii 20 drops; water, 2 pints. An hour later his pulse indicated the beneficial result. Potassium citrate was ordered instead of the sodium bicarbonate, as the latter appeared to be causing flatulence.

He again received 3xx of the above solution intravenously on June 16th. There was no diacetic acid in the urine.

On June 17th he was much better, asked to be allowed to get up, and was able to write a letter in the afternoon. He was not drowsy, breathing was quiet, and the breath had no peculiar odour. On the following afternoon he was drowsy and sick twice. He was transfused, 3xxx being given.

On June 19th he was sick three times, and was again transfused 3xxx. He had headache on the following morning, but was not sick. A hot vapour bath was given, but he did not sweat till he had been in one hour. He was then left in ten minutes. Later he was transfused, receiving 3xxx.

On June 21st his condition had improved, and he looked brighter. He said he had not felt better since admission to hospital, and asked for