emboli appear to have lodged; and in two such instances I find in my sections that these same emboli have penetrated the endothelium, and dissected the muscular coats of the vessels.

I am wholly unable to appreciate any effects which might be attributed to the treatments to which you say the growth has been submitted, except in the more superficial alveoli of the portion of the skin from old scar, where I recognize the effect of the x-ray treatment in a contraction of the alveoli, together with a general atrophic condition, affecting all superficial structures save epidermis, which appears to be entirely normal. The groups of cancer cells in this locality appear to have become markedly shrunken; they fail to react properly to the nuclear and protoplasmic stains, and there is consequently very little differentiation. It exhibits practically no karyokinesis.

The foregoing history, embracing the pathological reports, will, I believe, convince all who may be interested that in the study of cancer, more perhaps than in any other field of research to-day, is there need for "suspended judgement," for "the cultivation of that enthusiasm for truth, that fanaticism of veracity," which should be brought to bear on all questions involving human life.

Whether we accept or reject the theory upon which it is based, let the "trypsin treatment" receive the scientific test; while it is being tested, let there be suspended judgement. When the evidence is correlated and the final verdict rendered, if favourable, let it be accepted; if adverse—then on to the next.

INFLUENZA COMMENCING WITH SUDDEN UNCONSCIOUSNESS, AND EXCESSIVE DYSPNOEA.

By WILFRED HARRIS, M.D., F.R.C.P.,

PHYSICIAN TO OUT-PATIENTS, ST. MARY'S HOSPITAL, AND TO THE HOSPITAL FOR EPILEPSY AND PARALYSIS, MAIDA VALE.

THE following account of an unusual case will, I think, be both interesting and instructive from the point of view of diagnosis, and on account of the recent renewal of severity of the scourge of influenza.

A. G., a plasterer, aged 38, was admitted into St. Mary's Hospital in the morning of December 6th in a semi-conscious condition. From the account given by his mates who brought him, and from what he was afterwards able to remember himself, it appears that he was in his usual health when he went to work that morning, and while he was at work he commenced to feel giddy. This lasted for perhaps ten minutes, when he fell down suddenly unconscious. He was slightly convulsed at first, and his fellow-workmen left him on the ground, thinking that he would soon come round. He recovered sufficiently in about twenty minutes to be able to speak, and he was then brought up in a cab to the hospital. He says now that at the time he felt able to walk up, but judging from his condition on admission, that is more than doubtful.

State on Admission.—After being put to bed, he was found to be lying in a semi-conscious condition, just able to answer questions, though very slowly. His temperature was 101° F., the pulse 88, and his respirations 76 per minute and slightly stertorous. The pupils were contracted and equal, and reacted to light. There were no signs of any weakness of the limbs, but his legs seemed somewhat rigid, and the knee-jerks were brisk; the plantar reflexes were flexor, the urine normal, tongue coated, and bowels constipated. He complained of pain in his head, and there was also occasional coughing, which increased the headache, as when he coughed he put his hand across his forehead. On the evening of admission the temperature ran up to 102.4° F., though the pulse fell to 72 and the respirations to 24 per minute. At 2 o'clock the next afternoon, when I saw him for the first time, the temperature had fallen to 99.6° F., but the respirations were again very frequent—60 per minute, while the pulse-rate was only 56, strong, and of full volume. He was now fully conscious, but was still slow in answering questions. He complained of severe frontal headache and of pain in his back in the lumbar region, and he was able to tell us that he had had two similar attacks, one four years and the other fifteen years previously. Examination of the chest showed him to be perspiring freely; both sides of the chest moved well, but on percussion the note over the whole of the right front was distinctly high-pitched, and there were numerous sticky râles to be heard over the right upper lobe. The arteries were somewhat thick-walled and hard, but the urine was of good specific gravity, and contained no albumen. The optic discs and retinae were normal, and examination of the nervous system showed no abnormal physical signs.

The several diagnoses that were at once suggested as possibilities were—epilepsy, cerebral haemorrhage or thrombosis, cerebral tumour, uraemia, and pneumonia. Epilepsy seemed possible on account of the sudden onset

of unconsciousness, followed by slight convulsion, and this diagnosis was rendered more probable by the history of two similar attacks previously. However, the dyspnoea, the râles in the chest, and the pyrexia excluded this diagnosis. Apoplexy and cerebral tumour, too, were similarly excluded. Uraemia at first seemed possible, because of the definite arterio-sclerosis present, and the dyspnoea might have been the air-hunger of uraemia, while pyrexia is not uncommon with uraemic convulsions. The breath did not suggest uraemia, though its odour was heavy, and the high specific gravity of the urine, with absence of albumen, excluded this diagnosis too. Pneumonia seemed, perhaps, the most likely of all the above-mentioned possibilities, accounting for the pyrexia, dyspnoea, high-pitched percussion note, and the râles. The dyspnoea was, however, excessive for pneumonia, and the pulse-rate disproportionately slow. Although it is well known that in pneumonia the pulse-respiration ratio is altered from the normal 1.4½ to 1:2 or even less, I have never known such a disproportionate ratio as was present in this case of 88:76 on admission, and when I first saw him on the next day the pulse-rate was even slower than the respirations, being 56:60. Such an excessive frequency of the respirations is met with only in asthma, hysterical dyspnoea, or in air-hunger of uraemia, or acid intoxications, as in diabetes or salicylate poisoning. Moreover, although lobar pneumonia may be ushered in with a convulsion in a young child, especially one with a family neuropathic tendency, I do not know of such a commencement of the disease in an adult.

Neither of these possible diagnoses, therefore, seemed to fit the case, and one sign seemed to me particularly suggestive of influenza—namely, the slow pulse, only 56 when I first saw him, though the temperature was then 99.6° F. Looking at the case from this point of view, the other symptoms—the frontal headache and lumbar pain, pyrexia, sticky râles, and cough—all clearly pointed to this disease. Only the curious mode of onset with sudden unconsciousness remained to be explained, but, being familiar with the literature of the disease, particularly of the epidemic of the early nineties, I was aware of several cases having been recorded which started suddenly in this way. The after-course of my case proved this diagnosis to be correct. He was put on an effervescing quinine mixture every four hours; the frontal headache and the backache improved much on the third day, and his temperature, which reached its highest point of 102.8° F. on the third night, fell to normal during the early hours of the fifth day, and he rapidly became convalescent. The high-pitched percussion note and the râles over the right front of the chest disappeared on the third day, and no further signs of pneumonia developed, but that he had an abortive form of pneumonia, of influenzal origin, I have no doubt. We learnt later that the two previous seizures with sudden unconsciousness that he had had, four years and fifteen years ago, ushered in precisely similar attacks of influenza to this illness, being followed on each occasion by fever, severe headache, pains, and bronchitis which was afterwards recognized to be due to influenza. It will be noted that his first attack occurred in 1891, in the comparatively early days of the last invasion of this country with influenza.

TWO CASES OF CYSTINE CALCULI.

By F. A. SOUTHAM, M.B.Oxon., F.R.C.S.Eng.,

SURGEON TO THE MANCHESTER ROYAL INFIRMARY; PROFESSOR OF CLINICAL SURGERY, UNIVERSITY OF MANCHESTER.

CYSTINE calculi are of such rare occurrence that a brief account of two cases—one vesical, the other renal—which were recently under my care at the same time in the Manchester Royal Infirmary, may prove of interest.

In a series of 140 cases of vesical calculi which I have removed by operation, Case 1, recorded below, is the only instance met with where the calculus has consisted of cystine.

In the BRITISH MEDICAL JOURNAL of July 13th, 1878, I recorded details of 4 cases of vesical calculi consisting of cystine, which were operated upon by my father (who was surgeon to the Manchester Royal Infirmary, and Professor of Surgery at Owens College). Of these, 2 occurred in private and 2 in hospital practice.

The last case of the series, which is fully recorded in

the *BRITISH MEDICAL JOURNAL* of December 23rd, 1876, was operated upon in the Manchester Royal Infirmary in 1874, and since that date—as far as I have been able to ascertain—only 2 cases of cystine calculi have been met with in the hospital, one under the care of Mr. Walter Whitehead about twenty years ago, and the other under the late Professor Jones in 1892.

† An interesting point in connexion with the latter case, as illustrating the hereditary character of cystine, is the fact that the patient, an unmarried female, aged 20 years, was the daughter of one of the four patients (also a female) operated upon by my father 24 years previously. She was admitted suffering from renal symptoms, and nephrolithotomy was performed by Professor Jones, a small cystine calculus being successfully removed from the left kidney.

I have recently had an opportunity of examining a specimen of the urine of this patient, and find that at the present time (14 years after the operation) it still contains crystals of cystine in small quantity.

✱ It is interesting to note, as illustrating the persistence of cystinuria, that an examination of the urine of her mother, eight years after the operation for the removal of the calculus from her bladder, also showed that cystine was still present. This patient had no further urinary trouble, and recently died of paralysis (34 years after the operation), at the age of 67.

† The following are the details of the two cases recently under my care :

CASE I.

Cystine Calculus Removed by Suprapubic Lithotomy.

W. S., aged 32, was admitted October 17th, 1906, with the following history : Seven years previously he was suddenly seized with severe pain in the left lumbar region, and shortly afterwards he passed, per urethram, a small calculus, which was not preserved. Fifteen months ago he suffered from a

Fig. 1.— $\times 2$ lin.

similar attack of renal colic. He was then free from any symptoms until about two months previous to admission, when he began to be troubled with pain and increased frequency in micturition, and occasional sudden stoppage in the flow of urine. Blood had never been noticed in the urine, which was clear and acid, containing a trace of albumen. On sounding the bladder a calculus of large size was readily felt. Suprapubic lithotomy was successfully performed, and the patient made a good recovery from the operation. The calculus, which is mushroom-shaped, measuring $1\frac{1}{2}$ in. by $1\frac{1}{4}$ in., consists of cystine, and is of a pale yellow colour, with an irregular crystalline surface (Fig. 1). Examination of the urine subsequent to the operation showed that crystals of cystine were present on some days, while on other days none could be detected. No history of calculi in any other member of his family could be obtained.

CASE II.

Cystine Calculus Passed per Urethram.

C. W., 22, single, was admitted October 19th, 1906, with the following history : For about six months she had suffered from frequent attacks of pain, which commenced in the region of the left kidney, and extended across the abdomen and downwards into the thigh. About a month before admission, after a more severe attack than usual, followed by blood in the urine and increased frequency in micturition, she passed a small calculus. After this the attacks of pain became less frequent and less severe, and when admitted into hospital they had almost ceased, though the region of the left kidney was slightly tender upon pressure, and more resistant than on the opposite side. A radiograph gave a negative result. The urine, 1020, acid, was of a pale amber colour, and, examined microscopically, was found to contain the characteristic crystals of cystine, mixed with a few crystals of oxalate of lime. On some days the cystine crystals were present in such quantities that they could be seen with the naked eye, forming a deposit upon the cloud of mucus, which settled on standing, and glistening brightly if the specimen glass was held and rotated in the sunshine or electric light.

The calculus passed by the patient was pear-shaped, and of a pale yellow colour, with a glistening crystalline surface; it measured one-third of an inch in length (Fig. 2). The patient remained in hospital for three weeks, and as she was free from any attacks of renal colic she was then discharged, with instructions to return and report herself if there was any recurrence of the symptoms. On inquiring into her family history she was not aware that any of her relatives had ever suffered from symptoms of calculus.

Fig. 2.— $\times 2$ lin.

I am indebted to my late house-surgeon, Mr. J. Stirling Young, for the accompanying drawings.

The Milroy Lectures

ON

KALA-AZAR,

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS OF LONDON.

BY LEONARD ROGERS, M.D., F.R.C.P., I.M.S.,
PROFESSOR OF PATHOLOGY, CALCUTTA.

LECTURE II.

KALA-AZAR AS A DISEASE.

[ABSTRACT.]

IN my first lecture I dealt with kala-azar as an epidemic, tracing its origin, spread, and decline, and referred to a similar earlier outbreak in Bengal known as Burdwan fever. To-day I pass on to describe the disease itself—its course, complications, and terminations—and shall show you that it is not one whit less terrible when considered individually than collectively, for it literally kills by inches after most prolonged sufferings, which we are still powerless to check to any great extent. As the later stages of the disease are much better known than the earlier ones, it will be clearer if I first describe the typical advanced condition, returning to the still rarely recognized commencement of the fever.

THE GENERAL APPEARANCES IN THE CHARACTERISTIC STAGES OF THE DISEASE KALA-AZAR.

A number of lantern slides showing groups of cases were shown, and the salient points of each pointed out, the main features being the marked contrast between the tumid bellies due to the very great enlargement of the spleen, and sometimes also of the liver, contrasting with the great wasting of the chest, face, and limbs. Brief notes were given of a group of hospital cases which illustrated the different courses the disease might take, while a village group showed the great frequency of the affection in children, just as in malarial fever, the incidence decreasing steadily in the subsequent decades. Other groups of sporadic cases, then known as "malarial cachexia" in the Sylhet Valley, which was not invaded by the epidemic, showed the clinical identity of the two forms of the disease, which was the principal argument of the speaker in his 1897 report in favour of his view that the epidemic disease in the Brahmaputra Valley was but an intensified form of malarial fever. The extreme wasting in the last stages and the dropsical ascitic form