and constant of these is that it becomes deposited in an insoluble form on standing for a longer or shorter time. This insoluble dysalbumose, as it has been called, is generally to be found in a specimen that has stood for some weeks.

I have by me a bottle of urine which I filtered quite clear twelve months ago, and which now contains a dense deposit. This deposit may take place very rapidly; in my first case it sometimes took place in the patient's bladder, and consequently the urine looked like milk when voided. This change is apparently of the same nature as the coagulation by heat.

Other Tests.

Before concluding I should like to allude to certain tests for myelopathic albumose which have been mentioned by various writers, but which have proved in my hands absolutely worthless. Of these the best known is the so-called biuret test, performed by treating the solution with caustic potash or soda and a trace of copper sulphate. The result is supposed to be that in the case of albumin a violet colour is produced; in the case of albumoses and peptones a rose red. Professor Huppert of Prague, however, states somewhat emphatically that the biuret reaction given by peptone is in no way distinguishable from that of albumin.¹ If this be true with regard to peptone, we may suspect that it will *a fortiori* be also true of albumoses, which chemically stand much closer to albumin. My own experience with the biuret test convinces me that it is of no use whatever as a means of distinguishing the different proteids as they occur in the urine. A rose pink is always obtained if the amount of copper is small, no matter what is the kind of proteid under examination. I have therefore no hesitation in saying that in practice the biuret reaction is absolutely worthless in the examination of urine.

Another test has been brought out by Boston of Philadelphia, and is described at length in his book on clinical diagnosis.² It depends on the fact that albumose contains loosely combined sulphur, which in the presence of alkali unites with lead salts to form black sulphide. Boston says that many chemists regard this reaction as one of the most characteristic features of the body we are considering, and that it distinguishes it from the digestive albumoses. I may remark in passing that in practice we are hardly likely to meet with difficulty in distinguishing myelopathic from digestive albumose; the real problem is to distinguish it from albumin. However, I have applied Boston's test to urine containing albumin, digestive albumose (Witte's peptone), and myelopathic albumose respectively, and I find that the results obtained in all three cases are quite indistinguishable.

Again, Dixon Mann states,³ on the authority of Grutterink and Graaf, that with an equal volume of 50 per cent. acetic acid the albumose will become solid, so that the test-tube can be inverted without spilling. This in my experience is certainly not the case. However, I maintain that the simple tests I have already described are sufficient to enable us to identify this body, and that from the point of view of the practising physician there is no practical advantage in adding to their number.

There are many points with regard to the nature of this interesting body which I have not touched upon in the foregoing remarks. If I were to deal with them I should have to enter into various chemical problems which are outside the scope of the present communication, the purpose of which is to place clearly before my readers the means by which the presence of myelopathic albumose in the urine may be determined.

REFERENCES.

¹ Neubauer and Vogel, *Analyse des Harns*; 3te Aufl., p. 478. ² Boston, *A Textbook of Clinical Diagnosis*, p. 187. ³ J. Dixon Mann, *Physiology and Pathology of the Urine*, p. 128.

FRENCH CONGRESS OF GYNAECOLOGY AND OBSTETRICS.—The fifth periodical Congress of Gynaecology, Obstetrics, and Paediatrics, will be held at Algiers, April 1st to 8th, 1907, under the general presidency of Professor Queirel, of Marseilles, who will also preside over the Section of Obstetrics. Dr. Boursier, of Bordeaux, will be President of the Gynaecological Section, and Dr. Guinon, of Paris, of that of Paediatrics. The General Secretary is Professor Jules Rouvier, 52, rue Daguerre, Algiers.

RHEUMATIC FEVER AND AMYLOID DEGENERATION.

By JAMES M. BEATTIE, M.A., M.D.,

LECTURER ON PATHOLOGICAL BACTERIOLOGY AND SENIOR ASSISTANT TO THE PROFESSOR OF PATHOLOGY, UNIVERSITY OF EDINBURGH.
[From the Pathological Laboratory, University of Edinburgh.]

THOUGH the causes of amyloid degeneration are not clearly understood, still it is generally recognized that in the great majority, if not all cases, the condition is secondary to some infective agent. This infective agent is generally bacterial, and has been acting for a considerable length of time. The experimental work of Krakow, Maximow, Davidsohn and others has all gone to support the bacterial agent as the cause, and if we exclude the experiments of Czerny, which have not been confirmed by others who carried out similar work, the degeneration has been produced artificially only by bacteria or their products. It is true that cases of amyloid degeneration have been recorded as following cancer, leukaemia, malaria, and lymphadenoma; one of these is due to a parasite which can produce toxic products, very much allied to the products of bacteria, and, while the cause of the other three diseases is still unknown, I do not think I am stating too much in saying that the majority of pathologists favour the parasitic origin of at least two of them. In view of these facts, it seemed to me that in investigating and upholding the bacterial origin of acute rheumatism some help might be obtained by a study of the more chronic lesions which all recognized as sequelae of acute rheumatism, and the possibility of their experimental production by the streptococcus which has been claimed as causal.

At the commencement of my observations my attention was directed to the presence of amyloid degeneration in various organs of a case of cardiac disease following acute rheumatism. Since that time three other cases have been noted by me.

CASE I.

A. P., female, aged 28, was admitted to the Royal Infirmary, Edinburgh, on November 9th, 1904, complaining of breathlessness.

History.—Seven months previously she had arthritis in the right ankle and was treated for acute rheumatism. This was followed a month later by arthritis in the knees and ankles, and in October, a month before admission, she had pains over the heart and she became breathless. There was no history of any previous attack of acute rheumatism, and till June, 1904, her health had been perfectly satisfactory.

Condition on Admission.—There was a well-marked systolic murmur over the mitral area and other signs of definite cardiac disease.

After-history.—November 13th. There was a purpuric rash over the chest and upper part of the abdomen. On the flexor surface of the left forearm, about $\frac{1}{2}$ in. below the bend of the elbow, there was an egg-shaped swelling $1\frac{1}{2}$ in. long by $\frac{1}{2}$ in. wide. This was more definite on the radial than on the ulnar aspect. The left radial pulse at the wrist was hardly perceptible. The liver was slightly enlarged and tender on palpation. On November 16th there was marked diarrhoea. The diarrhoea continued. Tenderness over the liver increased, and very acute tenderness developed over the right lower ribs, especially the fifth and sixth anteriorly, and in the axilla, and continued till the death of the patient on December 4th, at 6.10 a.m.

Autopsy (December 4th, 11 a.m.).

Heart and Pericardium.—There was 1 oz. of clear serous fluid in the pericardial sac, no adhesions, and no recent pericarditis. The endocardium showed a few thickened patches of chronic endocarditis. Over the right ventricle there was an excess of fat. The right auricle was filled with recent blood clot and was slightly dilated. The right ventricle was also slightly dilated. The tricuspid and pulmonary valve segments showed no special change. The left auricle was dilated and filled with recent blood clot. The left ventricle was hypertrophied and slightly dilated. The aortic valve segments were slightly thickened, especially at their attached margin, but there was no evidence of any recent aortic endocarditis. On the inferior surface of the anterior segment of the mitral valve there was a large mass of fairly firm vegetations, in the form of irregular nodules. The largest of these was $\frac{1}{2}$ in. in diameter. On the auricular surface of the posterior segment there were some small recent warty vegetations, which spread upwards for $\frac{1}{2}$ in. over the wall of the auricle. There was neither ulceration nor rupture of any of the segments. There was chronic thickening of all the mitral segments; but no stenosis apart from that caused by the vegetations. From the vegetations a culture corresponding in all characters with the *Streptococcus rheumaticus* was isolated.

Lungs.—There was slight emphysema with collapse at the

base. There were no infarctions, and chronic venous congestion was only slight in amount. There were a few fragments of recent lymph on the diaphragmatic surface of the left lower lobe.

Liver.—There was slight enlargement, with a moderate degree of chronic venous congestion. No amyloid reaction was obtained with iodine, but on microscopic examination some of the smaller branches of the hepatic arteries in the portal tracts gave the reaction with methyl violet. The change was very slight, and in some sections only one artery showing the change could be found.

Spleen.—It was much enlarged, and there were several yellowish recent infarctions. The Malpighian bodies were distinct and translucent, and with iodine gave a definite amyloid reaction. Microscopic examination showed the amyloid change confined to the Malpighian bodies. In some only the reticulum at the periphery was affected, but in others the degeneration had spread almost to the centre.

Arteries.—The walls of the smaller arteries were swollen and homogeneous, and gave a distinct amyloid reaction, but the larger ones were unaffected. There was an embolism of the left brachial artery.

Kidney.—This was the only one present. It was enlarged, and contained old and recent infarctions, and with iodine it gave a very slight amyloid reaction. On microscopic examination there was amyloid change in some of the capillaries in the glomerular tufts. No degeneration was made out in any of the afferent or efferent arterioles, and only here and there in the walls of the straight arteries in the pyramids could any amyloid reaction be obtained.

The suprarenals and thyroid showed no amyloid change, but the intestine gave a very definite reaction with methyl violet.

There were congenital deformities in the generative organs, but as these could have no special relation to the amyloid degeneration they need not be discussed here.

CASE II.

C. M., male, aged 19, was admitted to the Royal Infirmary, Edinburgh, on February 17th, 1904.

History.—On February 7th, 1904, his left knee was swollen and very painful. On the 8th his wrists were swollen, and on the 9th he developed pain over the sternum. There was no history of any former attack of acute rheumatism.

Condition on Admission.—There was effusion in both knee-joints, but no pain or redness in any of the joints. The fingers were clubbed, and the shape of the chest and its localized bulging suggested old-standing cardiac disease. In the aortic area there was a soft systolic murmur with a suspicion of a diastolic, and in the mitral area there was a very distinct systolic murmur.

After-history.—On February 28th the patient was perspiring very profusely, but there was neither pain nor swelling in any of his joints. On February 29th there was slight pain in the left knee. On March 2nd the right knee was swollen, red, and painful. The patient recovered, and was discharged on April 5th. During his stay in the infirmary the urine contained albumen which varied from 2 to 10 gr. to the ounce. The albumen was absent when the patient was discharged.

On April 9th he was readmitted with considerable swelling in the right knee, which had been present for two days. There was also swelling in the left knee. He was discharged on May 4th, though there was still a slight trace of albumen in his urine.

He was readmitted on January 7th, 1905. There was marked dyspnoea and cyanosis, and slight diarrhoea. The heart murmurs were unchanged. On January 11th there was dullness with moist sounds at the base of the right lung, and the sputum was slightly blood-stained. On January 22nd diarrhoea was again present, and on January 23rd there was an erythematous rash over the trunk and face. This became more marked, and on January 26th it was distinctly purpuric, and was specially marked over the whole of the lower extremities. There was slight oedema of the feet. The patient was extremely prostrate, and died the same evening.

Autopsy (January 24th).

Heart and Pericardium.—The pericardium was thickened and universally adherent to the epicardium. The right auricle was dilated and hypertrophied, and contained some recent *ante-mortem* thrombus. The right ventricle was hypertrophied but not dilated. The tricuspid orifice was dilated and the valve segments slightly thickened. The pulmonary valve segments were healthy. The left auricle was dilated and hypertrophied, and the left ventricle was hypertrophied. The aortic valve segments were thickened, especially at their free margins, and were slightly incompetent. There was no stenosis and no recent endocarditis. The mitral valve segments were very considerably thickened, and the orifice was dilated. There was no recent endocarditis.

Lungs.—There were numerous fibrous adhesions in both pleural sacs and very numerous subpleural petechial haemorrhages over both lungs. On section, the lung tissue showed advanced chronic venous congestion, and several infarctions were present in the left lung.

Liver.—There was advanced chronic venous congestion but no amyloid reaction to iodine. Microscopically there was a definite amyloid reaction to methyl violet in some of the smaller branches of the hepatic artery in the portal tracts.

Spleen.—This gave a very definite "sago-waxy" reaction to iodine. The Malpighian bodies were enlarged and translucent.

Kidneys.—The surface was smooth, extremely pale, and somewhat mottled. On section, there was marked pallor of the whole organ, with, however, congestion of the veins in the pyramids. The larger vessels were not thickened. Both superficial and deep cortex were much swollen and of a uniform pallor. The Malpighian bodies were prominent, and gave a definite mahogany brown colour with iodine. Microscopically the capillaries of the glomerular tuft showed well-marked amyloid degeneration. The afferent and efferent arterioles showed the reaction only occasionally. The amyloid change was also seen in a few of the intertubular capillaries, and was very marked in the straight vessels in the pyramids.

Small Intestine.—The solitary glands were enlarged, and the amyloid change was quite definite in the capillaries of the villi.

CASE III.

J. W., male, aged 21, was admitted to the Royal Infirmary, Edinburgh, October 29th, 1903, with cough and with swelling of feet and legs of six weeks' duration.

History.—He had rheumatic fever at the age of 17, and has had bronchitis every winter since.

Condition on Admission.—There was palpitation and dyspnoea, bulging of the praecordia, and diffuse pulsation in the sixth interspace and in the episternal notch. There was a diastolic thrill over the apex beat, and on auscultation a pre-systolic murmur was heard in the mitral area, and a systolic in the aortic. There was dullness, with harsh vesicular breathing and rhonchi at the base of both lungs. The urine was alkaline, had a specific gravity of 1010, and contained a large quantity of albumen.

After-history.—On November 2nd there had been profuse diarrhoea for two days. This continued, and the patient died on November 9th.

Autopsy.

Heart and Pericardium.—The right auricle was dilated, and contained some *ante-mortem* clot. The tricuspid orifice was stenosed to a moderate degree, the valve segments were thickened, and there were some recent vegetations along the free margins of the cusps. The right ventricle was small, the wall was hypertrophied, and at the apex showed a moderate degree of fatty infiltration. The pulmonary valve segments were healthy. The left auricle was hypertrophied. The mitral orifice was narrowed and the cusps were thickened. There were no vegetations. The left ventricle was dilated and hypertrophied. The aortic orifice was stenosed, and the cusps thickened. There was no recent endocarditis. The pericardium was universally adherent, and there was some fibrous invasion of the myocardium.

Lungs.—Both were emphysematous and showed chronic venous congestion. In the lower part of the right lung there was a recent infarction.

Liver.—There was slight enlargement. Early chronic venous congestion and fatty change were the only pathological conditions noted. There was no amyloid reaction to the iodine test. Microscopically the branches of the hepatic artery in almost every portal tract showed a definite amyloid change. In some of the tracts the wall of the portal vein was also affected. There were a few patches of amyloid change in the capillaries between the liver cells, and especially in the intermediate zone of the lobules.

Spleen.—There was well-marked chronic venous congestion. The Malpighian bodies were enlarged and translucent, and gave a definite amyloid reaction with iodine. Microscopically all the Malpighian bodies showed the amyloid change. In some only the peripheral zone was affected, and in some the wall of the arteries was free from the degeneration. There was very slight amyloid change in the walls of the sinuses of the pulp, and this only in a few scattered areas.

Kidneys.—These were pale and enlarged. The cortex was swollen and translucent. There was definite amyloid reaction to iodine in the glomeruli and in the vessels in the pyramids. Microscopically amyloid change was present in all of the glomerular tufts. In some there were merely irregular patches near the periphery, in others the change was most marked near the centre, and in others the whole of the capillaries of the tuft were affected. The afferent arteriole showed the change irregularly. A few of the intertubular vessels were waxy. The straight vessels in the pyramids showed the degeneration in a marked degree.

Adrenals.—In the adrenals the amyloid change was very extensive both in the cortex and in the medulla.

Small Intestine.—The small intestine showed the degeneration in a marked degree, and there were a few patches in some of the mesenteric glands.

CASE IV.

M. D., female, aged 20, was admitted to the Royal Infirmary, Edinburgh, on March 10th, 1906, complaining of pain in the left side, dyspnoea, and swelling of face and ankles.

History.—She had rheumatic fever in childhood, and had had rheumatic pains frequently.

Condition on Admission.—She was cyanosed and had very marked dyspnoea. There was a well-marked systolic thrill in the episternal notch, and in the neck. A soft systolic murmur was heard in the mitral area, and in the aortic area a systolic and a faint diastolic. Over the whole of the chest there were numerous bronchitic sounds. The urine contained 4.2 gr. per oz. of albumen.

Death.—While in hospital the patient became very dropsical, and died suddenly on March 31st.

Autopsy.

Heart and Pericardium.—The pericardium was adherent to the epicardium over the anterior surface of the heart, and in the adhesions there was some calcareous deposit. The right auricle was dilated and its endocardium thickened. The tricuspid orifice was narrowed and the valves showed recent vegetations. The right ventricle was hypertrophied. At the apex there was fatty infiltration. The pulmonary valves were slightly thickened, especially at their margins, and there were several small clusters of recent vegetations at the corpus Arantii. The left auricle was dilated and its endocardium thickened. The mitral orifice was stenosed. The valve segments were thickened, and on the auricular aspect there were irregular vegetations, some calcified, others soft and easily broken down. The left ventricle was hypertrophied. The mitral orifice was narrowed, the cusps were thickened and contracted, and the chordae tendineae were also thickened. The aortic valves were incompetent, were very much thickened by old endocarditis, and also showed some recent vegetations.

The liver showed nothing specially important, and there was no reaction to the iodine test. Microscopically some of the smaller branches of the hepatic artery in the portal tracts gave a definite amyloid reaction with methyl violet.

The spleen was enlarged and soft. The Malpighian bodies were prominent, but gave no reaction with iodine. Microscopically there was very definite amyloid change in the fibrous reticulum of the Malpighian bodies, especially at their periphery, but very little atrophy of the lymphoid cells.

Kidneys.—There was enlargement and pallor. On section the cortex was swollen, pale, and translucent. There was amyloid reaction to iodine in the glomeruli and in the vessels of the pyramids. Microscopically the capillaries of the glomerular tufts showed the degeneration, and there was also very definite change in the straight vessels in the pyramids. The intestine was not examined.

These four cases present many features in common. They were all young subjects who had cardiac disease. In all there is a very definite history of rheumatic fever. The rheumatic attacks were frequent, and in all of them there is evidence that the rheumatic poison, be it what it may, was circulating in the body for from seven months onwards.

In the first case the history points to the first infection being only seven months before the death of the patient; but during that time the attacks had been frequent—in fact, the poison seems to have been absorbed almost continuously during these months.

In the second case the history is of a year's duration, with again frequent attacks of the disease. The third and fourth cases are of several years' duration.

Thus we have in these cases the usual history of amyloid degeneration: a toxin being absorbed fairly constantly for a prolonged period, and, so far as could be made out from the history and from the microscopic examination of the organs, the only poison acting here was that of acute rheumatism. There certainly was neither tuberculosis nor syphilis in any of the cases. Now it is generally admitted, as has been already stated, that the causal toxin is bacterial in origin, and in one case an organism corresponding with the *Micrococcus rheumaticus* was isolated. Lending support to the view that the amyloid degeneration was caused by the poison of rheumatism is the relation of the infection to the only definite clinical manifestation of the degeneration—the diarrhoea. This is recorded in three of the cases (in the fourth there is unfortunately no record, either clinically or *post-mortem*, of the condition of the intestine), and in all it appeared just a short time before death.

The distribution of the amyloid change is also very similar in the four cases, but as this is given in detail in the report of the cases it need not be again referred to.

These cases seem to me to give considerable support to those of us who maintain that acute rheumatism is a bacterial disease, and it is for this purpose and this alone that I bring them forward.

In conclusion, I have to thank Professor Greenfield, Dr. Byrom Bramwell and Dr. James for permission to use the notes of the cases, and my colleague, Dr. Carnegie Dickson, for the *post-mortem* record of one of the cases.

MYOMECTOMY DURING PREGNANCY AND LABOUR AT TERM IN AN ELDERLY PRIMIPARA :

WITH NOTES ON SIMILAR CASES.*

By ALBAN DORAN, F.R.C.S.,
SURGEON, SAMARITAN FREE HOSPITAL.

MYOMECTOMY has often been performed during pregnancy with the most satisfactory results. Gestation is, as a rule, uninterrupted, and when labour sets in the expulsive powers of the uterus seem to be but little impaired. This has been noted even in subjects where the wound in the uterine walls inflicted at the operation was of considerable length, breadth, and depth. The case which I will now relate is mainly of interest because the patient had reached the age of 35 years and was pregnant for the first time. The history of the subsequent first labour is therefore, in my opinion, worth reporting. I will also make some mention of a few other instances of labour in elderly primiparae following myomectomy during pregnancy, taken from the reports of the experience of others published during the current year.

A woman, aged 35, was brought to me for examination at the end of February, 1906. She had consulted Dr. Meerwald, of Maida Vale, on account of amenorrhoea. The last period began on October 25th, 1905. Morning sickness troubled her during November and December. Dr. Meerwald detected a swelling, with all the characters of a gravid uterus, above the pubes, but the pelvic condition appeared abnormal.

The patient was strong and healthy; the abdominal walls showed no lineae albae. The body of the uterus could be clearly felt in the hypogastrium, occupying two-thirds of the space between the umbilicus and pubes. The cervix was fairly long and firm; the os externum narrow and oval. A prominent conical body occupied the left fornix; it was very tender when pressed, and moved slightly with the uterus. On rectal examination it was found to lie in front of the bowel. The upper limits of the body in the pelvis could not be clearly defined. The patient's general condition was satisfactory.

Thus there was evidently a morbid growth connected with the posterior part of the lower segment of the uterus, most probably a fibroid likely to prejudice labour. Considering the small risk, even of interruption of pregnancy, which attends myomectomy, I decided to operate, although the tumour lay in a rather awkward position for manipulation without much disturbance of the gravid uterus.

Operation.—On March 6th I operated, with the assistance of Mr. Butler-Smythe. Mr. Morley administered ethyl chloride, followed by ether and then chloroform. The pelvis was elevated. I prolonged the abdominal wound to 1 in. above the umbilicus, so that I was enabled to draw out the gravid uterus and examine its lower and posterior aspects without dragging or pressing. I then discovered two subserous, pedunculated growths springing from the posterior aspect of the inferior uterine segment, close to the left border of the uterus. By cautious rotation of the uterus towards the right I was able to bring the two growths out of the lower part of the abdominal wound. Gauze pads were packed behind the uterus and in the left iliac region. The smaller growth was 2 in. long, very tough, and attached to the uterus by a short vascular pedicle, awkwardly near to the larger tumour. It would hardly have interfered with labour, but I found that I could not very well reach the bigger outgrowth until I had removed it. I enucleated it in part, split it open, and then was able to secure its pedicle by transfixion with No. 4 China twist.

The larger growth was now more conveniently placed for removal. It measured over 4 in. in length and 2 in. at its broadest part, and it ended in the very blunt point so prominent in the pelvic cavity when the patient was examined. The pedicle was relatively thick. I enucleated the growth to a certain extent after splitting it, and closed the remains of the capsule with a No. 1 continuous silk suture; then I transfixed it with stouter silk. The growth, a calcified fibromyoma, adhered strongly to the capsule, so that only a short piece of capsule could be saved. On that account I was compelled to transfix, as not until the transfixing suture was tied could I stop the bleeding. The peritoneum was closed with No. 1 Van Horn catgut; the integuments and sheath of the recti with interrupted silkworm gut.

There were no complications during convalescence, nor in the further course of the pregnancy, which went on to

* A paper read before the Obstetrical Society of London, November 7th, 1906.