CASES ILLUSTRATIVE OF PROGNOSIS IN HEART-DISEASE.*

By W. H. BROADBENT, M.D.,
Senior Assistant-Physician to St. Mary's Hospital.

CASE III.—Mitral and Aortic Disease of Rheumatic Origin: Pneumonia: Recovery from the Pneumonia.—J. P., a greengrocer, aged 34, had acute rheumatism fourteen years before. He had been ailing at various times since, and had tubercular deposit in the apex of the lung. He was seen, with Dr. Langmore, April 13th, 1866, when he had been ill about a week. After hard work, and exposure to cold and wet, he had had rigors, followed by cough, with expectoration of adhesive mucus, tinged with blood, and accompanied by dyspnoea—an attack of pneumonia, in fact. When I saw him he was perspiring profusely, his manner was excited; his hand trembled; he had been delirious in the night, and still believed in the illusions of the delirium. Pulse 110; respiration 40. There was dulness over a considerable extent of the lower lobe of the right lung, with fine crepitation and tubular breathing. The dulness not absolute, and the tubular breathing not well marked. There was also dulness over a small part of the base of the left lung, and fine crepitation was there audible. As to the heart, the impulse was forcible and extensive; most powerful about the fourth cartilage. The apex-beat was felt in the sixth space to the left of the nipple line. At the apex was heard a smooth systolic murmur (mitral). This was lost in passing towards the sternum, and succeeded by first a diastolic murmur, and then a systolic aortic murmur; the diastolic murmur being loudest over the ventricle; the systolic quite at the base; no second sound was heard in the neck. The prognosis given was favourable as to the existing attack. First, on the ground that the condition of the valves was stationary, and that the enlargement of the heart, consecutive on this, though considerable, was not extreme, and hypertrophy predominated over dilatation. Again, the pneumonia was accounted for by an adequate external cause—fatigue and exposure; and was not a development of the consequences of the heart affection. The dyspnoea was not greater than the extent of lung affected by pneumonia would explain, and there was no dropsy—strong reason for concluding that there was no great obstruction to the blood. The prognosis was verified by the recovery of the patient from the pneumonia, and his restoration to his previous state of health.

CASE IV.—Mitral Valvular Disease of Rheumatic Origin: very great Hypertrophy: Apoplectic Pneumonia: Dropsy: Death.—This case furnishes an instructive contrast with the last. Mr. B., aged 34, clerk in the office of a publisher, a strong-built man, had had acute rheumatism fourteen years before, only slight indispositions since, but he was soon out of breath on exertion. A week or ten days before I saw him, he had begun, after a little overwork, to suffer from cough and dyspnoea. Œdema of the legs set in and gradually increased. The cough had been relieved by treatment. When first seen by me he was up and dressed; he was unable to lie down on account of increased difficulty of breathing. The face was anxious, respiration embarrassed, there was still cough and a little blood-tinged expectoration, the legs were cedematous and congested, pulse frequent and feeble, but regular. There was dulness (not absolute) over the bases of both lungs, with crepitation, and imperfect entry of air. The heart was extremely large, the impulse very forcible, and felt over a large area; the apex-beat was in the sixth space at about the line of the axilla; a loud systolic mitral murmur was here audible. In this case the prognosis was unfavourable. The heart was of an enormous size, indicating the serious character of the mechanical obstruction to the circulation to which the valvular change had given rise. The hypertrophy had for many years compensated for the difficulty; but the balance, once disturbed, could not readily be restored. A very slight cause, a little over-exertion, had sufficed to effect this; and it was evident that the pulmonary congestion and the œdema of the legs were the direct consequences of the obstruction to the circulation by the cardiac disease. The respiratory embarrassment continued to increase, and the patient died. No *post mortem* examination could be obtained.

CASE V.—Aortic Regurgitant Disease: Degenerative in Character: Probable Damage by Exertion: Apoplectic Pneumonia: Death.—E. B., aged 51, coachman, married, the father of four children, having the look of a strong man of 45, came to me as an out-patient at St. Mary's Hospital, on October 16th, 1865. He had never had acute rheumatism, but had enjoyed good health till four years ago, when, after lifting a carriage, he was seized with fluttering of the heart, and felt faint; he was ill for three or four days, and had been more or less ailing ever since, but did not seem to have much the matter with him till eight

months ago, when, after another strain, he had beating of the heart pain in the cardiac region, and shortness of breath. He continued to work, however, till the week before he came to the hospital. When first seen, his breath was very short; he complained of palpitation; there was œdema of the legs; the heart was large, but the exact situation of the apex-beat could not be made out; the impulse was visible, but with difficulty felt by the hand; over the second and third spaces and adjacent part of the sternum, a double murmur was heard; the diastolic murmur being louder and rougher and continued downwards, the systolic heard more distinctly higher up; no second sound was heard in the neck. He gradually became worse, and on November 9th, at my request, became an in-patient, under Dr. Sibson. By this time, apoplectic pneumonia had set in, and the difficulty of breathing had increased, so that he was unable to lie down. He died on Nov. 13th. On *post mortem* examination, fluid was found in both pleural cavities, and blocks of pulmonary apoplexy in both lungs; the heart was large; the left ventricle hypertrophied and dilated; the mitral valves healthy, but the orifice large; the aortic much altered, the flaps being scarcely recognisable, but replaced by irregular masses, projecting towards the aorta, which could be everted into the ventricle, thus bearing out to some extent the idea that, when altered by atheroma, the valve had given way under a strain; the right ventricle and auricle were dilated and thickened; the aorta was extensively atheromatous. If in heart-disease a good aspect, and strong, well-built vigorous frame could warrant a favourable prognosis, such would have been the case here. But the history was one of progressive change in the valves, accelerated by pressure upon them; the symptoms had come on without any such exciting cause as exposure, privation, or overwork, but were due to the heart-disease alone. There was little place therefore for treatment; it could not remove the change in the valves or the obstruction to the circulation; the heart walls had failed to compensate for this, and the conditions which had given rise to the secondary pulmonary congestion and systemic dropsy could not be reversed. No result, but that which followed was to be anticipated.

CASE VI.—Mitral Valvular Disease: Complicated by Albuminuria. S. P., aged 47, greengrocer, was admitted as an out-patient at St. Mary's Hospital, June 5th, 1865. He was a strong, stout man; he had been subject for two or three years to rheumatic gout, which had been worse than ever during the preceding winter, and had left, as he expressed it, swelling in the ankles and pain in the elbow. For five or six months he had been short of breath, but three or four months ago he could do anything without suffering fatigue. He now complained of shortness of breath, pain in the chest, and swelling of the legs, which had become considerable. The heart was found to be very large, the apex-beat being far to the left of the normal situation, and much lower; no murmur could be detected at this visit. The action was frequent, about 120 beats per minute. The first sound was short and sharp like the second, and there was no pause after the second, so that the two might be compared to the ticking of a watch; the urine was albuminous. On the 9th, and subsequently, a distinct systolic mitral murmur was audible. The pulse was frequent, thready, and irregular. Here the prognosis was unfavourable; the change in the valves appeared to be recent and progressive; the symptoms were of a very serious character, and had come on rapidly. The urine, moreover, was albuminous. The dropsy increased, and soon put an end to his attendance, his last visit being on June 29th. As he lived some distance from the hospital, I was unable to watch the case further at home.

CASE VII.—Mitral Valvular Disease: Non-Rheumatic: Progressive. M. J., aged 60, a brushmaker, came to St. Mary's Hospital December 4th, 1865. She was a spare, active, healthy-looking woman, and had never had any illness in her life. She complained chiefly of pain in the loins, hips, etc., and the only symptom which called attention to the heart was occasional palpitation. The heart was not greatly enlarged; the apex-beat was felt in the fifth space, below and external to the nipple; over this point, or to the left of it, no murmur was audible, but nearer the sternum, internal to and below the nipple, a smooth systolic murmur could be heard, but only over a limited space. Ten days later (December 14th) the murmur at the point named was at times musical, *i.e.*, about every fourth beat, at the respiratory pauses. It was occasionally heard also at the apex, and was musical. On the 21st, she was not so well; the heart's action was slightly irregular. The murmur heard over the fourth and fifth left costal cartilages was not musical. A murmur at the apex, audible occasionally only, was slightly musical. She continued to attend till March 4th, during which time the area of impulse increased, and the heart's action was noted to become more irregular in force and rhythm; the character of the murmur also changed somewhat; she became too ill to attend at the time named.

CASE VIII.—Mitral Valvular Disease: Non-Rheumatic: Death.—

* Concluded from page 520 of last number.

J. D., aged 40, became an out-patient at St. Mary's Hospital, August 20th, 1866. She had never had acute rheumatism; she had been ailing six or seven years; her face was sallow and tintless; she complained of being soon out of breath, and on exertion or excitement was seized with pain in the præcordial region and epigastrium, extending to the back, and down both arms to the fingers. It came and departed suddenly, and was accompanied by fluttering of the heart. At the same time she became giddy, and immediately afterwards had an evacuation of the bowels. The pulse was regular and frequent. On examination, the heart's impulse was found to be extensive and violent, the apex beat was to the left of the nipple line, and at this point, and to the left of it, a systolic murmur was heard, which diminished gradually in loudness towards the sternum and upwards. This poor woman was living very poorly, and was much distressed mentally by her husband's being long out of work. I was able to supply her with good food for a time from St. Mary's kitchen, and she seemed to improve; the pain and palpitation becoming less frequent and severe; the breath was not so short. On October 20th, however, after hearing nothing of her for nearly three weeks, I was called upon to sign a certificate of her death.

CASE IX.—*Constrictive Mitral Disease: Death*.—S. A. B., aged 27, married, and having two children, came under me as out-patient at St. Mary's Hospital, on October 15th, 1866. She had been ailing for eight years and under treatment for two years, complaining of cough, shortness of breath, and palpitation of the heart on walking. She had had dropsy; she had never suffered from rheumatism. These symptoms were present in an exaggerated degree; there was fluttering of the heart on the least exertion; in the night the legs felt as if going dead; she had no appetite, and was frequently sick. She looked anxious and worn, and her breathlessness was evident; the pulse was scarcely perceptible at the wrist, where only an occasional beat could be detected; the action of the heart was very irregular and intermittent; the apex beat was beyond and below its natural situation, it was readily felt; the chief impulse was at the third and fourth cartilages and below, and a peculiar vibration was felt at times; a systolic murmur was heard on the lower sternum, and along this bone to the level of the third rib, also towards the left, to within an inch and a half of the apex beat, where it was loud and distinct; and the apex, and to the left of this it was not at all audible, nor over the base of the heart or aorta; the veins of the neck were not full. She was examined in the sitting posture. I gave this poor woman food and medicines calculated to give her strength, but she vomited everything. At my urgent request she entered the hospital, and was taken in under the care of Dr. Sibson on October 26th. Nothing could be done for her, and she died on November 6th. The heart was enlarged, especially the right side, and the tricuspid orifice was very large; the mitral valve was thickened and rendered rigid by infiltration into its substance; the orifice was so contracted as only to admit one finger; there were no deposits on its surface, and it had not undergone the usual degenerative change. During life, the seat of the murmur was a subject of question, the conclusion being that it was at the tricuspid orifice. The examination after death leaves it still uncertain. Wherever the murmur had its origin, however, the change in the mitral valve was the cause of the derangement in the circulation, and of the enlargement of the right ventricle. The prognosis in this case was only too plain; the disease in the valve had evidently been progressive, though at the age of the patient it could scarcely be expected that it was atheroma; but this consideration had not much weight under the circumstances. The symptoms of existing derangement in the circulation were so serious as to claim the entire attention to the exclusion of all questions as to the nature of the cause of the derangement. Vomiting is a sign of very bad omen in the late stages of heart-disease, and is not to be met by usual remedies. Its precise cause is not very clear; whether it is from the mechanical disturbance by the labouring heart, which agitates the diaphragm and with it the stomach, or a result of congestion, or whether it is induced through the pneumogastric nerve.

The remarks given with each case will render unnecessary any prolonged remarks on them collectively. The first three show that when the history gives reason to believe the valvular disease to be stationary, and the existing dropsy or pneumonia are traceable to some unfavourable condition, or accidental circumstance, *plus* the heart-affection, recovery may be anticipated. Case V may be instructively compared with Case II. These men were attending the hospital together; and of the two, the symptoms seemed more urgent in Case II; but the history and examination of the heart furnished ground for a very different prognosis. Cases III and IV again may be compared, the actual condition of the heart, and the cause to which the attack was traceable—slight and inadequate in the one, in the other amply sufficient to account for it—being here the indications which pointed to a different result.

CASE OF "RED SOFTENING" OF THE SURFACE OF THE LEFT HEMISPHERE OF THE BRAIN: WITH SUDDEN LOSS OF SPEECH AND HEMIPLEGIA.

By H. CHARLTON BASTIAN, M.A., M.D.,

Lecturer on Pathological Anatomy and Assistant Physician to St. Mary's Hospital.

THIS case presents several features of great interest in their bearing upon the physiology and pathology of the brain. The person was seen by myself only in the *post mortem* room; and I have to thank Dr. Alderson for having kindly granted me permission to publish the details of the case. It seems to me desirable to do so, principally because it throws a certain amount of light upon the question as to whether an injury of the convolutional grey matter of one hemisphere of the brain (that is to say, of a portion of the centre whence volition emanates) is adequate to produce a hemiplegia, without coexisting structural disease in or near the central ganglia of the same side. It presents other points of interest, however, as will be seen.

Frances D., aged 62, was admitted into St. Mary's Hospital, under the care of Dr. Alderson, on December 21st, 1866, with signs of renal disease. There was slight anasarca, from which she had been suffering only a short time. The urine contained some albumen; but the specific gravity was nearly normal, and no casts are reported to have been found. She remained in much the same condition for about two months, though she gradually became weaker, and suffered from occasional headaches previously to the commencement of the cerebral symptoms about to be mentioned.

March 7th. At 5.15 P.M., having previously been in her ordinary condition, and having been speaking to a fellow-patient only five minutes before, she was seized suddenly with trembling and great mental agitation. There were no convulsions, but she frequently endeavoured to look behind herself in a terrified manner. She was then found to have lost the use of the right side of the body, though there was no facial distortion or paralysis of any kind. How far sensation was lost on the paralysed side, does not seem certain. At first, she was unable to speak at all; but after two hours she was able to say "Yes," in answer to questions put to her; and this was her invariable reply, whatever the nature of the query. She seemed, however, to understand what was said, and also recognised those around her.

March 9th. When the sister in charge of the ward (who had been absent) saw her in the evening, for the first time since this attack, D. recognised her, smiled, and said "yes." On being asked by the sister what was the matter, after a pause, she said with great effort, and in a stammering way, "I can't tell," just plainly enough to be understood.

March 11th. She said, in reply to the same query, also with great effort, "I can't understand it"; but, on other occasions, almost invariably the only reply, remark, or greeting, was the word "yes," although this was accompanied by actions of a more intelligent nature. From the first she passed feces and urine involuntarily. At no time were there twitches and convulsions; and whilst the right arm and leg were paralysed, the muscles of the face were entirely unaffected.

March 14th. Breathing became very laborious. She became more unconscious—no longer appearing to recognise persons around her—and died the next day without fresh symptoms.

AUTOPSY.—The body was rather thin and emaciated; there was anasarca of the right lower extremity. *Brain*.—The subarachnoidal fluid was more abundant than natural, even considering age; the veins in the pia mater of the right side were very full and turgid, presenting a marked contrast to those of the anterior two-thirds of the surface of the left hemisphere, which were even less distended than usual. Here and there, however, on this side, there were small patches of effused blood in the pia mater, mostly over the sulci. The arteries at the base of the brain were all notably diseased; their coats being thickened, and containing large white calcified patches at intervals. On slicing the cerebral hemispheres, that on the right side was found to present nothing abnormal; whilst the left showed very many patches of *red softening*, which were almost always strictly confined to the grey matter of the convolutions. Those on the upper surface corresponded with the superficial effusions before-mentioned, though there was no trace of other patches till the brain was sliced in various directions. The principal patches of softening were met with in the third or inferior frontal convolution, together with a portion of the adjacent ascending parietal near its commencement; whilst a still larger patch was situated in that part of the superior frontal convolution, where it forms the edge, and descends into the great longitudinal fissure (about opposite the junction of the anterior third with the