REPORT OF A CASE OF PYÆMIA FROM VALVULAR DISEASE OF THE HEART: DEATH.*

By S. WILKS, M.D.; W. W. GULL, M.D.; and J. M. BRIGHT, M.D.

THE following is an account of the last illness of the late Dr. Ray of Dulwich, of whom an obituary notice is given in another page. The case possesses peculiar scientific interest from its extreme rarity, and from the cause of death being one with difficulty recognisable.

For many years Dr. Ray had been the subject of a loud cardiac *bruit*, but attended by so few if any symptoms, that its nature was altogether conjectural. He himself believed that it was exocardial, or altogether external to the heart. The *bruit* was inframammary, and had been described by some of the most eminent physicians in London as systolic, presystolic, postsystolic, and even diastolic.

A day or two before Christmas-day last, his illness commenced with defective memory, succeeded by nausea and vomiting, with chills followed by feverish symptoms, loss of appetite, weakness, and general *malaise*. As he did not recover at the end of a few days, it was thought that fever had attacked him. He then sent for his old friend Dr. Hughes Hewitt, who was throughout unremitting in his attention, and, by a constant attendance, did all that friend could to relieve the symptoms. Dr. Hewitt took early alarm at the symptoms, and sought the cooperation of Dr. Gull and Dr. Wilks, who thenceforth met him in frequent consultation.

About a fortnight after the commencement of his illness, he lay in bed, making no especial complaint, having occasional chills, followed by drenching acid sweats—so commonly regarded as characteristic of rheumatic fever—with slowness of pulse (about 80), furred tongue, and a sallow dusky hue of countenance, with hepatic pain and complete absence of bile in the evacuations. At the end of three weeks, some slight apparent improvement had taken place; but, at the end of the fourth week, he had a severe rigor, followed in two days by a second. He seemed more prostrate, and the pulse began to rise. These symptoms suggested pyæmia, which Mr. Hilton, who now saw him, immediately confirmed; but the source of the infection was not to be found. The same symptoms continued; the intense sweats, following occasional rigors, making the diagnosis of blood-poisoning more evident, when, from the fact of the existence of hæmorrhoids and possibly some slight ulceration, a suppurative phlebitis of the portal veins was conjectured to be the malady. About this time, another symptom appeared, referable to the brain. From the first, the memory had been at fault; the articulation was always slow, and frequently defective; but now Dr. Ray forgot certain words. His intellect was clear, but he not infrequently confounded one word with another; he was, in fact, slightly aphasic. The rigors, which at first occurred every day, afterwards every three, four, or seven days, now ceased for a short time; and, the feverish symptoms abating, the patient thought himself better, and made an effort to leave his bed and recline upon a couch. This remission was unhappily of short duration; the rigors again returned with increased severity; and, as they appeared on several occasions on alternate days, Dr. Ray thought he had tertian ague, and requested quinine in heroic, or, as he termed it, "leuconic" doses. He had now been ill six weeks, with the symptoms commonly regarded as characteristic of pyæmia; indeed, had he undergone any surgical operation, such a diagnosis would have been made without hesitation.

About ten days before death, the rigors ceased for a few days, but then recurred with greater violence than ever, leaving the patient terribly prostrate. Difficulty of breathing now supervened, and soon expectoration, at first bronchial, but afterwards tinged with specks of blood. The lungs did not expand freely; there were universal *râles*; and it was thought that lobular pneumonia had now set in to end the scene. With these chest-symptoms, Dr. Ray died on Saturday, February 22nd.

Post Mortem Examination in the presence of Dr. Gull, Dr. Wilks, and Dr. Hewitt.—On opening the body, it was at once seen that there was no pyæmia, in the sense ordinarily understood by the term, *i.e.*, as affecting either the systemic or portal veins, but that there was a pyæmia of the arterial system. The blood had been poisoned, but the source of the infection was the heart. The cause was clear, and the results were manifest in the viscera. The kidneys showed the well-known fibrinous plugs, embolic infarctions as they are sometimes called. The spleen contained several such masses, and was enlarged in consequence to about three times its usual size. The deposits in the spleen were soften-

ing down into a semi-fluid state. Whether this material was true pus or simply decomposed fibrine, there was no opportunity of ascertaining. The heart was somewhat enlarged in all its proportions; the aortic valves were healthy; and the mitral, with the exception of the disease about to be described, also healthy, unless, indeed, the orifice was somewhat larger than natural. On looking down upon the mitral aperture from the auricle, a projection was seen standing out from the posterior curtain; it was about the size of a walnut, and had a quantity of softening fibrine and clot attached to its summit. It thus had the appearance of an aneurismal pouch formed in the valve. On turning back the valve itself, the mouth of the sac was seen; this admitted the tip of the finger into its interior, but did not allow it to pass through the orifice which existed at its summit. This sac was evidently an old formation; but whether its walls had been originally made out of the valve, or whether it was altogether a separate formation, was not very obvious. Its exterior surface was smooth and continuous with the endocardium; the interior surface was at the entrance of the sac, evidently formed of the invested valve. The orifice at the summit was round, and appeared to have been a part of the original formation, but on the side of the sac there was a rent which was apparently recent; and on this ragged edge was fresh blood and disintegrated fibrine. It was thus not positively ascertained whether the pouch had originally been a true aneurism, of which the walls had subsequently thinned and become destroyed, or whether it was formed out of lymph as a result of endocarditis. The brain was apparently healthy; no embolus was seen in the larger arteries, but whether the smaller vessels were blocked could not be discovered without a more thorough examination; and, therefore, whether the slight aphasic symptoms from which the patient suffered were due merely to the disturbance produced by poisoned blood, or by a more direct cerebral lesion from obstructed vascular supply, is uncertain.

The pathology of the case was now clear. Embolic particles, or disintegrated fibrine, had been circulating in the blood, and had given rise to the symptoms resembling those of pyæmia. This term generally implies that the venous blood is infected; but in the present case it was the arterial. The reason, probably, why this affection is not often recognised is because the endocarditis, which gives rise to the blood-poisoning, so impairs the valves that the main symptoms are those dependent on the deranged mechanism of the heart. In this case the disturbance of the heart was slight, while the blood-infection was the main cause of the symptoms. How the *bruit* was produced may still be a question. Looking at the large sac situated at the mitral orifice, and in the direct course of the blood through it, there seemed every probability that it might, by its interference, give rise to an auricular or direct mitral murmur, and for the same reason prevent a perfect closure of the valves, and thus be productive of a regurgitant murmur. In all probability, however, from the almost total absence of symptoms, the *bruit* was caused by the passage of blood through the aneurismal sac itself during every systole of the heart. If this were the case a small amount of blood must have been continually passing back into the auricle. Thus we can imagine a very loud *bruit*, but very little attendant ill. In all probability the rupture of the sac was the cause of the symptoms attendant on his last illness; a fibrinous coagulum forming on the rent which disintegrated and infected the whole blood.

THE LEVÉE.—By command of the Queen a levée was held on Tuesday, the 3rd instant, at St. James's Palace, by His Royal Highness the Prince of Wales, presentations to whom at this Court were, by the Queen's pleasure, considered equivalent to presentations to Her Majesty. There were about 150 presentations, including the following members of the medical profession: Dr. Clarence Cooper, Surgeon Madras Army, by the Secretary of State for India; Sir William Fergusson, Bart., F.R.S., on appointment as Sergeant-Surgeon to the Queen, by the Lord Chamberlain; Mr. John Hilton, F.R.S., on appointment as Surgeon Extraordinary to the Queen, by the Lord Chamberlain; Mr. Prescott G. Hewett, F.R.S., on appointment as Surgeon Extraordinary to the Queen, by the Lord Chamberlain; Dr. T. G. Howell, Madras Army, by the Secretary of State for India; Sir William Jenner, M.D., on being created a Baronet, by the Lord Chamberlain; the Bishop of Labuan, F.R.C.S., by the Duke of Buckingham and Chandos; Mr. T. B. P. O'Brien, Staff-Assistant-Surgeon, by the Adjutant-General; Mr. James Paget, F.R.S., on appointment as Sergeant-Surgeon Extraordinary to Her Majesty, by the Lord Chamberlain; and Dr. James Yearsley, by the Duke of Argyll. The following gentlemen also attended the levée: Sir Henry Holland, Bart.; Rev. David Bell, M.D.; Dr. A. Armstrong, Inspector-General R.N.; Dr. A. Farre; Dr. Stanley Haynes; Dr. Sieveking; Mr. G. B. Childs; Mr. W. White Cooper; Mr. Cesar H. Hawkins, F.R.S.; Mr. R. W. Tamplin; Mr. T. Wakley; and Mr. H. Haynes Walton.

* Communicated by Dr. Bright of Forest Hill.