CASE
OF
CYANOSIS
OF FORTY YEARS' STANDING,
DEPENDING UPON CONGENITAL OBSTRUCTION IN THE
PULMONARY ARTERY, AND PATULOUS FORAMEN OVALE.

BY ROBERT J. SPITTA, M.B., LOND.,

HOUSE-SURGEON TO ST. GEORGE'S HOSPITAL.

COMMUNICATED BY PRESCOTT HEWETT, Esq.

Received November 28th, 1845—Read January 27th, 1846.

IN recording the present case, I propose to adopt the following order:—1st, To describe the attack which immediately caused death. 2nd, To detail the history from birth. 3rd, To give the *post-mortem* examination. And, lastly, To draw four very simple pathological deductions.

1. *Attack causing death.*—On March 3rd, 1845, at 5 P.M., Miss M—— B——, ætat. 40, was suddenly seized, whilst seated quietly before the fire, with dyspnœa and partial syncope, accompanied at first with convulsions, and afterwards with intense pain referred to the epigastrium, loins, and hypochondria. This condition continued 24 hours, when, after a slight struggle, she expired. The mind was perfect throughout.

2. *History from birth.*—The previous history of this lady is as follows:—Miss B—— was very diminutive in stature, and pigeon-breasted, but without any positive deformity. She had cyanosis from birth, indicated by intense blueness of the cheeks, lips, and tongue. The heart's action (as far as

could be learned from her family) was regular when she was at rest; but running, or going up stairs, or any sudden exertion, produced great palpitation and dyspnœa. She suffered very much from the cold. Her intellect was of average power; and, with the exception of occasional dyspepsia, her general health was good.

Miss B——'s first serious attack, similar to that of which she died, was in October 1840.

In November 1843, Miss B—— had anasarca of the abdomen and lower extremities. The pulse was 100, not weak, but irregular as to number and power. The carotids pulsed visibly, but the jugular veins were not turgid. Of this attack, which lasted three months, she completely recovered, and never afterwards became dropsical.

Her second attack of dyspnœa and syncope was in the beginning of May 1844; her third was in September of the same year; her fourth in February of the present year, six weeks before death; and her fifth, which proved fatal, was in March last.

I may be allowed to state that the four first attacks were precisely similar in character to the fifth, described at the commencement of this paper, but that they differed in being less severe, and in the circumstance that they all followed violent exertion, whilst the fifth came on spontaneously.

Having only seen Miss B—— casually, I much regret that I have no information to offer respecting the sounds of the heart, of sufficient accuracy; I beg, therefore, to close the present history with this additional remark—that she never suffered from rheumatism in any form.

3. *Post-mortem examination* was conducted by Mr. Raymond Gasquet, Mr. Basil Barrett, and myself, 42 hours after death, and I am permitted by those gentlemen to add their testimony to the accuracy of the following description:—

Surface of the body was purple in the dependent parts. No œdema even of the ankles. The body was neither corpulent nor emaciated.

Thorax.—The pectoral muscles were flabby, and contrasted

remarkably with the firmness of the heart. There was no fluid in either pleural cavity; and, excepting some old but very small adhesions, and opacity and induration of small parts of the pulmonary pleuræ, these membranes were healthy. The lungs were congested, especially in their posterior parts, which also broke up easily under pressure; but, with the exception of four spots, about the size of shilling-pieces, of miliary tubercles, and red hepatization of the tissue between the tubercles, their structure was healthy.

The pericardium contained no fluid, and was healthy. The heart was very heavy, from the great firmness of its muscular tissue; and although small abstractedly, yet, compared with the size of the body, was somewhat enlarged. The enlargement was principally from hypertrophy of its walls, the dilatation of its cavities being slight. The hypertrophy was chiefly of the right side, the right ventricle being as thick as the left ventricle, and the right auricle three times as thick as the left auricle. The right auricle was also somewhat dilated. The foramen ovale was patulous; and, as it opened into the left auricle, was four lines in diameter. The auriculo-ventricular valves were thickened, but quite moveable. The aortic valves were healthy. The pulmonary artery was so malformed at its root as to render an accurate description of it extremely difficult. Besides the three semilunar valves usually found there, it presented an adventitious membrane situated immediately above them, and stretched completely across the artery, like the diaphragm across the trunk of the body. This membrane was a line in thickness, and perforated in its centre, not by a circular foramen, but by a mere slit two lines in length and a line in breadth, with margins of a red colour, and fringed with fibrine of the blood. The three semilunar valves were thrown up as they are naturally in a healthy pulmonary artery during the systole of the heart, and fixed in that position by the adhesion of (what would have been) their free borders to the adventitious membrane. The ductus arteriosus was impervious.

Abdomen.—The kidneys were of natural size, but a little

granular, and contained many and large cysts. The bladder contained acid urine, which precipitated on the addition of nitric acid, and coagulated to one-fourth its bulk on standing half an hour after ebullition. The other abdominal organs were healthy.

4. *Pathological deductions.*—1st. From the presence of cyanosis immediately after birth, and the total absence of rheumatic fever during life, that the membrane which obstructed the pulmonary artery was a congenital malformation. 2nd. That the marked hypertrophy of the right side of the heart was the consequence of the obstruction in the pulmonary artery. 3rd. From the circumstance that either an obstructed pulmonary artery or a patulous foramen ovale may exist separately without the blue disease, that the cyanosis in this case was the result of the two above-named lesions combined. 4th. That the circulation of imperfectly oxygenated blood impaired no function so much as the formation of animal heat, of which, as we before noticed, Miss B—— was remarkably deficient.