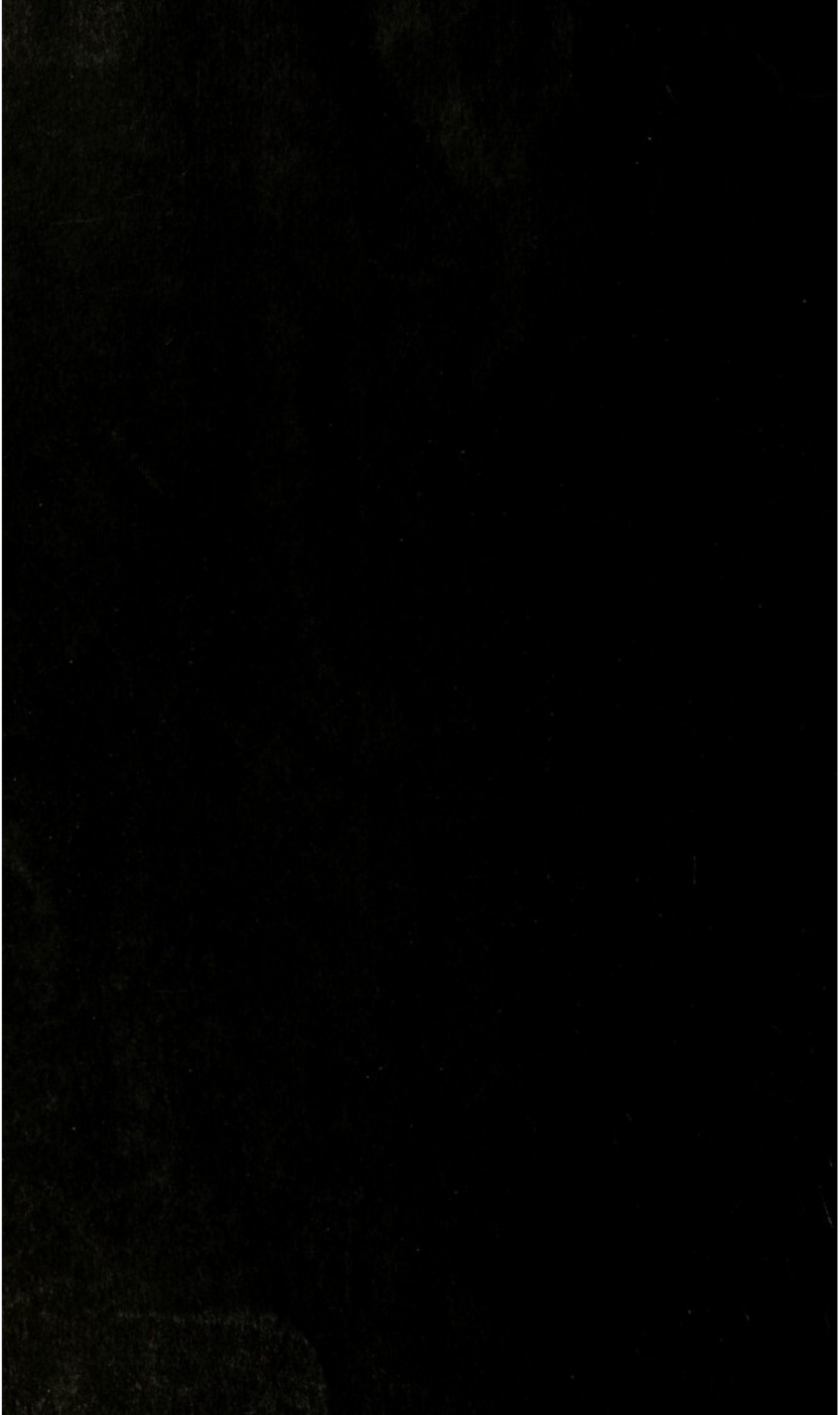
A case of tricuspid and mitral stenosis in which physical signs of pulmonary arterial reflux were present / by Sir Dyce Duckworth.



Reprinted from Vol. XXI of the 'Clinical Society's Transactions.']

A Case of Tricuspid and Mitral Stenosis in which physical signs of Pulmonary Arterial Reflux were present. By Sir Dyce Duckworth, M.D. *Read January* 27, 1888.

MARY S., æt. 23, a fancy box-maker, married three years, without family, was admitted to Elizabeth Ward in St. Bartholomew's Hospital under my care on May 31, 1887, suffering from general dropsy and dyspnœa. She had had scarlet fever at the age of five years, but never rheumatism or chorea. She remembered suffering from short breath on exertion when fourteen years old. For twelve months past she had been particularly subject to dyspnœa, palpitation, and sickness. Three months previously she caught cold and had been worse since. Dropsy began in the legs, and was next noticed in the face, then in the belly.

On admission, the face was very dusky and bloated. Extremities cold. No clubbing of nose or finger-ends. Jugular veins slightly full. Tongue furred. Pulse barely perceptible, irregular, 128 per minute. The heart's impulse was diffused, maximum distinctness of apex-beat in left fifth space in nipple-line, but impulse felt indistinctly an inch outside this point. Well-marked præsystolic thrill over left ventricle. Præcordial dulness began at fourth rib and extended to right beyond middle line of sternum. At the apex a præsystolic murmur was heard, followed by a systolic one not traceable into axilla. Over the tricuspid area a systolic murmur was also heard. The pulmonary second sound was accentuated. Hepatic dulness began at fourth right interspace, and the lower edge of the liver was felt indistinctly about 2 inches above umbilicus. Spleen not palpable. Over the lungs was some impairment of resonance at the base, with feeble respiratory murmur and crepitation. The urine was of sp. gr. 1025, deposited urates, and contained one fourth of albumen. The temperature was 98·2°. The legs were cold, very œdematous and dusky, with scattered purpuric spots.

She was ordered light diet, with 2 oz. of brandy; tincture of cannabis indica and spirit of chloroform, and compound jalap powder were given as medicines. After four days tinc-

ture of digitalis was prescribed in ten-minim doses with spirit of nitrous ether. Subsequently iron and digitalis were given. The heart's action became more regular, and the dropsy diminished. The albumen was reduced to one tenth. Early in July she was able to sit up in the afternoon. The physical signs remained much the same in respect of all the organs. A diastolic murmur was now heard at the left of the sternum in the area of the pulmonary artery. The præsystolic murmur was audible, followed by sudden first sound passing into systolic murmur, the pulse being 88 in the minute. The second sound was clear over the aortic area. The systolic murmur in the tricuspid area became more distinct, and was distinguishable from that in the mitral area. The albumen disappeared from the urine. The temperature remained subnormal often being as low as 96·4°. On July 27 and 28 it rose to 101° and 100·4° without any noticeable symptoms.

When the cardiac action had calmed down, a second centre of slightly marked præsystolic thrill was detected over the third and fourth left cartilages, and on auscultation a præsystolic murmur was faintly heard. This I was disposed to attribute to stenosis of the tricuspid valve. It was readily distinguishable from the diastolic murmur in the pulmonary arterial area, which was also conducted down the sternum. I therefore headed the board "Morbus cordis, mitral and tricuspid stenosis, pulmonary reflux." With rest and treatment the patient was much relieved, and left the hospital on August 14.

On September 8 she was readmitted, having speedily relapsed, and begun again to suffer from dyspnœa, cough, headache, and sleeplessness. Four days previously she coughed up some blood. The cardiac physical signs were unaltered. The pulse was 118, small, and regular, and the temperature subnormal. The urine contained a cloud of albumen. Digitalis was again given with nux vomica and iron, and 4 oz. of brandy were ordered daily. The sleeplessness called for morphia, cannabis indica, and ether. She did not respond to treatment. The urine became more albuminous, the pulse irregular. Later on the symptoms were aggravated by the prevalent fogs. On October 29 the temperature rose to 102°, and there was increase of dyspnœa with cough and muco-purulent sputa. There were signs of congestion at the base of the left lung, and increased hepatic engorgement with some jaundice. Blue pill, digitalis, and squill pill was now given each night, and for the gastro-enteric

catarrh tartrate of sodium draught was ordered. The sputa became bloody, apparently indicating pulmonary embolism. At this time the signs of reflux through the pulmonary artery disappeared. Death occurred on November 8. The body was examined next day.

The heart extended considerably across the mesial line. The apex was 2 inches beyond the line of left nipple. The heart was much enlarged and weighed 24 oz. Wall of right ventricle firm and somewhat thickened. Cavity of right ventricle much dilated, especially at the conus arteriosus. Tricuspid orifice markedly stenosed, barely admitting two fingers. Edges of the valve thickened and somewhat roughened. Pulmonary valves not definitely diseased. Corpora Arantii apparently larger than usual. Left auricle much dilated and rather thick. Mitral orifice, "button-hole" contraction, barely admitting little finger. Chordæ tendineæ much thickened. Left ventricle decidedly hypertrophied. Aortic valves doubtfully thickened. Pulmonary artery dilated. No ante-mortem clots.

On the right lung was some recent inflammatory lymph at the base in front. In right lower lobe a patch of consolidation, size of a small orange, red hepatization. Rest of lung much congested. Left lung much engorged. No infarcts found in branches of pulmonary artery. A good deal of clear fluid in abdomen. Liver "nutmeg," with increased growth of connective tissue. Spleen and kidneys small, hard, and engorged.

The points of interest in the case relate to its etiology, and to two of the physical signs observed during life. The frequent occurrence of mitral stenosis in women without history of rheumatic antecedents is well recognised. The mitral affection may have supervened on scarlet fever. In many of these cases, however, I believe that rheumatic valvulitis is often the starting-point, the articular symptoms being slight and unrecognised in early life. The suspicion of tricuspid stenosis was verified by the disease of that valve discovered after death. Had life been prolonged, it is not unlikely that further involvement of this valve would have occurred. Tricuspid stenosis is hardly recognised during life; when it occurs there is usually accompanying mitral stenosis. The pulmonary reflux must have been due to the dilatation of the pulmonary artery, which entailed insufficiency of its valves. When the blood-pressure became low this sign disappeared.

It is open to comment that signs of increased pulmonary

arterial pressure existed when tricuspid reflux was established. This is hardly to be expected. I have, however, on several occasions noted accentuation of pulmonary second sound, together with signs of tricuspid regurgitation and mitral disease.

(The heart was exhibited.)

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