Case : a large pedunculated thrombus hanging from the wall of the left auricle, associated with mitral stenosis / by John Hay.

With Johnston's Compliments.

(8)

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Case.—A large Pedunculated Thrombus hanging from the wall of the left Auricle, associated with Mitral Stenosis. By Dr JOHN HAY. (Illustrated by Pathological and Microscopical Specimens, and by means of Water-Colour Drawings and Lantern Slides.)

THE case which I bring before this meeting possesses the following points of interest:—

(1) A large pedunculated thrombus hanging from the septum ovale into the left auricle.

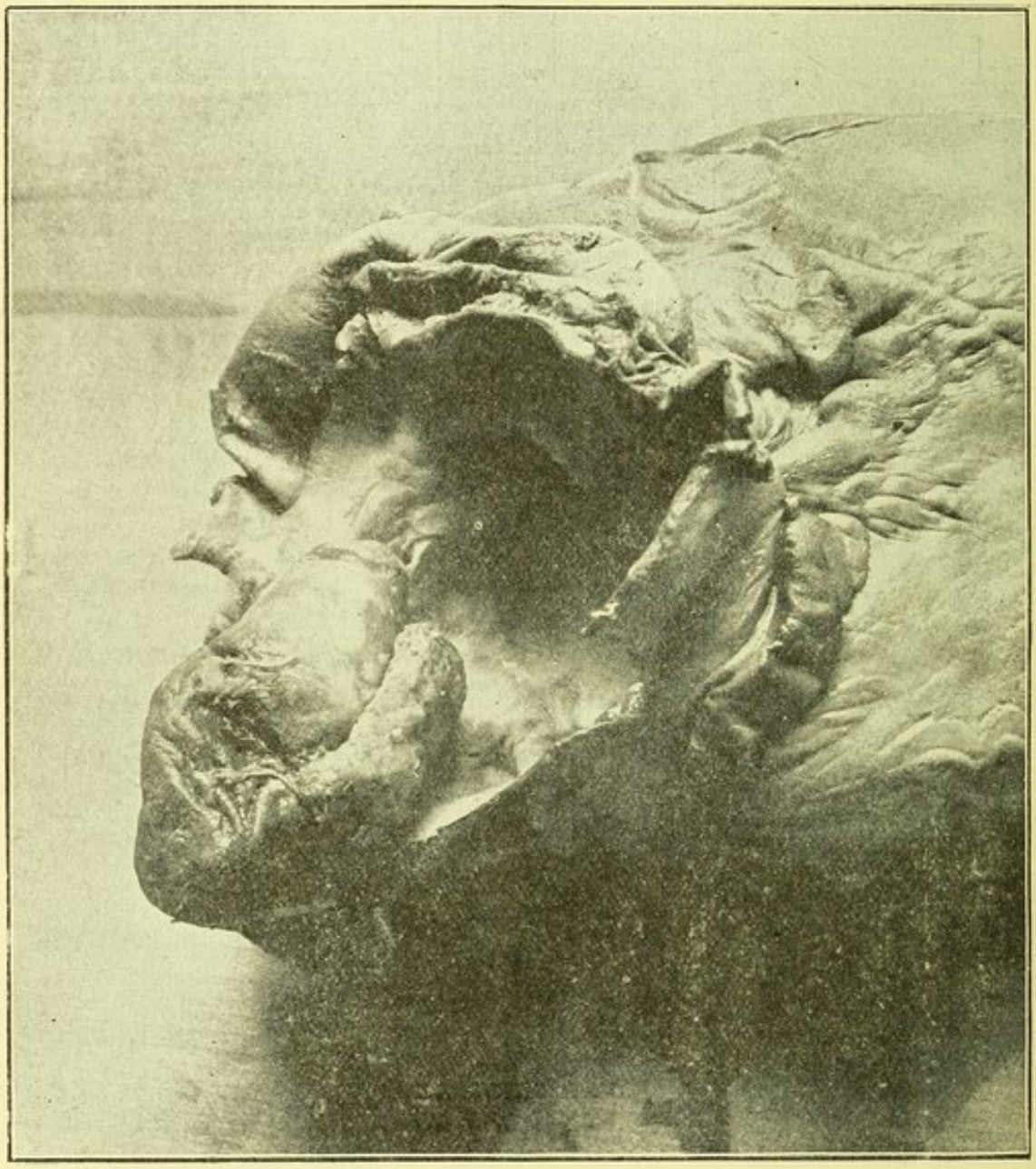
(2) Extreme stenosis of the mitral orifice.

(3) The existence of a presystolic pulsation to the right of the middle line, caused by the contraction of the right auricle.

I. *Clinical History.*

Emma S., æt. 17, was admitted into Mill Road Infirmary on 20th March 1899, *via* condition of cardiac failure. She was very small for her age—looking like a child of 12 rather

than 17. There was no history of rheumatism, chorea, or scarlet fever. Her health had been good until four years before admission, when she began to complain of shortness of breath and pain over the heart.



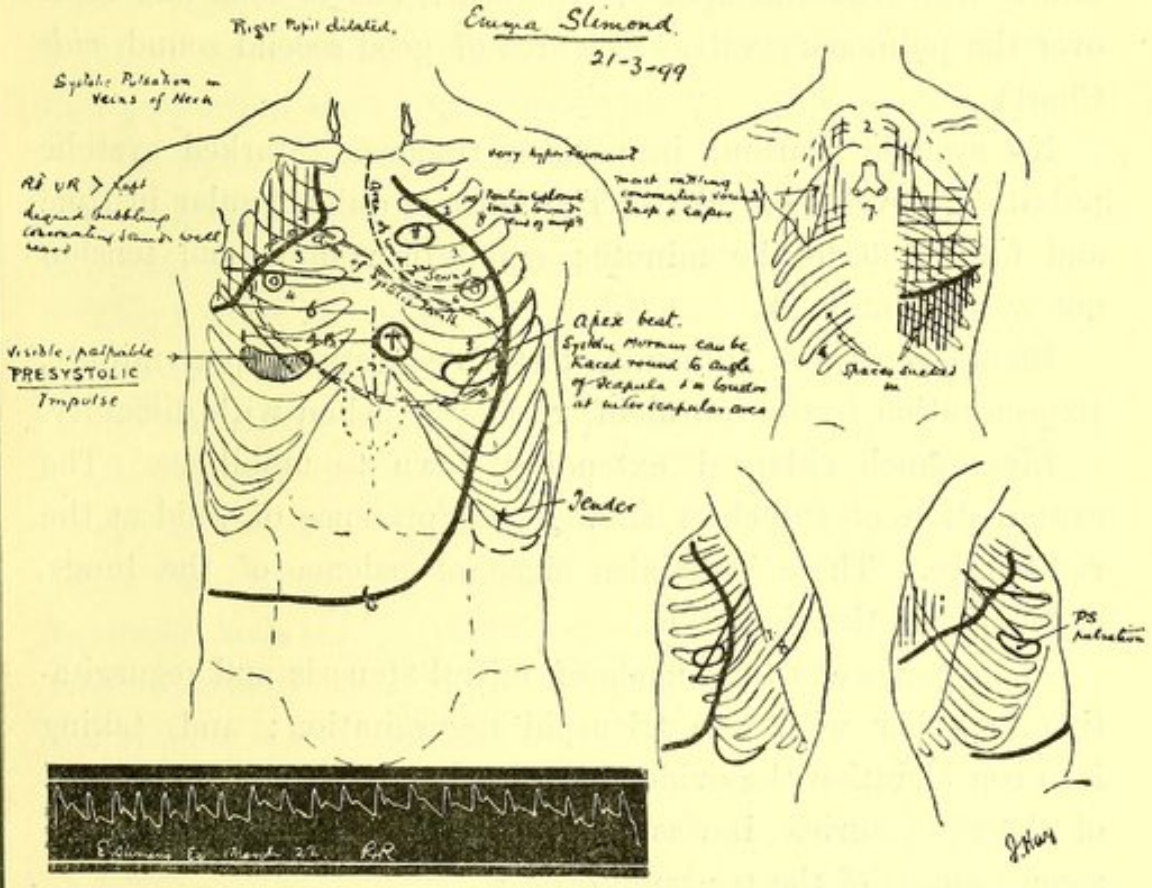
In the spring of 1898 she was an in-patient for a short period, suffering from dyspnœa and some œdema, the diagnosis then made being mitral and tricuspid regurgitation. She was discharged improved, but gradually became worse again, and previous to the last re-admission had an attack of hæmoptysis.

On admission she was cyanosed, especially about the ears,

and had to be propped up in bed. The legs were œdematous. Her condition was too serious to allow of a careful examination, but by the next day she had recovered somewhat, and the following notes were made:—

21st March 1899.—Patient propped up in bed, slight yellowing of skin and conjunctivæ, ears, lips, and cheeks congested and dusky, face otherwise pale.

Feet and legs bluish, congested and swollen, pitting on



pressure. On palpating the chest systolic thrill is felt over the præcordium, more especially over and around the apex beat, though it is also felt to the right of the sternum, over the second and third interspaces.

There is general systolic heaving of the sternum and epigastric pulsation, the apex beat being well defined in the fifth space, some four inches to the left of the middle line, whilst in the right fifth space is a well-marked pulsation, the outer limit of which is $4\frac{1}{2}$ inches from the mid-sternum. This pulsation is definitely presystolic in time.

The præcordial dulness is greatly increased, measuring 11 inches in the transverse diameter (*vide* Chart).

On auscultating, a rough, grating, harsh blowing systolic murmur, with squeaks and wheezings associated with it, is heard all over the front of the chest. This murmur is loudest over the apex beat, and is conducted into the axilla. It is also rather loud over the second left space.

No first sound is audible. The second sound is only very faintly heard at the apex of the heart, but is loud and clear over the pulmonary valves (for area of good second sound, *vide* Chart).

No systolic murmur in neck or trachea. Marked systolic pulsation in veins of neck. The pulse is quite regular in time and force, 120 to the minute; small in volume, and tension not well maintained.

Breathing rather difficult, forty respirations per minute. Expectoration frothy, purulent, viscid; expelled with difficulty.

Liver much enlarged, extending down to umbilicus. The examination of the chest showed the presence of fluid at the right base. There were also signs of œdema of the lungs, especially on the right side.

A diagnosis was then made of mitral stenosis and regurgitation, together with free tricuspid regurgitation; and, taking into consideration the evident great dilatation and hypertrophy of the right auricle, it was suspected that there might also be some stenosis of the tricuspid orifice.

The question of fluid in the pericardial sac was also discussed; but as the presystolic pulsation and the apex beat were both well to the outer limits of the dulness, it was thought that there was probably no great pericardial effusion.

Under treatment she improved, and on the 26th March xvii. oz. of clear serous fluid were withdrawn from the right pleural cavity, after which her breathing seemed easier.

In the evening of the same day she complained of pain all over her body, and half an hour later suddenly died.

II. *Post-mortem Examination.*

Owing to the objections of the relatives, as complete a post-mortem examination as one would have liked could not be made.

The pleural cavity on the left side normal; on the right there were old adhesions at the apex of the lung and along portion of the anterior borders.

The right pleural cavity contained a pint of fluid.

The lung substance was tough and firm, the left lobes dry, and presenting a recent embolism in the lower lobe.

The right lobes rather œdematous, and the anterior border of the middle and lower lobes was retracted from the heart, exposing most of the anterior surface of the right auricle.

There were no signs of tubercle, old or recent. Liver enlarged and nutmeg.

Spleen slightly enlarged and fibroid.

Pericardium contained 4 ozs. of clear fluid, which was found mostly at the back of the heart and around the great vessels. There were adhesions between the anterior surface of the right ventricle and the pericardium, some long and fine. These might partially account for the peculiar accompaniments to the systolic murmurs which were heard.

Description of Heart.—The heart weighed 21 ozs., about twice the size of the heart of an adult female. The right auricle extended beyond the right nipple. It lay directly in contact with that part of the chest wall which during life had presented a presystolic pulsation, the lung, as I mentioned before, having shrunk away from it. Its cavity was enormously dilated, and easily accommodated my closed fist; the wall was also considerably hypertrophied, more especially the auricular appendix, which showed patches of old pericarditis.

The tricuspid valve easily admitted my four fingers and thumb—the cusps being healthy, except for slight thickening.

The right ventricle was extremely dilated, and apparently four times the capacity of the left. Wall hypertrophied.

Definite dilatation of the conus arteriosus. Two moderator bands present. Endocardium mottled, especially round the orifice of the pulmonary artery.

The pulmonary artery showed yellow patches of degeneration scattered over its surface.

The valves normal.

The right auricle and ventricle formed much the greater bulk of the heart, the apex being formed entirely of right ventricle.

The left auricle was distinctly dilated, but not excessively so; wall hypertrophied; the appendix being much enlarged, and extending well round to the front. Before opening the auricle I felt something rolling about in it, and occupying a considerable portion of its cavity; this was found to be a large pedunculate thrombus, shortly to be described.

The endocardium of the auricle was much thickened and striated, especially round the orifice of the appendix.

The mitral orifice was very stenosed, the cusps being thickened and fibroid; the orifice just admitted the tip of my little finger. It was crescentic in shape. No vegetations on it.

From the ventricle, the great thickening and fibrosis of the cusps is very evident; also the involvement of the chordæ, and the fibrosis of the musculi papillares. Left ventricle small; wall firm, and good consistence. Opaque streakings of the endocardium, especially in the neighbourhood of the aortic valves.

The aortic valves normal and competent. Small patch of fatty degeneration above the opening of the left coronary.

The calibre of the aorta was less than normal.

III. *Description of the Thrombus.*

1. *Macroscopical.*—It is now rather difficult to form any adequate conception of its appearance when fresh, the specimen having lost its colour and being shrunken; however, the coloured drawing which I pass round will give an idea of its former colour and size.

It is attached by a slender but tough pedicle—$\frac{1}{4}$ of an inch long and the thickness of an ordinary pin—to the septum ovale. It is roughly pear-shaped. Length, $1\frac{3}{4}$ inches; breadth, 1 inch.

It is evidently composed of two parts—

(1) A solid portion.

(2) A saccular portion.

(1) The solid mass is attached to the wall of the auricle by the fine pedicle. It is smooth and glistening; tough, firm, and elastic to the touch, and of a light yellow colour, with several whitish patches on its surface.

(2) The saccular portion is large and irregular, attached to and covering the end of the solid portion.

This sac is of a reddish colour, its walls being irregular and easily torn, $\frac{1}{16}$ to $\frac{1}{32}$ of an inch thick. It is prolonged on to the solid part as a kind of appendix.

This sac is full of a soft pulpy material—a pale reddish-brown, grumous, puriform fluid. On making a longitudinal section of the thrombus, one finds that the solid half is formed of tough laminated material, the outer layers being the harder, though it is solid throughout.

From its position and size it is evident that the free end of the thrombus would lie over the stenosed mitral valve.

This would undoubtedly greatly increase the obstruction to the onward flow of the blood, and probably accounts for the excessive dilatation of the right auricle and ventricle.

It is also possible that the thrombus blocking the stenosed mitral orifice was the cause of the sudden death.[1]

2. *Microscopical examination.*—The whole heart, together with the thrombus, had been preserved in formaline 5 p.c. solution for six months before I cut any sections. Portions were then embedded and stained, the sections under the microscope being stained with Ehrlich's hæmatoxylin.

Examination shows the thrombus to be composed of structure-

[1] D. Walshe, *Diseases of the Heart*, 1873 ed., 357, etc. Wickham Legg, *Pathological Trans.*, xxix. p. 49.

less laminated layers of fibrin, enclosing small round cells (leucocytes), blood pigment, and large fusiform cells with elongated nuclei.

The main bulk is not organised.

One finds a granular, somewhat reticulate substance, with darkly stained leucocytes scattered throughout. These are more frequent in the less dense portions.

In some places, especially towards the surface, one meets with dark granular masses of pigment, which I take to be broken down red-blood corpuscles included in the fibrin.

The leucocytes are more numerous towards the surface, and appear to hang on to the sides of the spaces formed in the fibrin.

Towards the surface the matrix becomes denser and less granular, and on the surface there are endothelial cells. These appear to cover the whole surface of the thrombus.

In patches, one meets with portions of the thrombus containing a number of cells. These cells have large oval nuclei, and when one sees the outline of the cell it is found to be fusiform.

These organised patches lie just beneath the surface. The section under the microscope is such a patch. The tissue there is apparently organised. One strand is seen running through the centre of a mass of pigment; in this strand there are several very large nuclei.

The question necessarily arises as to the character and origin of these cells.

That they are endothelial seems to me to be the most reasonable explanation, and that they have spread over the surface of the thrombus from the endocardium of the auricle.

One knows what an important share the endothelium of a vessel takes in the organisation of a thrombus in its lumen, and it seems reasonable to suppose that the endocardial cells should not be less active.

That these cells should be derived from the white cells of the blood—even admitting the possibility—seems unlikely,

when one remembers that undoubted white cells are found wandering about throughout the whole of thrombus, and yet these larger fusiform cells are only to be found at or near the surface, the very place one would expect cells of endothelial origin to be.

In a case recorded by Sheridan Delépine,[1] he states that in the calcified thrombus or cardiolith which he examined, the organisation was due to the endothelial layers, and that he could find no sign of any vascularisation; whilst Voelcker[2] showed sections of a cardiac thrombus in which he demonstrated vessels running throughout the substance of the mass, the vessels possessing no muscular fibres, but having an endothelial lining of oval cells.

A point of interest here is that Voelcker's case was most highly organised in the centre, whilst Delépine's case and mine were organised towards the surface.

After a careful examination, I was unable to satisfy myself that there were any vessels to be made out in this thrombus.

IV. *Remarks.*

When one takes into consideration the density of the thrombus, the fineness of its laminæ, the patches of organisation, and more especially its size, one must come to the conclusion that in all probability it had existed for some considerable time.

The existence of an endothelial covering excludes the possibility of any recent deposit of fibrin, however any roughening of the endothelial surface might have started the fresh deposition of fibrin, and this probably occurred at the distal extremity, where we find the large saccular portion. This was probably laid down quickly as a solid mass, and then its central portion softened and broke down, owing to fatty degeneration

[1] "Description of a Cardiolith," Sheridan Delépine, B.Sc., M.B., *Trans. Path. Soc. Lond.*, vol. xli. p. 43.

[2] "Intra-auricular Cardiac Polyp," Voelcker, *Path. Soc. Trans.*, xliv. p. 31.

and granular disintegration of its parts,[1] so that we found, post-mortem, a thin-walled cyst, full of this broken-down clot, attached to and enveloping the rounded end of a much older, partially organised, laminated thrombus.

Briefly.—The presence of thrombi in the heart depends on two circumstances.

Firstly.—Some roughening or irregularity of the surface of the heart or valves, as endocarditis.

Secondly.—Retardation of the blood-flow, such as is found in the nooks and crannies of the heart's cavities, and due to the various stenoses.

The proportion of the fibrin elements in the blood in certain conditions must not be neglected as a predisposing factor.

Of the three recognised varieties of thrombi—warty, globular, and polypoid—the polypoid are of great rarity, and there are very few of any size recorded.[2] The polypoid or pedunculated usually start on the appendix of an auricle. If the stem or pedicle gets broken through, they then lie free in the auricle, and form a globular or ball thrombus.[3] These are beautifully smooth, and almost invariably associated with mitral stenosis. It is easy to imagine how easily the small fine pedicle of the specimen under discussion might have snapped, allowing the thrombus to lie free in the auricle.

Large pedunculated thrombi arising from the auricular septum are still rarer, especially of the size of the specimen on exhibition to-night. Perhaps the most interesting one recorded is that by Professor Gairdner,[4] where the thrombus hung down on to the tricuspid orifice, giving rise to the signs of tricuspid stenosis, this case being, I believe, one of the first, if not the first, in which the value in diagnosis of a tricuspid presystolic murmur was recognised.

I find another intracardiac thrombus recorded by Ewart and

[1] Weichselbaum, *Elements of Pathological Histology*, 1895, p. 208. Delafield and Prudden, *Pathological Anatomy and Histology*, 1897, p. 501.

[2] Coats, *Manual of Pathology*, ed. iii., p. 425.

[3] *A Handbook of Pathological Anatomy*, Prof. Boyce, Dr Abram.

[4] Gairdner's *Clinical Medicine*, 1862. *Edin. Hosp. Reports*, 1893.

Rolleston,[1] in which the thrombus arising from the fossa ovalis projected through the mitral orifice, and gave rise to the signs of mitral stenosis.

Rindfleish[2] describes a similar case. Voelcker[3] records a case already mentioned similar to mine, in which the thrombus was attached to the septum ovale, and had the appearance of a champagne cork. Its attachment to the septum ovale was broad, not slender, and the polyp was especially interesting in that it showed signs of vascularisation. The cardiolith described by Sheridan Delépine has been referred to. Polypoid thrombi are also mentioned by Allan Burns,[4] Laennec, Hasse, and Rokitansky.

Viewing the case more from the clinical than the pathological point of view, the presence of the visible auricular pulsation is of extreme interest.

Discussion has in the past raged round the question as to whether the left auricle ever reaches the anterior wall of the thorax, or gives rise to a presystolic pulsation. Only so lately as 1897, George W. Balfour, M.D., of Edinburgh,[5] stated that it did so "beyond all possibility of doubt," whilst G. A. Gibson,[6] William Russell,[7] and some others are equally emphatic in its denial. Sansom[8] states definitely that in children suffering from mitral stenosis, the left auricle does rarely impinge against the thoracic parietes, giving rise to a pulsation, presystolic in time, which can be seen and felt in the 2nd and 3rd left interspaces close to the sternum.

He also draws attention to the wavy pulsation one occasionally feels in placing the hand over the præcordium, and is of

[1] "Intracardiac Thrombus projecting through Mitral Orifice," Ewart and Rolleston, *Clinical Soc. Trans. Lond.*, vol. xxx. p. 190.

[2] *Pathological Histology, New Sydenham Soc.*, 1872, vol. i. p. 295.

[3] "Intra-auricular Cardiac Polyp," Voelcker, *Path. Soc. Trans.*, xliv. p. 31.

[4] *Diseases of Heart*, 1809.

[5] G. W. Balfour, M.D., *Journal of Pathol. and Bacteriology*, vol. iv., 1897, p. 75.

[6] G. A. Gibson, M.D., *Journal of Pathol. and Bacteriology*, vol. iii., 1894, p. 34.

[7] Wm. Russell, M.D., *Investigations into some Morbid Cardiac Conditions*, Edinburgh, 1886, p. 41.

[8] *Diagnosis of Diseases of Heart and Thoracic Aorta*, 1892.

opinion that this is caused by the contraction of an enlarged right auricle, followed by the contraction of the right and left ventricles.

In the present case, however, the pulsation is unique, in that it is definite, localised, and presystolic; the conditions being very favourable to its production, and as follows:—

Firstly, An exceptionally hypertrophied and dilated right auricle, lying well to the right of the sternum, and therefore impinging against softer and more yielding intercostal tissues.

Secondly, The retraction of the lung from the front of the pericardial sac left the heart directly in contact with the anterior thoracic wall, and in a most favourable position for the transmission of its pulsations.

This pulsation could in no way be described as a 'wavy movement,' but was a definite localised impulse, extending over an area of an inch and a half in its long diameter, and proved both by clinical methods and post-mortem examination to have been presystolic in rhythm, and produced by the right auricle.

Professor Balfour of Edinburgh, who has been rather interested in the case, suggests that it is also an example of hypoplasia of the arterial system, on account of the small calibre of the aorta and the diminutive size of the patient.

In conclusion, I wish to express my thanks to Dr Nathan Raw for permission to publish this case.