Clinical lectures on circulatory affections. Lecture 1. Persistence of the arterial duct and its diagnosis / by G. A. Gibson.

CLINICAL LECTURES ON CIRCULATORY AFFECTIONS.—LECTURE I. PERSISTENCE OF THE ARTERIAL DUCT AND ITS DIAGNOSIS.

By G. A. GIBSON, M.D., D.Sc.

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CLINICAL LECTURES ON CIRCULATORY AFFECTIONS —LECTURE I. PERSISTENCE OF THE ARTERIAL DUCT AND ITS DIAGNOSIS.

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(Plate I.)

Congenital affections of the heart do not lend themselves in general to ordinary clinical teaching. Most of the lesions are so complex in their nature that the resulting symptoms are difficult of explanation. They are without doubt of the deepest interest to a speculative mind, but for you in your early student days they are but a waste of time, which may be much more usefully employed otherwise. There is, nevertheless, amongst the many different congenital lesions of the heart one particular variety of malformation so easy of detection as to be instructive even for the junior student of clinical medicine. This is persistence of the arterial duct, which will form the subject of lecture to-day, in order to give me the opportunity of formulating its diagnosis, as well as to provide me with an occasion of replying to certain strictures recently passed on me.

The communication between the aorta and pulmonary artery ought to be, and usually is, entirely obliterated within eight days at latest from the date of birth. From causes into which it would be at present as idle as fruitless to inquire, this channel occasionally remains permanently open, maintaining a communication between the two great arteries during the lifetime of the individual.

This malformation is easily recognised during life by the results of physical examination, yet the statement of Walshe remains true to this day: "Clinical histories of open arterial

duct are few and far between."[1] Walshe himself seems to think that symptoms of cyanosis, with enlargement of the right side of the heart, absence of apical murmur, and the existence of a long diastolic or double murmur, would be the characteristic clinical features of the disease. The cases to which he refers were all complex, as have been most of those since published, amongst which that of Foulis[2] may be cited as an illustrative example. But little light is thrown upon the condition in any of our standard works, and the nearest approach to the truth in diagnosis lies in the remark of MacCallum,[3] that there is no cyanosis and sometimes a systolic murmur in the second left intercostal space.

In consequence of the higher pressure of the blood in the aorta, as compared with the pulmonary artery, there must be a current from the former to the latter, and this stream will be almost, if not quite, continuous. It will of necessity flow with its greatest velocity during, and immediately after, the ventricular systole, when the aortic blood pressure is at its highest; from that phase it will gradually become less swift, as the pressures in the two great vessels approximate more nearly to each other during the period of repose. It must therefore be expected that, as evidence of patent ductus arteriosus, there will be a long murmur, beginning a little after the commencement of the first sound, filling up the short pause, and continuing beyond the second sound. The murmur may be, in fact it almost invariably is, accompanied by a well-marked thrill. The localisation of murmur as well as of vibration will naturally be in the second or third left intercostal space, just outside of or inferior to the conventional pulmonary area.

Five cases leading to the diagnosis of this affection have been under my care during the last few years. Two of them have not been recorded; two others have been briefly narrated;[4] the fifth is the case to be considered to-day. From a careful study of these cases, the diagnosis has come to seem in my eyes almost as exact as the solution of a mathematical problem. The rhythm of the murmur is laid down definitely, and figured diagrammatically in my work on the Heart,[5] from which this figure is borrowed, and the assemblage of rational symptoms and physical signs is also described in another part of the same work.[6] In the most recent paper on congenital heart disease,[7] however, Thomson has not mentioned the very characteristic rhythm of the murmur, which

[1] Walshe, "Diseases of the Heart and Great Vessels," London, 1873, 4th ed., p. 565.

[2] Foulis, *Edin. Med. Journ.*, 1884, vol. xxx. p. 17.

[3] MacCallum, *Johns Hopkins Hosp. Bull.*, Baltimore, 1900, vol. xi. p. 70.

[4] Gibson, "Diseases of the Heart and Aorta," Edinburgh and London, 1898, pp. 310, 312.

[5] *Ibid.*, p. 161.

[6] *Ibid.*, p. 303.

[7] *Edin. Hosp. Rep.*, 1900, vol. vi. p. 57.

might have been expected in two of his nine cases, seeing that in these two there was no lesion beyond the persistent duct, save open foramen ovale, which gives no definite physical signs. It is but right to say that persistence of the arterial duct is often associated with other congenital lesions, such as obstruction and incompetence at the pulmonary orifice, imperfection of the septum

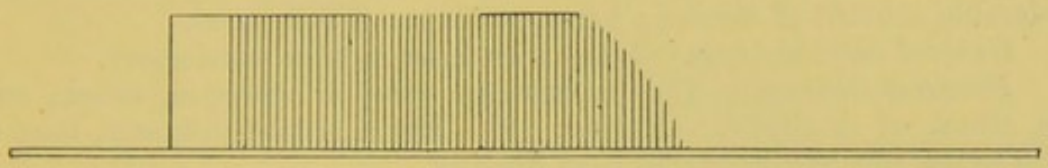


Fig. 1.—Continuous systolic and diastolic murmur in patent ductus arteriosus.

between the ventricles, patency of the foramen ovale, and even transposition of the aorta and pulmonary artery. In complex cases of this kind, to which category that of Foulis[1] belongs, the diagnosis is necessarily obscure. You will permit me, nevertheless, to reiterate that the recognition of patent arterial duct in itself is of easy and certain attainment.

In reviewing the work to which reference has been made, one of my critics, whose review bears the initials "D. L. E.," while expressing in the main a generous appreciation of my labours, has asserted that some of my views are of doubtful value.[2] He states, for example, that "wise and conservative authors are wholly decided in their teaching that dependence upon the characters of murmurs alone is usually a very shaky method of establishing a diagnosis." Again he says: "It is hard to convince one now that any localization or transmission of a murmur may be said to be pathognomonic, particularly of lesions so difficult to diagnose as is tricuspid obstruction. To instruct one's readers to depend very largely upon murmurs in making a diagnosis, seems narrow and somewhat antiquated. Other signs of the mechanical effects of a lesion must be found before any diagnosis can be positively made." Yet once more he says, after some appreciative remarks of my work as a whole: "It tends, however, toward teaching one to diagnose cardiac conditions rather from the standpoint of clinical observation, than from a consideration of the pathological changes in the heart and the resulting mechanical disturbances, and one feels that he has gained interesting ideas from reading the work rather than that he has been put upon a solid basis in formulating an exact diagnosis." How far the case now to be considered bears out my confidence or my reviewer's criticisms, you will be able to decide for yourselves. The following are the notes, slightly abridged, from the case-book:—

Mrs. B., æt. 31, was sent to me, 28th January 1900, on account of weakness and uneasiness in the chest.

[1] *Loc. cit.*

[2] *Amer. Jour. Med. Sc.*, Phila., 1900, vol. cxix. p. 451.

History of Patient.—*Family tendencies.*—As the father and mother of the patient are in good health, while her brother, who was the only other child of the parents, is robust, there seems no hereditary liability to disease. She has four children, who are all strong. One child died of convulsions when a baby.

Dietetic habits.—Mrs. B. has always been able to have plenty of good food. She does not take much tea, but seems to have taken a considerable amount of alcohol; her appetite is not very good.

General surroundings.—These have always been satisfactory.

Previous illnesses.—The patient had measles, whooping-cough, and an attack of smallpox, as a child. She has also had influenza four or five times, but has otherwise been healthy.

Present illness.—In June the patient was confined, and she never picked up her strength properly since. She went on, no doubt, doing her ordinary work, though she always felt tired and "done," and thought this would pass off. On Saturday, 20th January, she felt she would have to give in, and kept her bed nearly all Sunday; on Monday, as she felt no better, she went to see Dr. L. Thomson, who ordered her to take a complete rest. As she did not improve at all rapidly, Dr. Thomson recommended her to come to my ward, and she was therefore admitted on 28th January.

Condition on Admission.—*General facts.*—The patient is a thin, sallow woman, whose face is much pitted by smallpox. She lies with her shoulders propped up on the pillows, as she feels choked if she lies down in bed. There is no clubbing of the fingers, and her lips have a very faint bluish colour. Her weight is 6 st. 13½ lb.; about a stone less than in July 1899. The temperature is subnormal.

Alimentary system.—The lips are dry and cracked; the teeth are very bad; the gums are somewhat spongy; the tongue is very tremulous, with a thin brown fur down the centre, and a faint white fur at the sides; the mouth is apt to be very dry, with a bad taste in the morning; the fauces are somewhat congested. The appetite is not very good, and there is much thirst. The patient is very much troubled with flatulence, and the bowels are inclined to be constipated. The abdomen is not very prominent, and there are no well-marked *striæ gravidarum.* The liver extends from the fourth rib to the costal margin in the mammary line. The greater curvature of the stomach is found to be midway between the xiphoid cartilage and the umbilicus.

Hæmopoietic system.—There are no enlarged glands, and the spleen is of normal size. There are 4,100,000 hæmocytes per c.m., and the quantity of hæmoglobin is 60 per cent.

Circulatory system.—The patient complains of a pain in the region of the præcordia, which began about ten days before admission; beginning a little to the inner side of the left nipple, it radiates downwards and outwards. She has always been subject to palpitation on exertion or excitement. She does not complain of breathlessness. There is slight bulging of the second, third, and fourth left costal cartilages, especially the second, close to the sternum. Diffuse pulsation is visible over the whole præcordia. The apex beat is seen in the fifth left intercostal space as far out as 4½ in. from midsternum. It is very diffuse. There is slight venous pulsation in the supraclavicular fossæ, as well as

in the suprasternal notch. The maximum intensity of the apex beat is found on palpation to be 3½ in. from midsternum. There is a thrill over the base of the heart. It is felt with its greatest intensity in the third left intercostal space, 2½ in. from midsternum. It begins shortly after

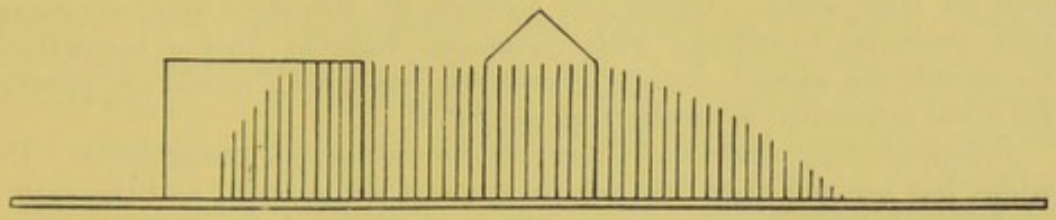


FIG. 2.—Continuous systolic and diastolic murmur, with accentuation of the second sound, in patent ductus arteriosus.

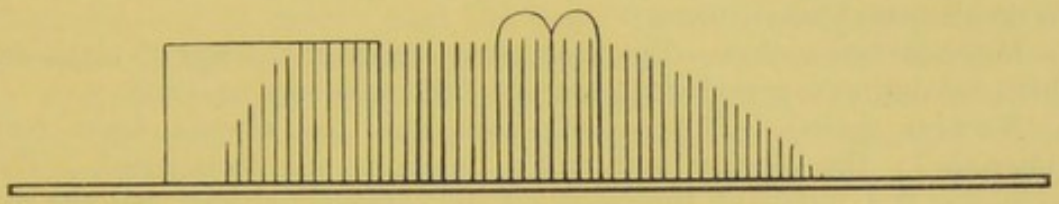


FIG. 3.—Continuous systolic and diastolic murmur, with doubling of the second sound, in patent ductus arteriosus.

the apical impulse, and continues almost to the next apex beat. During its continuance the shock of the pulmonary cusps can be very distinctly felt. The upper border of the heart is at the border of the third rib; the right border is 1½ in. from midsternum; and the left border is 4½ in. from midsternum.

At the base of the heart an almost continuous rushing murmur is heard, beginning shortly after the first sound, which is quite clear at its commencement, and continuing almost to the beginning of the next first sound. It is best heard in the third left interspace about 2½ in. from midsternum, but is heard all over the base of the heart. The second sound is heard distinctly in the middle of this murmur; it is distinctly accentuated, and sometimes doubled. The first sound in the aortic area is feebly heard, but the second sound is distinct. The murmur is not so distinctly heard as in the other areas. The first sound in the mitral area can be clearly heard. The murmur is conducted from the base of the heart, beginning before the second sound, and continuing for some time after it. Both sounds are distinct in the tricuspid area, but the murmur is conducted as above. The volume and pressure of the pulse are moderate; the artery walls are slightly thickened; the rate is usually about 72, and the pulsation is regular in time and equal in force.

Respiratory system.—The patient has been subject to a dry irritating cough for a great many years. It often came on when she rose in the morning or lay down at night. There was never much sputum with it, except when she took a "cold," to which she has been very subject. Sometimes the cough was so severe that she says it made her nose bleed, or the sputum was streaked with blood. The rate of the respiration is 24; it is regular, and of costo-abdominal type. The chest is

very thin; there is marked hollowing above both clavicles; both sides of the chest move equally, but the movement is somewhat limited. The vocal fremitus is everywhere normal. The percussion sound is slightly muffled at the right apex, both in front and behind. There is vesicular breathing with prolonged expiration above the clavicle and scapular spine on both sides. Below the clavicle, on the right side, somewhat harsh vesicular breathing, with occasional rhonchi during inspiration. The sounds are almost normal elsewhere, but more rhonchi are present. The vocal resonance is nowhere exaggerated.

Integumentary system.—There are no subjective phenomena. The patient has become very thin within the last six months, and the skin is dingy in colour.

Urinary system.—The quantity of the urine is from 40 to 50 oz. It is straw coloured. Its reaction is alkaline. The sp. gr. is 1015. There are no abnormal constituents.

Reproductive system.—The catamenia began at the age of eighteen. There has been no period since her last child was born in June.

Nervous system.—The patient says she has always been very "nervous"; the sensory and motor functions are unimpaired. The vasomotor and nutritive functions are not obviously altered, but she has become very thin lately, and she is very subject to cold hands and feet. As to the cerebral and mental functions, her intelligence is good; she is often troubled with headaches, but she attributes them to constipation. She is not a good sleeper.

Diagnosis.—Patent ductus arteriosus.

The rhythm of the murmur may be represented by the diagrams drawn for you upon the black-board, in the one of which the accentuation, and in the other the doubling, of the second sound in the pulmonary area are conventionally indicated.

Now, in this case, which may be taken as one capable of putting our means of physical diagnosis to the test, the only possible conclusion was that which was arrived at. With a thrill and murmur beginning a little after the first sound, and continuing until after the end of the second sound, there could be no question of a valvular lesion. We know from the investigations of Goltz and Gaule,[1] as well as other observers, that the pressure within all the cavities of the heart changes enormously with each phase of cardiac activity, being alternately positive and negative according to the systole and diastole of each chamber. It follows, therefore, that the only possible source of a murmur continuing longer than any one phase of cardiac activity, must lie in one of the great vessels, while from the anatomical position of the murmur it was perfectly obvious that it must be caused by a stream passing from the aorta to the pulmonary artery. It is possible to conceive of some such communication between the two vessels as a small aortic aneurysm opening into the pulmonary artery, and producing physical signs somewhat resembling those

[1] *Arch. f. d. ges. Physiol.*, Bonn, 1878, Bd. xvii. S. 103.

which have been laid before you. Roberts,[1] Wade,[2] and Walshe[3] have described such cases, and several similar instances have recently been described by Gairdner,[4] who has summarised the observations of these authors. Curiously enough, Walshe speaks of the *continuousness* of the murmur when systolic, or of a double murmur. Two points negatived such an idea in this case. In the first place, there was very little alteration in the condition of the arterial walls; and, in the second place, the murmur did not begin, as an ordinary systolic murmur does in a case of aneurysm of the first part of the aorta, along with the first sound, but it followed it by an appreciable interval. It seemed to me, therefore, that no other conclusion was permissible than that of permanence of the arterial duct. It was possible to go even a step farther in the diagnosis, and to decide that the case was one solely of persistent ductus arteriosus, without any attendant lesions, since there was no systolic murmur with its maximum intensity in the conventional pulmonary area which might denote an obstructive lesion at that orifice, while there was also a total absence of any diastolic murmur in that area which might indicate regurgitation. In this connection let me recall to you that there was little, if any, cyanosis, such as is usually present in congenital lesions of the heart proper. There was no increase in the number of the hæmocytes or in the quantity of the hæmoglobin, and there was no trace of clubbing of the fingers or arching of the nails. As an example of persistent arterial duct, the patient was accordingly demonstrated to my senior clinique on 1st February. She was also shown at the Saturday clinique in the University theatre on 3rd February 1900. She was further discussed by me with my friend Dr. John Thomson, who, as you all know, takes a great interest in congenital heart affections.

The patient was treated by rest and tonic remedies, under which she considerably improved in every respect, so that she was able to leave hospital on 9th March, feeling very much stronger, and weighing 5½ lbs. heavier than on admission. Upon 29th May 1900, Mrs. B. was readmitted on account of a sharp febrile attack, which followed a distinct rigor felt on the previous day, and was found, on examination, to have a temperature of 104°·6 F., a pulse rate of 130, and a respiration rate of 56. She had been delivered of a seven-months child nine days before. She was expectorating a large quantity of sputum, which was at once viscid and frothy, slightly blood-stained, and containing large numbers of diplococci. On examination of the chest, it was found that there was some muffling of the percussion sound over the whole of the right lung, with inspiratory crepitations. There could be no doubt that she

[1] Roberts, *Brit. Med. Journ.*, 1868, vol. i. p. 421.
[2] Wade, *Med.-Chir. Trans.*, London, vol. xlv. p. 211.
[3] Walshe, *op. cit.*
[4] Gairdner, *Glasgow Hosp. Rep.*, 1899, vol. ii. p. 1.

was suffering from acute pneumonia. By the following day there was dulness over most of the right side, along with bronchial breathing and pectoriloquy. The patient was treated with strophanthus and ammonium carbonate, along with alcohol freely given. As the pulse was unsatisfactory and the heart sounds feeble, she also received oxygen by inhalation and hypodermic injections of strychnine. In spite of all our endeavours, however, she steadily sank and died on 3rd June. The post mortem examination was performed the day after death by Dr. Fleming, whose report, slightly abridged, is as follows:—

PATENT DUCTUS ARTERIOSUS: LOBAR PNEUMONIA. Dr. Gibson. A. B., æt. 31, died in Ward 27, June 3, 1900. Autopsy, June 4, 1900.

External appearance.—The body was somewhat emaciated, and the muscularity was poor. There was slight lividity, with some rigidity of the arms, and marked rigidity of the legs. The face was much scarred with smallpox.

Thorax.—There was about $\frac{1}{2}$ oz. of clear straw-coloured fluid in the pericardial sac. The heart was unusually exposed on removing the sternum, owing to retraction of the anterior border of the left lung. There were fairly firm pleuritic adhesions over the whole of the left lung, and similar adhesions at the apex and lower lobe of the right lung. The heart was not weighed, as it was removed along with the other thoracic organs. The pulmonary artery was enormously distended with blood; on separating it from the aorta, and tracing the pulmonary artery to its bifurcation, the left branch was found to be united to the surface of the aortic arch just beyond the left subclavian artery by a very short ductus arteriosus. The ductus was in fact so short, that at first sight it seemed as if the left pulmonary artery was actually adherent to the aorta. The duct was patent, and the opening admitted a 12–14 bougie. The greatest transverse measurement of the heart was $4\frac{1}{2}$ in., and the greatest vertical measurement 5 in. There was a marked "milk spot" on the anterior surface of the right ventricle near the base, and one of more recent formation over the anterior surface of the left ventricle near the apex.

The left ventricle was fairly firmly contracted; the right was somewhat flaccid. The right ventricle contained a considerable quantity of clot, mostly coloured, and the pulmonary artery contained fluid blood and coloured blood clot. There was no patency of the foramen ovale, and no opening in the interventricular septum. The pulmonary cusps were healthy, as were also those of the aorta. The walls of the two vessels were almost free from atheroma. The coronary arteries were thickened and somewhat tortuous, especially the posterior. The mitral and tricuspid orifices were healthy, although the latter was considerably dilated. The left ventricle was normal as regards thickness of wall and length of cavity. The right ventricle was dilated and hypertrophied, the wall measuring $\frac{3}{16}$ in. in thickness. The heart muscle showed a distinct degree of brown atrophy, but little fatty infiltration or degeneration.

Lungs.—Right lung.—There was a layer of yellowish lymph over

the middle lobe and the lower part of the upper lobe. The greater part of the upper and the upper part of the lower lobes were consolidated. The consolidated lung was in the stage of red hepatisation of lobar pneumonia, and the lobules were well demarcated in the pneumonic area. The rest of the lung was slightly œdematous and congested. The anterior margin was somewhat emphysematous. The bronchial tubes were congested, and contained slightly blood-stained viscid mucus. *Left lung.*—This was œdematous and passively congested posteriorly. The lower part of the lower lobe was partially collapsed. At the apices of the upper lobes in both lungs there were a few small tuberculous nodules evidently quiescent, and the pleura over these nodules was slightly thickened and puckered.

Abdomen.—Liver.—Weight, 4 lbs. 12 oz. The gall bladder contained a small quantity of somewhat glairy pale yellow bile. The liver showed marked fatty infiltration, with some chronic venous congestion.

Spleen.—Weight, 7 oz. It was soft, slightly diffluent, pale in colour, acutely congested, and showed on section a few scattered hæmorrhages.

Kidneys.—Left.—Weighed 6½ oz. *Right.*—Weighed 6 oz. The capsules stripped freely. The kidneys showed a distinct but early catarrhal change in the cortex. The cortex was swollen, especially in the deeper layer, and was markedly mottled in appearance.

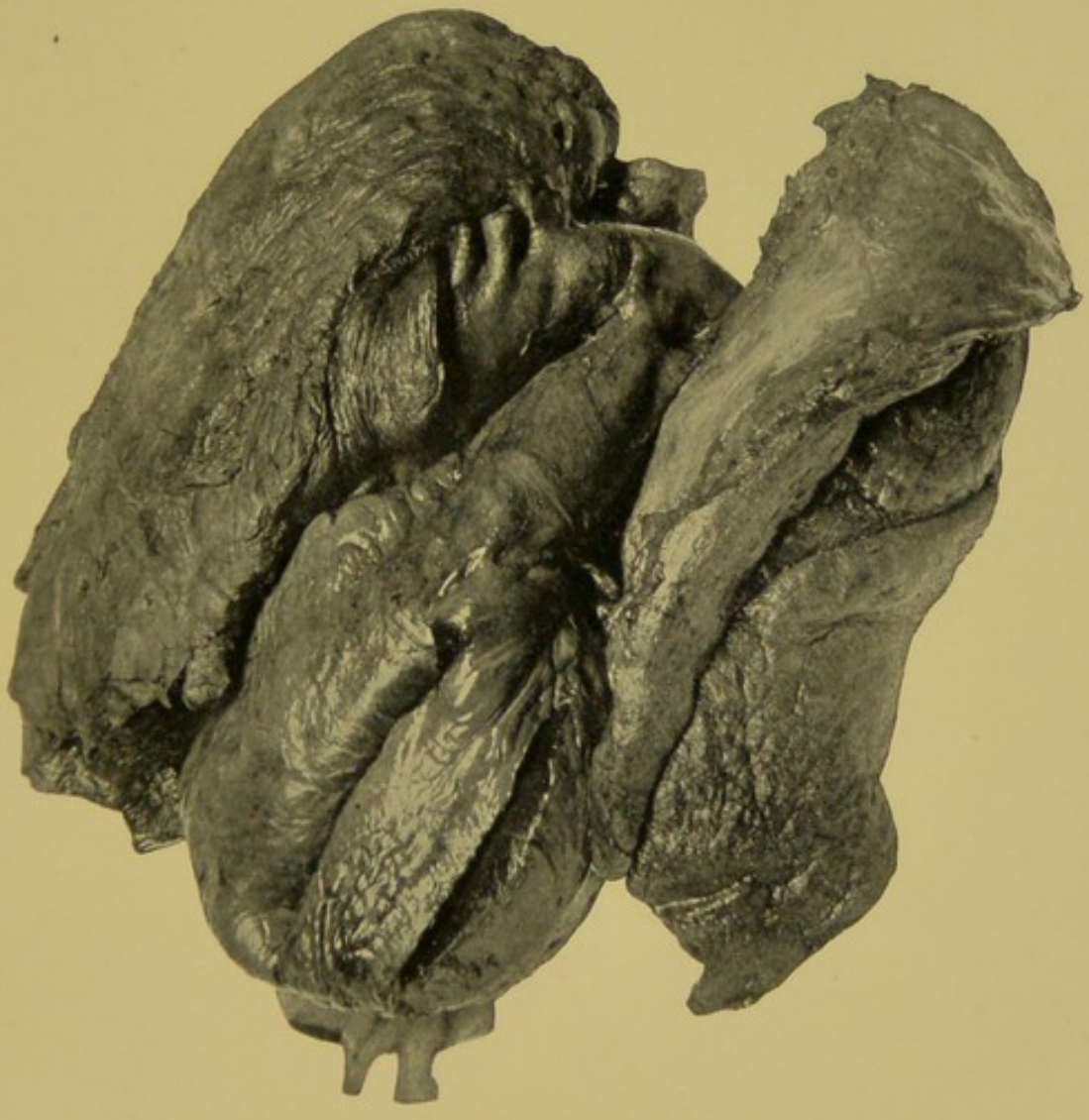
R. A. F.

The result of this post mortem examination fully sustained the confidence reposed by me in the principles of physical diagnosis. With the clinical facts such as I have laid before you, no conclusion other than that arrived at was possible; and if by any chance the diagnosis had been falsified, it would have been necessary to agree with Gairdner,[1] when he says, writing of a bit of difficult diagnosis: "We shall have to rewrite our whole cardiac diagnosis and pathology of murmurs." My belief in the principles of physical diagnosis was absolutely justified, and the facts may be held to be a complete answer to the statement of my critic: "It is hard to convince one now that any localisation or transmission of a murmur may be said to be pathognomonic." One word more on this subject. It seems to me rather difficult to understand the exact condition of mind of the reviewer when he ventured upon the remark already quoted, depreciating the diagnosis of cardiac conditions from the standpoint of clinical observation, instead of from the consideration of the pathological changes in the heart and the resulting mechanical disturbances. When there are any resulting mechanical disturbances, it may be possible to deduce from them some conclusions as to the pathological changes in the heart, but in the total absence of any such mechanical disturbances, it is probable that even my critic may have to fall back upon that clinical observation which he apparently holds in such light esteem.

Let me, in conclusion, gather up briefly the essential facts

[1] *Edin. Hosp. Rep.*, 1893, vol. i. p. 229.

upon which the diagnosis of persistent ductus arteriosus may be founded with perfect confidence. There may be no dyspnœa, cyanosis, œdema, or other evidence of disturbance of the general circulation, and the recognition of the lesion may depend entirely on the presence of a few physical signs. Inspection may fail to yield any facts of diagnostic importance; palpation usually reveals the long thrill following the apical impulse, and enduring beyond the recoil of the blood on the semilunar cusps, which may be felt during the thrill; percussion may not show any enlargement of the cardiac dulness; while auscultation gives convincing evidence of the lesion in a murmur which may be regarded as almost pathognomonic. Beginning distinctly after the first sound, it accompanies the latter part of that sound, occupies the short pause, accompanies the second sound, which may be accentuated in the pulmonary area, or may be, and often is, doubled, and finally dies away during the long pause.



Lungs and heart from case of persistent arterial duct showing it uniting the aorta and pulmonary artery