

inal idea. The point of greatest importance is that a diagnosis of appendicitis should not be based on any one symptom. There is no such thing as a pathognomonic symptom of appendicitis. Pain in the region of the appendix may mean something else. Not long ago a physician made a diagnosis of appendicitis for a man who was traveling in the East and urged immediate operation. The patient returned to St. Louis at once, suffering great pain at the classical point. He asked me to operate for appendicitis. I had him go to bed in the hospital. When I saw him in the morning, there was as beautiful a streak of herpes zoster as I have ever seen, occupying the line of pain. Recovery was prompt.

As to treatment, I believe in operation as soon as the diagnosis is made, unless there is a positive contraindication. In making a diagnosis we must be on our guard, because in the early hours in young children there may be something else brewing. I have seen three cases recently, in which I was called by competent internists to operate, and which occurred in children of five and six years, who within a day developed pneumonia. One of them was rolling around in bed moving his limbs freely. I examined the child and elicited no pain in the abdomen. I asked for ten hour's delay, and at the end of that time the doctor told me the child had an unmistakable attack of pneumonia. First, make a careful diagnosis and then act, but never harbor the idea that appendicitis patients do not die; nor should you operate on the strength of one symptom or a combination of indistinct symptoms.

When you are sure of your diagnosis and the patient is still in fair condition, operate at once. If you wait until pus is forming, then you must follow some other plan of treatment and use your best judgment in selecting the proper time for operation later in the interval. An appendix which has been inflamed must be removed some time.

DR. JOHN B. DEEVER, Philadelphia: We have heard a great deal about race suicide within the last few years from one of our most distinguished citizens, but there is no one thing that contributes so largely to a decreasing population as does sterility, the result of pelvic appendicular peritonitis in young girls and young women.

PERSISTENT PATENCY OF THE DUCTUS ARTERIOSUS

DEGENERATION OF THE CARDIAC MUSCLE AND CORONARY ARTERIES, AND OF THE SINO-AURICULAR BUNDLE, FOLLOWED BY RUPTURE OF THE RIGHT VENTRICLE*

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One of the most interesting problems of physiologists to-day is concerned with the structure and action of the heart. So much experimental work has been done during the past few years on the cardiac muscle, nerves, valves, and blood supply, as well as on the arterial system, by means of measuring apparatus and surgical operations, that any extraordinary case of heart failure which can be studied during life and post-mortem should be made available for future reference, as an addition to our sum-total of knowledge of congenital anomalies, as well as of inflammatory disease.

It is for this reason that I have ventured to make a slight contribution to the literature on congenital cardiac malformation, which in this case was associated with a sequence of disorders of the heart and its valves, resulting in a rare condition of rupture of the right ventricle, and in the patient's death at the age of 26.

In presenting this report I must express my regret that no sphygmographic tracings or electrocardiograms

were made from the heart, the arteries, or the veins; but a brief study of the conditions as a whole may not be unprofitable, although a full discussion of such a case would far exceed the limits of this paper.

REPORT OF CASE

History—The patient was a married woman. Of her early history there is little known; she told me she had always had rheumatism and backache, had suffered frequently from grip, and that her physicians in Berlin, Germany, said she had heart trouble since she was a little girl. Her parents and grandparents were healthy. I first saw her April 3, 1905, and her condition was as follows: Height, about 5 feet, 4 inches; weight, 120 pounds; body, plump; color, good; some acne on face; catarrh of throat and nose; lateral and antero-posterior curvatures of the spine due to muscular weakness, simulating both scoliosis and kyphosis; aortic pulsation in the abdomen noticeable; heart enlarged 1 cm. to right and left, with a murmuring, noisy systolic murmur heard both at base and apex, extending to right of the sternum and almost to the axilla; lungs normal; bowels distended with gas; diarrheal movements; stomach larger than normal; liver normal; left kidney in left groin; pelvic organs normal; urine normal.

After a summer of rest and hygienic life the cardiac murmurs nearly disappeared, but the patient continued to suffer from backache, diarrhea, and catarrh of the throat. The following year she went through a successful pregnancy in better health than usual, even omitting colds and grip. During the summer of 1907 she had a nervous strain which was followed in the early autumn by a condition of flabby muscles and flabby heart, with loud systolic murmur over the pulmonary artery in the third space, and a strong thrill felt over the third and fourth interspaces at the right of the sternum, and frequent palpitations. Her intestinal indigestion was very troublesome, and the facial acne most annoying. Hygienic treatment, baths, massage, careful diet, and occasional doses of codein gave considerable relief, while digitalis, even in minute doses, caused most uncomfortable bounding and beating of the arteries all over the body.

At the end of December, 1907, I referred the patient to Dr. Thayer of Baltimore, who sent me the following diagnosis:

"There is evidently a congenital malformation, but one which from her history and condition, may well be compatible with a long life. The long machinery murmur over the right ventricle with systolic accentuation, together with the accentuation of the second pulmonic sound, and the rather large right side suggest a septum defect (ventricular). This lesion is also more compatible with long life than most other cardiac defects. A striking feature of this case is the fact that the murmur is so loud at the base and in the first left interspace, and that the accentuation high up is late, a late systolic, almost a diastolic accentuation, but the murmur still continues throughout both cycles of the heart with the heart sounds heard, as it were, superimposed. The loudness of the murmur high up and the change in its accentuation would suggest to me an open ductus arteriosus."

1. I have taken the following notes from Dr. Thayer's memorandum: "Weight, 121½. Color is good. General nourishment good. Pulse, 21 to the quarter at the beginning of examination, regular. Radial, not palpable. Pressure, by estimate, 120-25; measurement, maximum, about 115; minimum not easy to estimate. Thorax, symmetrical. Costal angle about 90. Movements, equal. Resonance and respiration clear in front. Back perfectly clear. Heart, P. M. I. in the fifth space, approximately normal position, 8.6 cm. from the median line, begins above in the second space and extends about 5.5 cm. to the right of the median line in the fourth space. No thrill in the precordial region, although the impulse is rather well felt in the second and third left spaces. Sounds, at the apex, first clear excepting for a soft systolic gradually fading away. Second, clear, followed by a very soft diastolic. As one reaches the fourth space a characteristic long machinery sound with systolic accentuation lasting through the whole cycle is audible. This is loudest in the third space, well heard in the second, also over the right side and out over the left chest. In the first left space under the clavicle the murmur was also very long and well heard with late systolic or, perhaps, even diastolic accentuation. The second pulmonic is loud and sharp. The second aortic is also of fairly good intensity. Right kidney descends below the costal margin. Liver is just felt descending below the costal margin on deep inspiration; no enlargement. From the character and position of the murmur one is justified in assuming a septum defect, and from the murmur which is heard up under the clavicle in so high a position, an open ductus Botalli."

* Read in the Section on Practice of Medicine of the American Medical Association, at the Sixty-first Annual Session, held at St. Louis, June, 1910.

During 1908 the patient was fairly well so long as she lived a quiet life and refrained from lifting heavy objects, which gave her pain in the abdomen and slight palpitation of the heart. In November she suffered from another attack of influenza, with tonsillitis, slight bronchitis, and a little pleurisy at the base of the left lung. The mild fever left her very much exhausted, with a hoarse voice and irritating cough, due apparently to a large lingual tonsil and relaxed uvula, but with no new cardiac symptoms, either objective or subjective. She continued hoarse, and although the cough was gone by the end of February, 1909, her heart began to be more dilated and to have the murmurs and irregularity first heard in 1906. After a visit to Lakewood, in April, however, she gained several pounds in weight and felt better, although there was now in addition to the hoarseness a peculiar thin voice.

On April 22, 1909, I referred the patient to Dr. E. Terry Smith of Hartford, who reported a paralysis of the left vocal cord, caused probably by pressure from the heart on the abductor fibers of the left recurrent laryngeal nerve.

A report by Dr. Goodale of Boston, a week later, confirming Dr. Smith's diagnosis, was as follows:

"April 30, 1909. Left vocal cord fixed in median line, immovable on phonation and respiration. Left arytenoid moves inward slightly on phonation. There is no evidence of thickening or irregularity in or about the arytenoids.

"The preservation of motion in the left arytenoid on phonation is due to the contraction of the interarytenoid muscle, indicating preservation of function in the superior laryngeal nerve. This movement, together with the absence of irregularity or thickening in the left arytenoid joint, renders it probable that there is no ankylosis in the joint itself. It is, of course, possible that Mrs. X.'s abductor paralysis arises from grip or laryngitis, but this is, to my mind, less probable than that it arises from pressure along the course of the nerve, for the reason that general infections do not, as a rule, affect the abductor fibers alone of one cord."

The patient was then sent to Dr. Percy Brown of Boston, who made a Roentgen diagnosis of her heart as follows:

"The base of the heart, examined from both behind and in front, presents an undue and abnormal bulging on its left side, which markedly changes the shadow usually recognized as the normal in form and size. This appearance is in close juxtaposition with the descending arch of the aorta, and may to a certain extent involve that structure. The dilatation is general, however, rather than a sharply localized expansion, which would tend to rule out the likelihood of aneurism as being the etiologic factor. I am rather of the opinion that it is due to a hypertrophied and dilated left auricle, secondary to an insufficient mitral valve. This dilatation is the only apparent cause of the mechanical inference with the function of the recurrent laryngeal branch."

For one month, while the patient was living quietly and taking simple vocal lessons, the heart symptoms again improved and the voice became nearly normal, but on June 1 she complained, when walking, of severe paroxysmal pains in the abdomen and legs, especially in climbing stairs or going up hill. The heart was not then larger than usual and the murmurs were faint; pulse 80; systolic blood-pressure was 118, and hemoglobin normal; urine normal. On June 5 the apical mitral murmur of 1906 returned, and the left area of dullness extended 3 cm. to the left of the nipple; pulse 100. On June 11 the pulse was 70 and regular, but a hard, suffocating pain was centered in the stomach and abdomen, at times accompanied, when walking was attempted, by severe pressure below the diaphragm. This persisted for one week, when some edema was noticed in the face and abdomen; the patient passed 500 c.c. of urine in eighteen hours full of urates, but containing no albumin, the specific gravity being 1040; her radial blood-pressure was 140. Digitalis being contraindicated, warm Nauheim baths were given, plus strophanthus and diuretin; the urine increased to 900 c.c. for a few days and the abdominal girth was reduced 6 cm.; at the same time the heart sounded stronger, and the edema left the face. Then, however, the stomach became irritable, the heart began to be very irregular with loud aortic murmur, and all baths and heart tonics were replaced by calomel, Epsom salt, and codein. On July 5, the voice again showed signs of the former

paralysis, and a laryngeal cough was almost constant; the pulse was 105, pressure 130; the legs were very much swollen; the stomach and bowels were full of gas and the urine scanty. On July 9 she could not rest for a moment, owing to the laryngeal cough and cardiac discomfort; at 5 a. m. of the 10th she broke into a profuse cold perspiration; at 7 a. m. she seemed faint and lapsed into a short stupor, after which until death no pulse could be detected at the wrist, even though hypodermic stimulants were freely injected; the carotid pulse was 104, very irregular, and the patient had difficulty in breathing, was restless and thirsty all day, dozing at brief intervals; she took a little nourishment and passed about 7 ounces of thick urine. At 10 p. m. she had another period of fainting with profuse perspiration and gasping, after which she could with difficulty be aroused, her speech was somewhat incoherent and thick, and she was still very restless. At 12:55 a. m., after swallowing a few drops of liquid, she suddenly threw up her arms, opened her eyes widely, gasped once or twice, and died.

So much for the patient's symptoms while under my care. Her earlier medical history is very meager. She was born in Berlin, Germany, a perfectly normal birth and a typically healthy baby, it was thought, but the physicians of her childhood are dead. A letter from Dr. Max Ph. Meyer, Geh.-Sanitätsrath, gave the following information: There was a systolic murmur over the mitral valve, which was louder during the fever of measles in 1895. In 1899, after a period of fatigue, the aortic valves were not found to be free from chronic inflammation, the tones not being quite clear. A note from Dr. Geheimrath Wolf says: "After two examinations, I found a good compensation in the heart (*ein gut compensierter Herzfehler*)."

Owing to the peculiarity of the symptoms in this case and the lack of early history, together with her early death, it seemed wise to seek for a postmortem diagnosis in order to make it possible to use for future cases whatever could be learned of the exact causes for the cardiac weakness in this patient. Dr. Jessie W. Fisher, pathologist to the State Asylum for the Insane at Middletown, Conn., performed an autopsy with the following result:

"*Necropsy.*—Some twelve hours post-mortem. Rigor mortis moderate. The patient was a well-developed, well-nourished, but undersized woman, 26 years of age, with distended abdomen, edematous feet and legs, and cyanosed face and neck. Panniculus adiposus was 3 cm. in thickness. The stomach was much distended with gas, and the abdominal cavity contained at least 2,000 c.c. of straw-colored fluid.

"*Thorax:* Both parietal and visceral layers of the left pleura were adherent laterally, posteriorly, and to the diaphragm. The right pleura was free.

"*Left Lung:* Weight was not taken. The lower lobe showed hypostatic congestion, the remainder of the lung being crepitant, presenting no abnormalities. Bronchi free and unobstructed.

"*Right Lung:* Lower lobe showed a slight amount of hypostatic congestion. Otherwise the tissue was normal.

"*Heart:* The pericardium was smooth and glistening and covered by a very thin layer of fat. The pericardial sac contained about two ounces of fluid blood. When the sac was opened, three ruptures on the right ventricle appeared, parallel to the descending branch of the anterior coronary artery, and 3 cm. to 4 cm. to the right of it. The first was 2.5 cm. from the pulmonary valve, and was 9 mm. in length, slit-like in appearance, involving only the superficial fibers. The second was near the apex of the heart and measured 15 mm. in length externally and penetrated the ventricle by a minute aperture. The third, immediately below and on the apex of the right ventricle, was 15 mm. in length externally, but presented only a pin-point opening internally.

"At autopsy the ruptures seemed to be in the left coronary artery, but on later dissection of epicardial fat the first rupture lay 3 cm. to the right, and the second and third ruptures 4 cm. to the right of the left coronary.

"The ruptures lay in almost a straight line with each other, and the blood had separated the epicardium above and below them, giving the appearance of a thrombosed vessel.

"The ductus arteriosus was patent and had developed into a good-sized vessel, with walls 1.5 mm. in thickness. The diameter of the opening into the aorta was 1 cm., and into the pulmonary artery was 4 mm., where it was surrounded by a thickened ring, and slight fold of membrane. It was funnel-shaped with large end in aorta and 1 cm. in length. The aortic opening of the duct was located opposite the left subclavian artery.

"Heart weighed 395 gm. (Osler gives the weight of a normal female heart as 250 gm.)

"The heart was in systole. The right side was dilated and hypertrophied, containing fluid blood. The right auricle and ventricle were nearly twice as large as normal, and the right ventricular wall was almost as thick as the left, which was also hypertrophied. The foramen ovale was closed, but the fossa was 2.5 by 1.5 cm. At its base the Eustachian valve formed a membranous valve 1.5 cm. in width and 4.5 cm. long, containing muscle fibers.

"Beneath the epicardium of right ventricle there was a marked accumulation of fat, which covered the myocardium and coronary arteries completely.

"The myocardium was thickened, pale in color, and flabby, especially in the right ventricle.

"The auriculoventricular node was well marked, lying between the fossa ovalis and enlarged Eustachian valve. The fibers seemed to pass to the left, where they were lost.

"The ventricular septum was thickened without any indications of defect.²

"The pulmonary artery was almost twice the size of the aorta; it was soft, and the aorta slightly thickened around the duct and valve. Insertion of the duct into the aorta was almost at a right angle.

"The cusps of the pulmonary valve were smooth, thin, transparent and competent. The cusps of the tricuspid valve were smooth, glistening and competent. The aortic cusps were slightly thickened throughout and the aortic opening stenosed. The aorta showed plaques of sclerosis around the valve. The cusps of the mitral valve showed slight thickening along the free borders. This opening was small and stenosed. The descending branch of the left coronary was small and walls thickened, but not calcareous. The right coronary was larger than the left, and like it thickened throughout its course.

"Measurements: Tricuspid, 11.5 cm.; mitral, 8.5 cm.; pulmonary, 7.0 cm.; aortic, 5.0 cm.; left ventricle, 1.3 cm.; right ventricle, 1.1 cm.

"Larynx and Trachea: The recurrent laryngeal nerve was normal in appearance, as was the vagus. The larynx, vocal cords and trachea presented no evidence of pathologic change, except for a small tracheocele situated just above the bifurcation of the trachea. Microscopically, the recurrent laryngeal nerve showed no degeneration with Marchi's stain.

"Microscopic examination of muscle near site of rupture: eosin and methylene blue sections stained poorly, nuclear outline faint or nuclei entirely absent. Muscle fibers separated from each other, and intervals filled with red blood cells. Cross striations very distinct. Some fragmentation. Soudan III sections showed muscle cells loaded with fine granules of fat. Hematoxylin and eosin same as methylene blue and eosin.

"Kidneys: The kidneys were pale in color. The capsule peeled easily, leaving a smooth surface. On section the tissue was pale; the cortex was a trifle narrower than usual, but the markings were distinct. On microscopic examination of the kidneys, capsule of Bowman showed considerable thickening and swelling of the cells lining the capsule, and the epithelial cells lining the tubules were swollen and granular. All of the vessel walls showed extreme thickening.

"Liver: The liver was slightly enlarged, but was not removed for examination.

Anatomic Diagnosis.—"Rupture of the heart and sclerosis of coronaries, patent ductus arteriosus, aortic and mitral stenosis and chronic endocarditis of aortic and mitral valves, atheroma of aorta, hypertrophy and dilatation of right heart,

tracheocele, slight chronic nephritis, fatty degeneration of myocardium."

Having the autopsy report, it was not difficult to explain the symptoms during the life of the patient, for, doubtless, the primary cause of all the pathologic findings in this heart was the persistent patency of the ductus arteriosus. Dr. H. Gideon Wells³ has found in all literature only forty-one cases of uncomplicated ductus arteriosus reported with corroboration of the diagnosis at autopsy, and in only twenty of these cases did the subject reach maturity.

In Maude E. Abbott's⁴ tabulated series of 19 cases of patent ductus arteriosus, the oldest patient was 53, the youngest 6 years old; nine were males, ten females. Out of 106 cases of patent ductus arteriosus, there were eighty-seven cases complicating the defects, leaving the above-mentioned nineteen cases which were proved at autopsy to have the simple defect of patent ductus arteriosus. She gives as causes for this defect the loose musculature of the artery, the sudden change in cardiac blood-pressure at birth, and the alterations in relative position of the ductus and great vessels due to stretching, etc. The usual funnel shape of the ductus is caused by the back pressure of blood from the aorta into the pulmonary vein, but where there is congenital aortic stenosis the current should be reversed. The pulmonary artery and the right ventricle must naturally dilate, and arteriosclerotic patches are not uncommon in the neighborhood of the patent duct and aorta. As for symptoms of a patent ductus arteriosus there may or may not be a systolic thrill, but the "long machinery murmur" is nearly always produced, and the accentuated second pulmonary sound distinguishes this anomaly from pulmonary stenosis. Several other writers have also mentioned the paralysis of the left recurrent laryngeal nerve, due to pressure on that nerve by the patent duct or enlarged auricle, which causes a laryngeal spasm prolonged for hours at a time, giving the right ventricle no time to recover from the added resistance to its contraction, and therefore aiding permanent dilatation of the right side of the heart with all its attendant evils.

My case differed in many points as to symptoms from that reported by Dr. H. Gideon Wells. His patient was 42 years old, suffered from symptoms of gall-duct disease or of acute yellow atrophy of the liver, and died from a hemorrhage of the bowels with a temperature of 106, after a few days in the hospital. The previous history of his case was negative, and the examination of the heart when the patient entered the hospital showed systolic murmurs from the second to the sixth right interspace, so that a tentative diagnosis was made of aortic dilatation with enlarged heart. In the case of my patient, there was no other disease of the body and no symptoms not explained by the condition of the heart, which, however, was well hypertrophied and did not appear by percussion, palpation or auscultation to be unequal to its task. This patient was very susceptible to influenza, having one or two attacks every year. These attacks always increased temporarily the cardiac irritation, and the murmur was at such times much more noticeable than at other times.⁵ In 1906, when the patient went through a successful pregnancy, the mur-

3. Wells, H. Gideon: Persistent Patency of the Ductus Arteriosus. *Am. Jour. Med. Sc.*, 1908.

4. Abbott, Maude E.: Statistics of Congenital Cardiac Disease (400 cases analyzed). *Osler's Modern Medicine*, iv, 390.

5. Schlagenhauser's case of acute aortic endocarditis from the influenza bacillus in a case of patent ductus arteriosus suggests, along with my case, the possibility of the infection from influenza being more virulent than in ordinary cases.

2. The thickness of right ventricle wall is normally 4-7 mm.; left ventricle, 20-25 mm.; left auricle, 3 mm.; right auricle, 2 mm.

murs were very slight, not recurring until after a nervous strain six months later.⁶

It is probable that our patient might have had no symptoms from the large patent ductus arteriosus if she had had no influenza and no other subsequent cardiac complications. Death from a patent duct is usually thought to occur in infancy, although the condition is one which might be overlooked in autopsy, and 9 out of 41 patients whose cases are reported by Wells lived more than forty years.

The characteristic signs of patent ductus arteriosus are in the main those indicated by Dr. Thayer after examining our patient. Osler's⁷ terse symptomatology of a persistent ductus Botalli is as follows: "Loud, especially vibratory, systolic murmurs, with the point of maximum intensity over the upper third of the sternum, associated with a lack of marked symptoms of hypertrophy of the left ventricle." Thayer suspected also a septum defect on account of the character of the murmur over the third and fourth space, but this defect was not found on autopsy. Communication between the aorta and pulmonary artery might cause much the same symptoms as from the communication between the auricles. Their etiology is different, but the physical signs of hypertrophy with systolic murmurs at apex, and generally a diastolic murmur, or continuous murmurs⁸ are common to both conditions. A septum defect is, however, frequently found in hearts in which there is also patent ductus arteriosus. W. Langdon Brown⁹ says: "The deficiency in the exit of blood from the right ventricle will be made good to some extent by the ductus arteriosus remaining open."

Let us now look at the patient's subjective symptoms, as explained by the condition of the heart at the autopsy, and then at the objective symptoms which called forth various hypotheses.

For the last five years of her life the patient complained frequently of palpitations of the heart, sometimes at night, sometimes on exertion. It is possible to explain these symptoms by the mitral stenosis, since out of seventy-three cases of irregularity of the heart, Lewis found that 52 per cent. were instances of mitral stenosis. In many of these patients symptoms of valvular lesion were absent, the patient complaining of short-windedness, fluttering in the chest or neck, and gastric discomfort, the palpitations sometimes occurring in the middle of the night. Such was the case in my patient, the mitral lesions at times being impossible to define, but owing to the slight sclerosis of the mitral valves, there was at times some stenosis of that orifice, which would cause an increase of the interauricular pressure and eventually a condition of fibrosis of the auricles as well as in the ventricles. Lewis¹¹ says that it is possible that fibrosis by interfering with the circulation in restricted areas of the musculature sets up a state of irritability. Anemia of the muscle does the same thing, and may also result in extra systoles, and more or less permanent irregular tachycardia. "Unexpected death in patients with fibrotic hearts is a well-recognized fact." The aortic murmur, not always present, was caused by an incompetent thick-

ened edge of one cusp of its double valve. Osler¹² thinks that this condition is generally well compensated, and may not shorten life. Broadbent, on the contrary, finds that 40 years is about the limit of life in this condition. Where there is any sclerosis of the aorta, however, there is apt to be a narrowing of the orifice of the coronary arteries. Sudden death may be caused by the blocking of one of these coronary arteries, or there may be a more deliberate sclerosis or a narrowing of the coronaries, which leads to fibroid degeneration of the cardiac muscle and eventually to fatty infiltration or fragmentation of the muscle fibers.

In regard to the degeneration of the coronary arteries, Abbott says:

I believe that the commonest site for rupture of the cardiac muscle is on the anterior wall of the left ventricle [in our case it was the right] near the interventricular septum, about the junction of the lower and middle thirds, and the reason that this is the "seat of election" is because this area is supplied by the descending branch of the anterior coronary artery (the artery of the septum), which is a very common branch to undergo thrombosis, or embolism, thus leading to infarction of the supplied area of heart muscle. This vessel has been called the artery of sudden death, because disease of it is so frequently followed by serious results.¹³

In this connection an interesting question arises: Was the intense pain in the legs and abdomen of this patient due to sclerosis of the coronaries and referred to the abdomen instead of being felt in the heart? Or may there have been a sclerosis of the femorals with intermittent claudication, as well as sclerosis of the aorta and coronaries? The victims of angina may be due to a chronic arteritis in the gastric and mesenteric vessels, as well as to the heart. The radial blood-pressure in my patient never exceeded 140, but it is said that even low blood-pressures may co-exist with early arteriosclerosis. Certainly the intense throbbing in the abdominal aorta ought to have shown a higher pressure there than that in the radials.¹⁴ Osler¹⁵ says that spasm or narrowing of a coronary artery, or even of one branch, may so modify the action of a section of the heart that it works with disturbed tension, and there may be stretching and strain sufficient to arouse painful sensations, like the intermittent claudication of the arteries of the legs in a case of cramp.

At the autopsy there was thought to be a condition of thrombus of the anterior coronary artery with rupture in three places, but on careful dissection and microscopic examination the arteries were found to be unruptured, and it was the muscle of the right ventricle at a distance of 3 or 4 cm. from the artery, parallel with it, which ruptured, close to the interventricular septum where its circulation was probably the poorest. Abbott writes that this is a rare condition. She says:

"We have four cases of rupture of the heart in this museum, in three of which the seat of rupture is in the situation above described, i. e., the left ventricle wall. Another possible explanation may be that the auriculoventricular bundle of His is distributed so extensively in the septum that disease of the artery supplying the septum may be productive of more serious disturbance than that of the artery in another part of the heart."

There was no sudden pain in my patient's case to note the formation of a thrombus, only restlessness and anx-

6. One such case is reported in which the diagnosis of patent ductus arteriosus was made in infancy, and again ten years later, and also at the age of twenty years, when Balfour confirmed the diagnosis, after which time the condition began to disappear until, at the age of thirty-five the lesion seemed to have healed entirely.

7. Osler, William: *Practice of Medicine*, p. 769.

8. Osler, W.: *Practice of Medicine*, p. 367.

9. Brown, W. Langdon: *Physiological Principles in Treatment*, p. 308.

11. Lewis, Thomas: *Paroxysmal Tachycardia*, Heart, No. 2, 1.

12. Osler, W.: *Practice of Medicine*, p. 729.

13. Dr. Osler speaks of this in his address on Angina Pectoris, *New York Med. Jour.*, 1897, lxiv.

14. Osler: *Modern Medicine*, iv, 285.

15. Osler, William: *Lancet*, London, March 26, 1910.

lety, followed by collapse, when the heart ruptured, not only once but three distinct times. The patient's father died probably from thrombosis of the coronaries several days after an operation for appendicitis, and as Blumer¹⁸ suggests, there may be an inherited tendency to the production of thrombogen or thrombokinase. The effect of ligation of the coronary artery must be similar to thrombosis. Lewis says:¹⁹

In the case of the right coronary, the area devascularized includes the right auricle and the greater part of the right ventricle. The muscle involved blanches and then becomes livid. The right auricle continues to beat less and less strongly, while the ventricle follows even less strongly to final fibrillation. Tachycardia was obtained in nine out of the twelve cases of right coronary artery ligated, cardiac irregularities most often following obstruction of this artery, and paroxysms occurring in from fifteen to sixty minutes after inter-

from ventricular exhaustion. The rate of rhythm of the ventricular contraction varies from 140 to 120. In my patient the pulsation in the left carotid was from about 160 to a beat too rapid to count, followed by sudden stillness. At the autopsy the blood found in the pericardial sac amounted to only 60 c.c., too little of itself either to stop the heart by pressure or anemia, many a stab wound of the heart being larger than all of the ruptures in the right ventricle. Therefore, it seems to me that we may be justified in attributing death ultimately in this case, to the trouble in the sino-ventricular conducting system²⁰ rather than to the rupture, and that neither the patent ductus arteriosus nor the aortic stenosis would have been incompatible with long life. In all the cases of heart-block or extrasystole which have come to autopsy, there have been shown either gummata, anemic necrosis on account of throm-

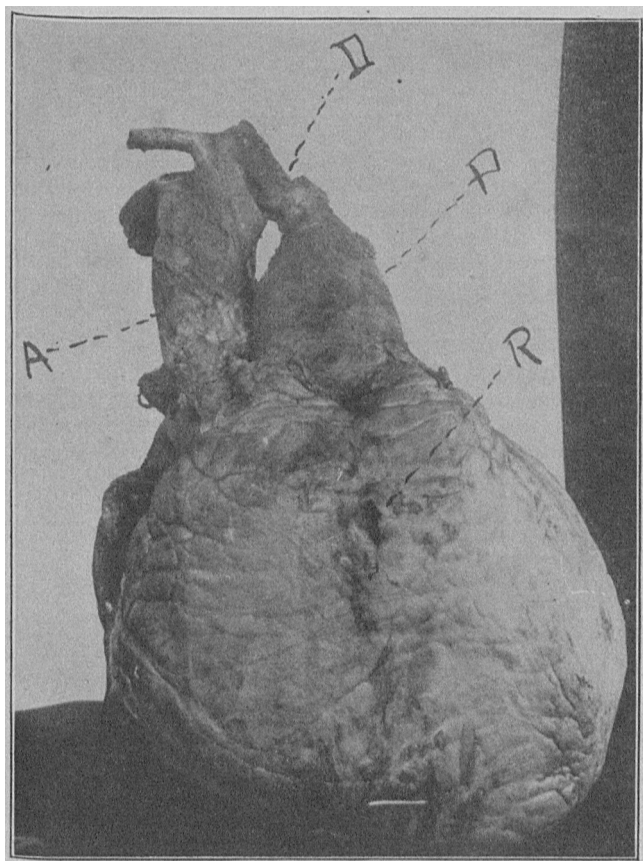


Fig. 1.—Heart from subject with persistent patent ductus arteriosus; A, aorta; D, ductus arteriosus; P, pulmonary artery; R, ruptured right ventricle.

ference with the vascular supply, the paroxysms varying from a few seconds to thirty-five minutes in duration. But if tachycardia is caused by a lesion in the cardiac muscle of the right ventricle itself, independently of nervous control, heart-block and a stop of the auricular beat can be also caused by irritation of the vagus.

Therefore, in this case, the anemic condition of the cardiac muscle, or of the bundle of His, caused by a lack of blood-supply from the sclerosed right coronary artery, may have been the cause of the palpitations and discomfort, while the dilatation of the stomach and intestines may have produced pneumogastric irritation which further increased the cardiac irritability, resulting in fibrillation of the ventricle and sudden death

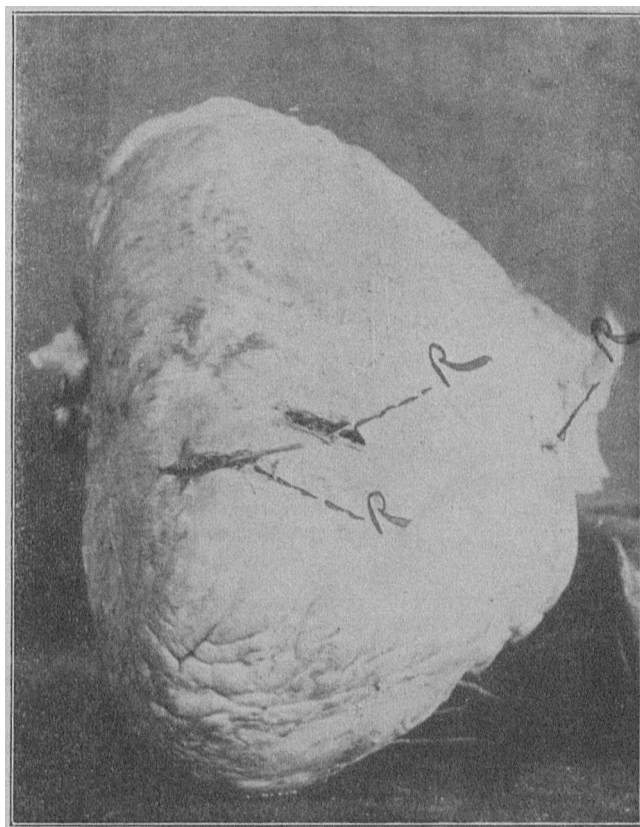


Fig. 2.—Rupture seen from apex of heart.

basis of the nutrient arteries, fibrosis, new growth, athetoma, or fatty infiltration.²¹ The common cause is the same, i. e., interference with the conducting bundle (the auriculo-ventricular bundle), or its node.²² This is probably one of the reasons why digitalis failed to be of service in the case in question, and explains the

20. Retzer has renamed the bundle of His the sinoventricular conducting system. This he considers a neuromuscular apparatus possibly connected with the sympathetic ganglia, the end organs of the conducting system being surrounded by a plexus of non-medullated nerves. Retzer: *Anat. Rec.*, 11, 149; *Johns Hopkins Hosp. Bull.*, 1908, p. 208.

21. Thomas Lewis in a recent article on auricular fibrillation has formulated the following data as to the action of digitalis. "Digitalis retards the ventricular rate in clinical auricular fibrillation by enhancing a previously existing auriculo-ventricular heart-block. The rapid and irregular impulses showered on the ventricle from the fibrillating auricle are hindered in their passage from one chamber to the other by the action of drugs of this class. The influence of digitalis is exerted, directly or through the vagus, on the junctional tissues between auricle and ventricle."

22. Brown, W. L.: *Physiological Principles in Treatment*, p. 251.

18. Blumer: In *Osler's Modern Medicine*, iv, 504.

19. Lewis, Thomas: *Auricular Fibrillation and Its Relationship to Clinical Irregularity of the Heart*, *Heart*, 1910, No. 4, 1, 368.

symptoms of fluttering or big thudding beats, or the long pauses often complained of by the patient, when taking this remedy, and which were dismissed as being irregularities due to indigestion and not to organic change in the heart. In a recent case of rheumatic endocarditis, Mackenzie²³ observed a delay in the *a-c* interval, which was greatly increased by digitalis so that several ventricular beats dropped out altogether,

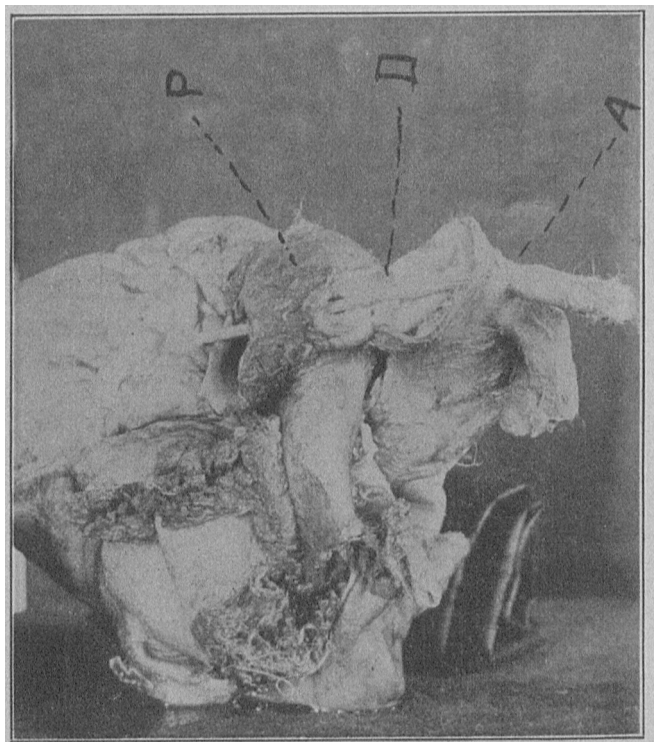


Fig. 3.—A, aorta; P, pulmonary artery; D, ductus arteriosus laid open, showing the opening into both the aorta and pulmonary artery.

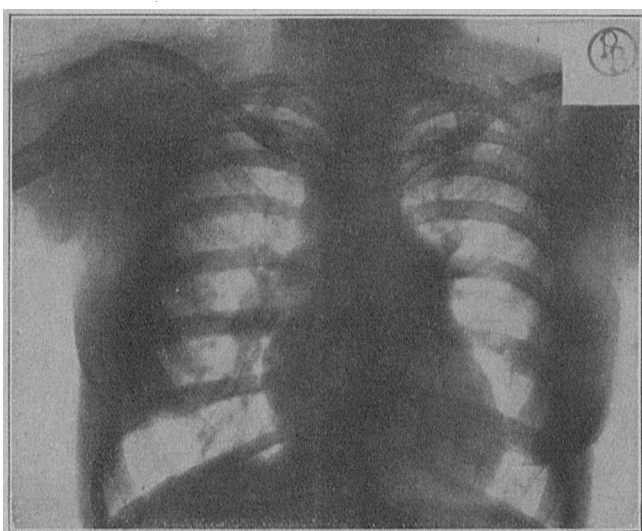


Fig. 4.—Radiograph taken by Dr. Percy Brown, Boston, three months before the death of the patient.

and the pulse became markedly irregular. Stimulation of the vagus was also capable of converting delay of auriculoventricular conductivity into actual heart-block.

During all this time, despite the futility of treatment, the patient's color was very good. She was healthy

looking, cheerful, interested in everything but herself, and never anxious as to her ultimate recovery, an optimism which was shared by all the consultants who examined her, as well as by me, up to the last thirty-six hours, when all the disagreeable symptoms rapidly increased, the most troublesome of all being the continuous laryngeal irritation, and which no hypodermics or any form of treatment could arrest until the final rupture of the heart and death.

The kidneys and liver shared in the general discomfort of this patient for the last two weeks of her life. The liver may be looked on as a sponge-like safety-valve to the heart, which works to its own destruction until edema and ascites occur. If death follows soon, the liver shows its engorgement; if death is postponed, the liver shrinks and shrivels. The edema and ascites in this case remained stationary, and the liver was not noticeably enlarged or fatty.

The condition of the kidneys and their function in this case is best described by Osler. "There is a great lessening in the total amount of urine excreted; darker color, strongly acid reaction which is caused by sarcocollactic acid made from the poor arterial blood, and slight albumin." In other words, a mild nephritis, with thickening of the wall of the arteries such as was found at autopsy.

It is evident that we have one great lesson to learn from the history of such a case as this, namely, the necessity of early diagnosis of congenital defects in the heart, with consequent insistence on a hygienic life, protection as far as possible from infectious diseases (including rheumatism and common colds as well as influenza and children's diseases), and avoidance of nerve or muscle strain. Careful study of cardiac murmurs should teach us to know congenital anomalies, in order to differentiate them from endocardial inflammatory changes in the valves; and wherever it is possible, the diagnosis should be confirmed by autopsy and the cases studied and published for the benefit of the medical profession in the care of similar cases.

ABSTRACT OF DISCUSSION

DR. WILDER TILESTON, New Haven, Conn.: I should like to emphasize the paralysis of the recurrent laryngeal nerve. Pressure by a dilated ductus arteriosus must now be added to the list of possible causes of recurrent laryngeal paralysis. A few years ago v. Schroetter, jr., of Vienna, reported a case of patent ductus with laryngeal paralysis, exactly similar to Dr. Mead's. At that time this was the only case of the sort to be found in the literature.

SIMPLIFIED OPERATION FOR CHALAZION

ROBERT SCOTT LAMB, M. D., WASHINGTON, D. C.

Ophthalmic surgeons have from time to time had recurrences after incising and curetting chalazia and I desire to call attention to a simple procedure which prevents the reforming of secretion in a not quite obliterated cyst wall. After anesthetizing the conjunctiva a chalazion forceps grasps the lid surrounding the tumor and the usual incision of the conjunctiva and the sac beneath is made at right angles to the lid margin. The contents are then carefully removed with a curette and the bleeding controlled. A strabismus hook is then heated red over an alcohol flame, or an electric cautery point is used to gently touch the interior of the sac and so destroy the secreting surface. This has the obvious advantage over extirpation of simplifying the operation and the after-treatment and of lessening the trauma. Previously I had used tincture of iodine, and at another period phenol, but nothing has been quite so satisfactory in my hands as the above procedure.

23. Carter, Alfred H.: Article on Heart Disease, Quoted in the Internat. Med. Ann., 1908, p. 288.