

paid to the cleanliness of the patient and his surroundings.

The country population experiences but few epidemics of typhoid fever in the early autumn, when the doors and windows can be thrown open, and when the application of water can be advocated with the perfect approval of both the patient and the nurse. Like the most of contagious diseases, the infecting material of typhoid may unquestionably be inhaled with the atmosphere which disseminates it.¹³ Such accords with our experience in country practice; such are the views of our patrons, and with each passing year we find it more difficult to secure nurses who are willing to engage in the care of typhoid patients.

Discussion.

Dr. Chapin reported that in 1864 he had seen several cases similar to those reported in the paper.

Dr. J. H. Musser, of Philadelphia, thought that before any conclusion can be drawn, the relations of the first case, so far as the infection of drinking-water is concerned, should be worked out. He regretted that, in connection with the report of the Adams family, no record had been given of the disposition of the stools. In this connection, he said, the epidemic of Plymouth, Pa., well known to all, was of interest. It was there shown that the cases arose from one case of typhoid living at the head of the stream which supplied drinking-water to the communities afterward afflicted.

Dr. Davis, in reply, stated that the stools of the Adams family were not disinfected, but were thrown out upon the ground at a distance. He had failed to mention this fact because he did not think that the poison could reach the well in the twenty-four days which preceded the outbreak of the second case.

A paper was then read entitled

NON-VALVULAR HEART MURMURS.

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By non-valvular heart murmurs I mean heart murmurs that are produced without structural change in valves. Such murmurs are common. They often cannot be distinguished from those that are produced by valvular deformities. The physical signs which accompany valvular lesions enable the diagnostician to locate the morbid process and to determine the character of interference with the circulation which it produces, but does not make it possible to decide on the pathological character of the lesion.

I call attention to the following cases to emphasize the fact that not unfrequently the murmur of a non-valvular lesion is the same in character and location as a valvular one, and that it may be accompanied by the same changes in the size and shape of the heart and other organs.

A man about 45 years old, entered Mercy Hospital suffering with distressing dyspnoea. He coughed little. He was moderately oedematous about his feet. He complained of no pain. His heart was enlarged to the left moderately. Its area of dullness extended about two-thirds of an inch to the left of the nipple line and to the centre of the sternum. The apex beat was strong and easily discernible, though the chest wall was thick. It was moderately diffused and not quite regular. There was throbbing just beneath the sternum of moderate force. No thrills could be felt over the heart. The precordia was not prominent. A systolic murmur was audible all over the heart, but was loudest and clearest at the apex and lowest and most obscure over the aorta. The patient's liver was about one inch broader than was normal. He had no abnormal temperature while he was at the hospital. His urine was normal in amount, slightly cloudy, of a dull straw color and a specific gravity of 1028. It contained considerable albumen. When the latter was coagulated by heat and permitted to settle thoroughly

ly it constituted about one-sixth of the bulk of the urine tested. Granular casts, a few granular epithelial cells and some amorphous granular matter was found in the urine's sediment.

The patient began to be dyspnoic about a week before he entered the hospital. His feet became oedematous at the same time. He had been slightly short-breathed for months. A year prior to this last illness he suffered with acute articular rheumatism. He had been a laborer and was accustomed to use alcoholics freely.

The urine indicated a chronic nephritis. The enlargement of the liver, evident engorgement of the lungs, and dilatation of both ventricles with probably (as the force of the apex beat indicated) some hypertrophy, and a cardiac murmur most intense at the apex, pointed to a mitral valve lesion. The former rheumatism afforded a cause for endocarditis. The shortness of breath which the patient experienced during the year prior to his last illness was supposed to be of cardiac origin. It was thought that the nephritis might have grown out of a passive hyperæmia of the kidneys.

A few days after he entered the hospital a pain of considerable intensity and persistence suddenly developed in his right forearm. The arm also rapidly became oedematous. There was tenderness about the painful point. This new lesion was evidently due to an embolus. In a week the arm was normal in size and all pain had disappeared. He gradually improved so that he could lie down to sleep. The oedema of his feet lessened. Again he was taken worse and became rapidly very dyspnoic. His breathing grew shorter and more labored as his lungs filled with an oedematous exudate until it ceased.

On post-mortem examination his kidneys were found enlarged and presenting the usual aspect of parenchymatous nephritis; the liver was slightly enlarged from congestion. His lungs were oedematous and a small amount of serum was found in the left pleural cavity; the heart was enlarged and soft. The walls of the ventricles were normal in thickness. No lesion or deformity of the valves was discoverable, but a fibrinous clot whose largest mass was a half inch in thickness, was intimately intertwined amongst the chordæ tendinæ of the mitral valve. This was evidently not a new clot for it was firmly adherent to the heart's wall at several points. It was tough and firm. The mitral orifice was not abnormally large. The murmur must have been due to the interference which this clot presented to the closing of the valves and a bit of it formed the embolus which caused his arm to swell. The renal disease was plainly the primary one. The blood-state which nephritis produced predisposed to the clotting of the blood in the dilated heart. The interference with the circulation which the clot caused was precisely that which a contraction of the mitral valves by scar-tissue or other inflammatory change might produce, therefore the physical signs which resulted were the same.

Another case of cardiac murmur due to a heart thrombus, I observed develop in a similar case of nephritis. A young man of thirty-five had nephritis for at least two months before a physician was consulted. When seen by me he was quite oedematous, suffered much from nausea and occasionally from dyspnoea. A severe headache tormented him at night. He had supposed the oedema of his feet was a rheumatic swelling of them. He made only about one half the normal quantity of urine. It was turbid and contained an abundant sediment. In the latter granular and epithelial casts were numerous. There was much amorphous granular matter, a few granular epithelial cells and oil droplets. The urine was strongly albuminous. During the two weeks that followed, the patient grew gradually weaker with, from day to day, varying degrees of comfort and discomfort. He then kept his bed continuously although the oedema had nearly disappeared and the nausea and vomiting had wholly. His urine had increased so that it was little less than normal. While for the most part free from pain or suffering, he felt his weakness greatly. Often at night he felt anxious for air and demanded that the windows be opened, though his breathing was not increased in frequency and there was no evidence of obstruction to the bronchial tubes. His heart was easily excited from the beginning of his illness. Usually his pulse varied from 80 to 90 beats per minute, but slight exertion made it quicker. It was rather small and soft. His skin had the sallow anæmic hue which characterizes chronic parenchymatous nephritis.

About two weeks before his death I found at my morning visit that there was audible over the heart a double systolic beat. The two systolic sounds were perfectly distinct and exactly alike in character. His pulse was then 130 per min-

¹³ Experiments of Sicard. See Jour. Am. Med. Association, Editorial, Vol. xviii, p. 467.

ute. He felt extremely weak and momentarily faint. The systolic reduplication persisted during the half hour that I sat by him. I returned to him about three hours later and then found the double systole gone but a plain murmur audible along the lower right border of the sternum and over the lower end of the bone. It was loudest and clearest in the second intercostal space just to the left of the sternum. It was less plainly audible over the aorta than at the lower end of the sternum and was inaudible at the apex. The murmur was soft, low and systolic in time. Over the pulmonary artery the second sound was wanting. The heart had not changed in size or shape. For a week its area of dulness had extended from the left nipple a trifle to the right of the sternum. The apex beat was scarcely discernible and could not be plainly felt. There was no substernal throbbing. The cardiac murmur which had thus developed persisted to the patient's death. It seemed to me plainly a case of cardiac thrombosis in the right ventricle and interference with the action of the tricuspid valves upon that side.

The patient died 16 days after the cardiac murmur developed. An autopsy displayed kidneys with the characteristic appearances of the patient's disease. The heart was moderately enlarged, a little soft and of normal color. The interior of the left ventricle and auricle was normal. In the right ventricle, adherent to its walls, intertwined with the chordæ tendinæ and muscular papillæ was a large clot of a dull white color and very firm consistence. In the ventricle it was spread out and much divided, but was concentrated into a large, ropy mass a third of an inch in diameter and three inches long, which extended into the pulmonary artery.

These cases are especially interesting as illustrations of the length of time clots may exist in the heart, though not perfectly organized, and the possibility of cardiac murmurs being produced by them. The thrombi formed in these two cases resembled those that are commonly produced just before death when, as often happens, the heart beats with rapidity and, feebleness but they were firmer and their interiors were dryer. When they were removed from the heart the endothelium was torn off in small spots where the fibrin had become attached.

It would be difficult to explain the development of a heart murmur in the second case except by supposing a cardiac thrombus existed such as was found after death. There also existed a blood state which disposes to thrombosis and when the murmur developed the heart was beating with great rapidity and feebleness. In a word the most favorable conditions existed for the formation of a thrombus.

A third case of cardiac murmur with associated physical signs which simulated mitral insufficiency, was first seen at my office:

A woman about 35, with a small family, came to me with feet much swollen, considerable shortness of breath on exertion, frequent coughing, irregular and rapid beating of the heart. Her face and chest were rather thin. She was tall. I found her pulse medium in size, soft, quick and irregular. She had little appetite. She did not complain of indigestion. Her bowels were constipated. She thought her urine was a little less than normal in amount. A sample brought some days later contained no albumen and appeared normal. She menstruated regularly. Her cough was bothersome because of its frequency. She expectorated, not copiously, a frothy sputa. A physical examination of the chest exhibited the usual evidences of a diffuse bronchitis of moderate severity. The precordia was a little more prominent than natural. The heart's apex beat could be faintly felt and scarcely seen. No substernal throbbing could be felt except upon deep pressure in the epigastric region where there was considerable tenderness. The area of cardiac dulness extended to the left nipple and for two inches directly below it; and to the right border of the sternum. No abnormal fremitus could be felt over the heart. A soft systolic murmur was plainly audible at the apex and to the left of it, and less plainly near the lower end of the sternum. At the base the first sound was normal but over the pulmonary artery the second was accentuated. The liver was a little depressed and slightly broader than natural. The

case seemed plainly one of mitral insufficiency with slight dilatation of both ventricles, considerable cardiac weakness; bronchitis and moderate emphysema; moderate engorgement of the liver and general œdema. Relief was readily obtained by cardiac tonics. In two weeks the œdema was altogether gone; her cough almost gone; her breathing was easy except when quite rapid movements were made. Her appetite returned. In every way she felt greatly improved. A physical examination revealed very little change in the signs of cardiac disease except that the heart beat with regularity and greater force, the apex was more plainly visible though its impulse was feeble. I lost sight of the patient after this for 4 months. I was then asked to visit her. She had been in the country and for the first 3 months did well. During the last month her old troubles—cough, dyspnoea, œdema, inability to eat and great debility returned. She was confined to her bed and compelled to maintain a semi-recumbent position. She was able to sleep very little because of her cough and dyspnoea. Her abdomen was distended with ascites. Her urine was scant and contained a small amount of albumen but no sediment. Her heart was very rapid and irregular. Otherwise the physical signs of cardiac disease were the same as when I saw her first. The pulse was small and soft. She was so imperfectly cared for at home that I urged her to enter Mercy Hospital. She lived only four days longer. An autopsy demonstrated firm adhesions between the pericardial surfaces except posteriorly. In front, underlying the lower part of the sternum and extending to the left of it to the apex, was a hard, immobile plaque of partly calcified fibrous tissue, about 2 inches broad and 4 inches long. It covered most of the anterior surface of the heart. It was cut with difficulty and at points was completely calcified. The ventricular walls were moderately hypertrophied. The valves were quite normal and the valvular orifices were not distended.

She never recalled any attacks of pericardial pain or tenderness, but had acute articular rheumatism two years before she was first seen by me. After that she suffered from bronchitis frequently and was constantly dyspnoeic when she exerted herself much.

This case again illustrates the fact that the usual combination of signs of a valvular lesion may coexist though the cardiac valves are without flaw.

Pericardial adhesions can rarely be diagnosed and certainly not when a fibrous or calcified pericardium makes systolic retraction of the sternum and intercostal spaces impossible. Such adhesions are occasional causes of systolic murmurs. The mechanism of these murmurs cannot be certainly explained. They are, however, probably due to irregular contractions of the muscle fibres composing the heart's wall, so that unusual currents or eddies are produced in the blood within the heart.

Anæmic murmurs are so common and usually so easily recognizable, that they scarcely need comment. The signs to be relied upon for their diagnosis as stated by the best authorities are the following: Anæmic and functional murmurs are usually soft, blowing and systolic. When most characteristic they are clearest and loudest about one or one and a half inches to the left of the sternum in the second interspace. Sometimes they are most audible a little lower or even at the apex. When at the apex they are usually heard in a very circumscribed space. They are never loudest over the aorta. Hypertrophy of the heart is not associated with anæmic murmurs though dilatation often is. Murmurs in the veins of the neck are frequently present in anæmic states.

Murmurs due to valvular disease may also be soft and blowing. Though functional murmurs are almost uniformly systolic, they may rarely be diastolic. Murmurs heard at the base of the heart and well to the left of the sternum are uniformly anæmic, but occasionally may be confounded with pulmonary murmurs and the rare mitral murmurs which are heard plainest over the auricle. These latter when charac-

teristically located are close to the sternum. Functional murmurs, I believe, are often loudest at the apex. They are, however, rarely much diffused or propagated to the left into the axillary space, but as in the case of fibroid pericarditis, which I have just described, they may be diffused and transmitted as valvular ones commonly are. An intense anæmia is not much more liable to be accompanied by a cardiac murmur than one of moderate severity.

Soft systolic murmurs occasionally develop as convalescence is being established in rheumatism when the blood has become moderately thin. In two such cases I have heard the murmur only in a small space at the apex. The heart in each instance was slightly dilated, beating rapidly and with feeble force. Under tonic treatment in the course of two or three weeks, the hearts regained their vigor and the murmurs disappeared. Such murmurs are undoubtedly functional and do not signify endocarditis. An endocarditis which can produce a murmur usually develops before convalescence begins and very rarely disappears entirely. Accidental or functional murmurs are quite as likely to develop after rheumatism as after other fevers and prostrating diseases.

It is true that for the diagnosis of a heart disease the *tout ensemble* of cardiac physical signs is of the greatest importance, the mode of their development, the condition of the patient before and at the time of examination, the existence of a former illness such as rheumatism or chorea, the progress and complication of the affection, in a word the medical history of a case is of equal importance. A diagnosis often cannot with safety be based upon physical signs only.

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Discussion.

Dr. Newcomb, of New York, referring to his own experience as examiner for an industrial branch of one of our large insurance companies, stated that he frequently made examinations of individuals who had just come in from work, frequently too after a long walk, and that he had found that murmurs are sometimes distinguishable under such circumstances which are not at all audible in repose. In certain cases of long-standing debility there is a regurgitation through a valve which is not due to disease, but due simply to the relaxation of the orifice. Murmurs of overaction are explained as due to the fact that the action of the ventricle becomes so powerful that the mitral valve is not able to close the orifice completely, and blood is forced back into the auricle.

Dr. Kennedy, of Michigan, remarked that in examinations of boys for the Navy, murmurs have been found which could not be accounted for except as a result of cigarette smoking. Murmurs were also found in the recumbent posture which could not be found in the erect posture.

Dr. Davis explained that the time at his command had not been sufficient to go into the consideration of the cause of this condition. All the explanations that have been given are purely theoretical. We have no experimental proofs as to the causation of these murmurs. The formation of a fibrinous clot as a cause of the murmur is not essential, but probably does occur frequently. He felt confident that this was the cause of the murmur in his second case, from the character of the murmur and from the character of the clot.

The title of the next paper was:

THE IMPORTANCE OF POSITION IN EXAMINATION OF DISEASES OF THE HEART.

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The heart (the only organ in the body which is never at rest) with its investing membranes, the serous and fibrous pericardium, is situated in the

median line of the thorax, and occupies the entire space between the two lungs and pleuræ. This pericardial sac includes the heart, the origin of the large blood vessels, and possesses very complete fixation. Next to the pelvis Paul claims the pericardial sac to be the least movable region in the human body. Its apex is fixed to the skeleton by three ligaments, one coalescing with the inter vertebral ligament of the third cervical vertebra; another uniting with the middle aponeurosis of the neck is inserted into the hyoid bone, the other passes anteriorly to the sternum.

The base of the pericardium is fixed to the centre of the diaphragm and through the medium of this muscle is united posteriorly to the spinal column, and anteriorly to the ensiform appendix.

Now, the heart, which may be compared to a hollow muscle acting like a great force pump to propel the blood through all parts of the body, is kept in place by the great vessels which spring from its base, and by the attachments of its investing sac, the pericardium, which has been shown by before stated anatomical facts to be fixed solidly to the skeleton, and therefore changes its position but little, during the respiratory movements of the diaphragm, hence the apex beat in a normal heart is but slightly raised or lowered during inspiration or expiration, and whether the patient is examined either lying down or standing; but on the other hand quite marked displacements may be observed in abnormal cases where hypertrophy has increased the weight of the organ, and the resistance of the ligaments has been gradually overcome.

Altered posture of the body always displaces the normal heart transversely which sinks from three to six centimeters when lying on the left side, and from one to three centimeters during right decubitus. Position also illustrates the effects of gravity on the heart in the greater force and more extended apex beat, when the chest is bent forward, a procedure that should always be practiced when this beat is obscure.

In position the axis of the heart is directed from above downwards and to the left, two-thirds of its bulk passing to the left, and one-third to the right of the median line of the sternum. Its upper border corresponding to a line drawn across the sternum on a level with the upper border of the third costal cartilages, its apex lies immediately behind the sixth left costal cartilage inside the mammillary line. The lungs passing downward from their apices meet each other behind the sternum at the level of the second rib and are separated only by the mediastinal fold of the pleura until they reach level of the fourth rib where they again diverge. Thus it will be seen that both auricles and part of the ventricles are completely covered by lung tissue, which is a very bad conductor of heart sounds, but after the divergence of the lungs at the level of the fourth rib we have about one-half of the right ventricle and the apex of the left, between which and the chest wall no lung tissue intervenes, yet the extent of heart surface not covered by lung tissue is subject to constant variation; during deep inspiration the edges of the lungs may converge so as to cover the entire surface of the heart, and on the other hand during forcible expiration the margins of the lungs recede to such an extent that the larger part of the heart's surface comes close to the chest wall.

The extent of the movement of the thorax in nor-