

by morphine? Who will not prescribe this drug in the sudden heart failure of myocarditis—the asystole with the accompanying oedema of the lungs of this affection? Who does not give it in rheumatism for its tonic cardiac effect? I plead for the continuous use of opium or morphine also in myocarditis to prevent angina pectoris, or to lessen the effect or defer the dreaded asystole. I have had patients take for months and months small doses of the deodorized tincture or the extract of opium, thereby checking waste, reducing the susceptibility to peripheral sensations which fret an irritable heart, replacing exciting stimulants, as alcohol and strychnine, calming a perturbable nervous system, and lessening the necessity for food to the relief of digestion, metabolism, and elimination.

In cases of weak heart after exhausting disease, after prolonged mental and physical pain, and without organic lesion of valves or muscle opium is of advantage.

In cases of failing compensation, with the onset of stases, the heart is supported, especially if the unfortunate possessor is an impressionable subject who frets and fumes because of the ordinary irritations of life.

In the gradual engorgements from myocardial dilatation, in chronic parenchymatous nephritis, and in arteriosclerosis it is of value. If the patient is hypochondriacal or hypersensitive the second daily dose of opium invites sleep and induces a feeling of well-being.

The dyspnoea of myocarditis is relieved or prevented by continuous small doses of morphine for a very long time. I have seen a form or stage of myocarditis with restlessness, Cheyne-Stokes breathing, dyspnoea, and rapid pulse helped by continuous doses of opium. The tachycardia of Graves' disease is relieved, and in three of my instances it appeared to contribute to the cure of the disease. In the nervous and irritable subjects opium is almost necessary to induce comfort.

**A REVIEW OF THE CASES OF DISEASE OF THE HEART
MUSCLE TREATED IN THE SECOND MEDICAL DIVISION
OF THE BELLEVUE HOSPITAL OUT-PATIENT
DEPARTMENT FROM NOVEMBER, 1903,
TO JUNE, 1905.**

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AMONG the 2127 patients treated during this period there were 89 cases of heart disease, of which 57 were classified as valvular disease, 22 as disease of the heart muscle, and 10 not at all, owing to incomplete data.

In a dispensary service the small percentage of patients returning for treatment necessarily limits the opportunities for extended observation of individual cases, and increases the difficulty of making a diagnosis of myocardial disease, whose recognition, even under the most favorable conditions, is very perplexing.

AS REGARDS OUR CLASSIFICATION. All diseases of the heart muscle, if they are evidenced at all during life, have one result in common—namely, insufficient work by the heart—whereby the circulation is so slowed that the demands of the body are not satisfied. Clinically we recognize this cardiac insufficiency by a syndrome of symptoms—subjective cardiac disturbances, objective changes in the heart and pulse, œdema of the lungs, dyspnoea, swelling of the liver, œdema of the legs, decreased quantity of urine, etc., and of these the last four are the most reliable.

Having decided that cardiac insufficiency exists, the difficult part of the diagnosis is now to determine what has caused the cardiac insufficiency. Whether lung disease, nephritis, obesity, muscular overstrain, coronary sclerosis, adherent pericardium, or chronic myocarditis is the underlying causal factor. Strictly speaking, valvular disease, in view of its effect on the heart muscle, should also be considered a cause of this condition.

Dividing our cases on this very practical etiological and clinical basis, which is much in favor with German clinicians, we find the cardiac insufficiency to be due in 4 cases to chronic nephritis, in 5 cases to disease of the coronary arteries, in 2 cases to muscular overstrain, in 1 case to adherent pericardium, and in 10 cases to chronic myocarditis.

Group 1. Cardiac insufficiency in chronic nephritis. The sequence of events in these cases is somewhat as follows:

The nephritis, generally of the interstitial variety, has already produced a cardiac hypertrophy, but is not itself in a terminal stage. Then myocarditis, muscular overstrain, coronary sclerosis, overindulgence in alcohol, or some other heart-weakening event supervenes and damages the heart muscle so that it is no longer able to do the extra work which nephritis entails, and a condition of cardiac insufficiency results.

Uræmia frequently follows from the impairment of the kidney circulation, but there are a certain number of cases in which the uræmic symptoms are absent or slight, where the heart symptoms dominate the picture, and where treatment of the cardiac condition restores the patient, at least temporarily.

CASE I.—W. C., an ice man, aged thirty-two years, was treated in Bellevue Hospital in February, 1903; July, 1904, and from January to June, 1905, for chronic parenchymatous nephritis. On the last of these occasions he suffered from marked œdema of the legs and face, ascites, and chronic uræmia. His heart was hypertrophied, showing a heaving apex beat, ringing and accentuated

aortic second sound, and a very high tension pulse. During the latter part of May he passed from 2950 to 3550 c.c. of urine daily, showing a specific gravity of 1009 to 1012, a marked quantity of albumin, and granular and hyaline cysts. He came to the dispensary June 25th of this year complaining of distressing attacks of dyspnoea at night, lasting an hour or more, and shortness of breath on exertion.

Status Præsens. A well-built man, somewhat emaciated, with pallid skin and mucous membrane, dyspnoic, and showing slight cyanosis of the lips. There is no oedema of the legs. Numerous subcrepitant rales are heard over the base of the left lung. The apex beat is feeble and displaced outward. The percussion area on the right extends 1 cm. beyond the right sternal border. The action is rapid, regular, and a gallop rhythm is heard over the entire præcordium. The aortic second is slightly accentuated, the pulse is 118 per minute, regular, and shows some tension. The superficial arteries are thickened markedly. The edge of the liver is felt 9 cm. below the free border of the ribs in the midclavicular line. He is passing but 1770 c.c. of urine in twenty-four hours, with a specific gravity of 1010.

Under cathartics, digitalis, and nitroglycerin the pulse came down to 90. His dyspnoea almost disappeared and the quantity of urine increased.

It is interesting to note that although the urine, as the result of nephritic congestion, was reduced from 2950 c.c. (his output on leaving the hospital) to 1770 c.c., the specific gravity still remained low. Romberg calls attention to this peculiarity of cases of cardiac insufficiency in interstitial nephritis—the passing of a decreased quantity of light-colored urine of low specific gravity instead of dark-colored urine with high specific gravity, which we ordinarily find in the nephritic congestion of cardiac insufficiency. In the absence of albumin this would afford a valuable clue as to the original cause of the heart condition.

The above case is the only one of the group presenting this feature.

CASE II.—K. I., a laundress, aged forty-five years, had scarlet fever and diphtheria in childhood, and has been a heavy drinker for some years. For the past three weeks she has had dyspnoea on exertion, swelling of the feet and legs, and numerous attacks of shortness of breath at night, forcing her to sit up in bed.

Status Præsens. An obese dyspnoic woman with alcoholic facies, slight icterus of the conjunctivæ, and marked oedema of both legs. There are subcrepitant rales at the base of the right lung. The apex beat is displaced outward in the fifth space and is feeble. The heart is very irregular in action with the first sound of poor muscular quality. There are no murmurs, and no accentuation of the second sound. The pulse is 120 to the minute, very irregular

in force and frequency, and of low tension. The liver is enlarged and tender, the rounded edge being felt 10 cm. below the ribs in the midclavicular line. The urine, of a specific gravity of 1027, shows a large quantity of alumin and numbers of granular casts.

The patient was given tincture of digitalis every three hours, and two compound cathartic pills every night for four nights. She returned after four days of this treatment very much better. The oedema had disappeared, the pulse, although still irregular, had dropped to 90 and become a little tense. The apex beat was somewhat heaving, and the aortic second sound distinctly accentuated.

Four months later she had another attack of cardiac insufficiency, caused by heavy drinking. Her obesity also undoubtedly played a part in producing the heart condition.

The remaining 2 cases of this series complained of dyspnoea on exertion; showed enlarged hearts with gallop rhythms, rapid, regular, and low-tension pulses, and urine with rather high specific gravities, marked quantities of alumin and granular casts.

These 4 cases are the only ones whose selection as types of this form of disease of the heart muscle was warranted by the prominence of the cardiac symptoms.

In addition to these there were 14 cases of nephritis showing a high tension pulse and cardiac hypertrophy, and suffering from either dyspnoea on exertion or constant dyspnoea. This dyspnoea could not be considered an evidence of cardiac insufficiency, but rather a result of the high tension pulse. There were also 5 cases showing cardiac hypertrophy, and a high-tension pulse with no subjective disturbances.

Group 2. Cardiac insufficiency due to sclerosis of the coronary arteries.

CASE I.—Mrs. A., aged fifty-eight years, housewife, gave the following history: Her father died of nephritis. Her mother from apoplexy and her husband from angina pectoris. She had scarlet fever when young, but never rheumatism. During the winter of 1904-1905 she suffered from dyspnoea on exertion. In January, 1905, the dyspnoea became almost constant. Several times she had severe nocturnal attacks of shortness of breath, obliging her to sit up on a chair for an hour or so. On the night of February 7th she had a severe pain over her heart, running up to the right shoulder and down the inner side of the right arm, accompanied by dyspnoea and a cold perspiration. This lasted more or less all night, and she volunteered the information next day that she thought she was going to die.

Status Præsens. A thin, pallid, dyspnoeic woman with no cyanosis or oedema. The lungs show numerous subcrepitant rales at the right base. The apex beat is felt 12 cm. from the median line in the fifth space, and is not heaving. At the apex is heard an almost continuous peculiar rumble with at times a suggestion of

gallop rhythm. The muscular element of the apex sound is very poor, and the second sounds are feeble. The pulse is 100 full, regular, and of low tension, and intermits occasionally. All of the superficial arteries are sclerosed. The liver is not enlarged.

February 9th. No murmurs were heard over the heart, the dyspnoea was better; 628 c.c. of urine were passed in the twenty-four hours, with a specific gravity of 1025, $\frac{1}{2}$ gram of albumin to the litre, full of urates, and showing microscopically a few hyaline casts.

13th. The apex beat was slightly heaving (she had taken small doses of digitalis and nitroglycerin). That afternoon there was a slight attack of pain in the right shoulder and arm. During the night she started up suddenly in bed and seemed in great pain, and died in a few minutes.

In this case the insufficiency of the left ventricle was evidenced first by the feeble and dislocated apex beat, the poor muscular quality of the first sound, the suggestion of gallop rhythm, and the rapid pulse; and secondly, by the dyspnoea, the congestion of the right lung, and the decreased quantity of urine.

The attacks of angina and cardiac asthma seem to show definitely that sclerosis of the coronary arteries was the underlying cause of the cardiac insufficiency.

CASE II.—W. H., a clerk aged fifty-seven years, has never had lues or rheumatism, and is of excellent habits. During 1898 he had several fainting spells, and his physician told him he had "heart trouble." In 1902 he began to have attacks of pain in his chest. The pain would begin in the left axilla, radiate across the upper part of the sternum, to the right side, and frequently down the right arm to the wrist, where it seemed to centre "over the pulse." It felt as if a "red-hot iron were being drawn across the chest," and he would immediately sit down and remain quiet. These paroxysms lasted from a few minutes to a half-hour. After a few months the pain ceased to affect the right wrist, but was localized "under the breast bone."

At present, May, 1904, the pain comes on frequently, sometimes at night, but generally after a full meal or when he is excited ("excitement acts just like a full meal"), or when walking against the wind. He is also troubled with shortness of breath on but slight exertion, epigastric oppression, and belching of gas.

Status Præsens. A pallid, slightly dyspnoeic man, of full habit. He shows no pretibial cedema. The apex beat, heaving and with sudden fall, is felt in the fifth space 11 cm. from the median line. All of the superficial arteries pulsate. There is dulness over the base of the sternum and in the adjacent portions of the second and third right interspaces. Systolic and diastolic murmurs are present over the entire præcordium, the former being heard all over the upper portion of the chest. The pulse is 96, regular, and of Corrigan type. There are a few moist rales at the base of the right lung.

The liver is not enlarged, but very tender on pressure. He is passing 1500 c.c. of urine in the twenty-four hours with a specific gravity of 1019, no albumin or sugar, and a few hyaline casts.

A week later he had a number of typical attacks of the cardiac asthma at night, and his general condition was worse, the dyspnoea becoming more marked, and the heart action faster. I did not see him again, but his family physician told me that he became constantly dyspnoeic, took to his bed, and after five days of terrible suffering died. The chief symptoms during his last illness were the dyspnoea, a rapid and irregular pulse of low tension, several attacks of oedema of the lungs, extreme irritability of the stomach, oedema of the legs, and a very much decreased quantity of urine of high specific gravity, with traces of albumin. The anginal pains ceased. A very typical picture of cardiac insufficiency arising from disease of the coronary arteries.

In this case sclerosis of the aorta evidently produced the lesion of the aortic valves, and later disease of the first portion of the coronary arteries. The coronary affection, as in the preceding case of this group, was shown first by the numerous attacks of angina pectoris and later by the attacks of cardiac asthma. The coronary disease would seem to be the determining factor in the production of the cardiac insufficiency and not the valvular trouble.

The 3 other cases of this series showed typical attacks of angina pectoris of a less severe form and evidences of cardiac insufficiency; but although there was dyspnoea on exertion, there was no attacks of cardiac asthma. They also showed accentuated aortic second sound, and in 2 cases note is made of the low tension and irregularity of the pulse.

The first 2 cases of this group present classical pictures of disease of the heart muscle from coronary sclerosis, and a very interesting association of attacks of true cardiac asthma and angina pectoris, both arising probably from the same cause. The distressing shortness of breath in this condition suggests those cases of arterial sclerosis and interstitial nephritis, accompanied by high tension pulse and dyspnoea, but the differential diagnosis is not difficult.

In the 3 remaining cases the only evidences of coronary sclerosis were the attacks of angina and the few physical signs pointing to sclerosis of the first portion of the aorta.

Group 3. Cardiac insufficiency from muscular overstrain.

CASE 1.—T. M., laborer, aged thirty-eight years, came to the dispensary June 8, 1904, complaining of shortness of breath and swelling of the feet and scrotum.

He had been a heavy drinker for some years. On June 1st he lifted a bar of iron weighing about 300 pounds. A few minutes after he experienced a dull præcordial pain, and that night was troubled with palpitation. The pain continued and the next day

he was short of breath and his feet were swollen. The swelling and the shortness of breath have increased.

Status Præsens. A well-nourished, thick-set, plethoric man, dyspnoëic, with slight cyanosis of the lips, ears, and finger tips, and marked œdema of the feet, legs, prepuce, and scrotum. The carotids are pulsating violently. The lungs are not enlarged, and show signs of a slight bronchitis. The apex beat is diffuse, not forcible, and most intense below and external to the nipple. The cardiac dulness extends $1\frac{1}{2}$ cm. to the left of the nipple line and 1 cm. beyond the right border of the sternum. The action is rapid. The first sound at the apex is valvular in character, and the pulmonic second sound is accentuated. The second aortic sound is indistinct over the aortic area, but heard clearly at the apex. A distinct localized systolic murmur is heard at the apex. Over the aortic area are heard clearly faint systolic and diastolic blows. The pulse is 120, regular, of slightly increased tension, and with a quick rise and fall. There is some œdema of the lower zone of the abdominal wall and the superficial abdominal veins are slightly dilated. The liver dulness begins at the fifth space in the nipple line, and the edge is felt distinctly 6 cm. below the free border of ribs. The urine on several examinations showed neither albumin nor casts.

The patient was sent up to the ward and twenty-four hours later the heart had quieted down, all murmurs had disappeared, the œdema was much less, and in a few days he was discharged cured.

On June 25, 1905, this patient still remained in good health, had no dyspnoëa on exertion, and physical examination showed a normal heart. After exercise the pulse was 88 to the minute.

The history and physical examination of this case were confirmed by the late Dr. J. M. Polk.

The second case was that of a coal-passer, whose quite heavy work was done in a room where the temperature ranged between 130° . His heart gave out, the chief symptoms being dyspnoëa and palpitation. I saw him one year after this for the first time, and he still complained of dyspnoëa on exertion and palpitation. Objectively he showed nothing except perhaps a rather easily quickened pulse.

Group 4. Cardiac insufficiency from adherent pericardium.

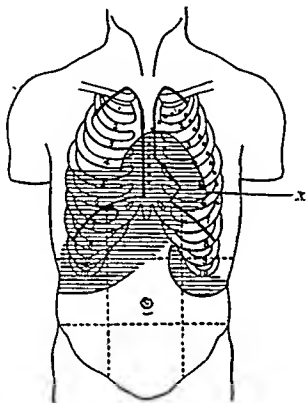
R. D., aged fifteen years, a school boy, was seen in July, 1904, complaining of shortness of breath.

During childhood he had measles, whooping-cough, and scarlet fever. From 1899 to 1900 he had lived in a malarial district, and suffered from numerous attacks of "fever and ague." In 1901 he was taken sick rather suddenly and his physician diagnosed heart trouble. He was in bed for two months, and various other diagnoses (sarcoma of liver, congestion of liver, etc.) were made. Three or four months later his feet became swollen and he began to suffer from dyspnoëa on exertion. At times he became blue. For the

past two years he has been comparatively well, but is always short of breath on exercising.

Status Præsens. A large, well-nourished, well-built boy of good color. He became a little short of breath as the examination progresses. There is no pretibial œdema. The right external jugular vein is prominent, but there is no diastolic collapse or regurgitant pulsation seen.

A gallop rhythm is heard at the apex, the pulmonic second sound is accentuated, and the aortic second normal. No murmurs are



X, apex beat which is seen distinctly in the fifth space eleven centimetres from the median line. An area of systolic retraction represented by the dotted line is present. It extends from a point immediately to the left of the centre of the sternum almost to the axilla and up under the clavicle. A very distinct rebound (synchronous with the second sound) is seen and felt over this area. The apex beat is heaving and moves less than one centimetre to the left, but became more distinct when the patient turns on his left side. The cardiac dulness extends two and one-half centimetres beyond the right border of the sternum, and twelve centimetres to the left in the fifth space.

heard. The pulse is regular, small, of low tension, 100 to the minute, and is not paradoxical. The motility of the lung border overlying the heart was not tested. The lung shows dulness and decreased expansion at both bases (due to the large liver and spleen). There is no Broadbent's sign. The abdomen appears decidedly enlarged. The liver dulness begins in the fifth space and the rounded edge can be felt 7 cm. below the free border of the ribs at the ensiform, and 12 cm. below at the nipple line. The bard edge of the spleen is felt 10 cm. below in the nipple line. There

is no free fluid in the peritoneal cavity. In this case the early and persistent congestion of the liver and spleen points to an insufficiency of the right ventricle, whose wall, as a result of the anatomical relation, is affected much more than that of the left ventricle by an adherent pericardium. The blood and uric examinations are absolutely normal.

Group 5. There remains a group of 10 cases showing evidences of cardiac insufficiency, in which the underlying cause was provisionally diagnosed as chronic myocarditis. The direct recognition of this condition is extraordinarily difficult, and our rather questionable conclusions were reached more by a process of exclusion than anything else. That is, none of the cases showed evidence of nephritis, muscular overstrain, coronary sclerosis, obesity, or adherent pericardium.

In none of our patients was the heart hypertrophied. That is, there was no suggestion of heaving about the apex beat, nor any accentuation of the second sound. Myocarditis *per se* does not cause hypertrophy, but myocarditis often attacks an hypertrophied heart, in which case the diagnosis is always more than problematical.

The two following histories form typical examples of this group of cases:

CASE I.—J. F., printer, aged twenty-eight years. He has had measles, scarlet fever, whooping-cough, three attacks of inflammatory rheumatism, and three attacks of gonorrhœa. His habits have been alcoholic. For the past four weeks he has suffered from shortness of breath on exertion, for three weeks from cough, and for two weeks from œdema of the feet and ankles.

Status Præsens. His lungs show signs of bronchitis. The apex beat cannot be seen or felt. The heart percusses to the right border of the sternum and 12 cm. to the left of the median line in the fifth space. The action is very irregular, and there are no murmurs or accentuation of the second sounds. The first sound at the apex is prolonged and weak. The pulse is 100, of low tension, and markedly irregular in force and frequency. The radials and brachials are thickened. The edge of the liver is felt 6 cm. below the ribs in the nipple line. The urine is of a specific gravity of 1020, and shows a trace of albumin, but no casts.

The patient died in the hospital a few days later, with a diagnosis of alcoholic delirium and acute cardiac dilatation.

CASE II.—T. K., longshoreman, aged fifty-three years. He has had numerous attacks of arthritis involving the small joints of the hands and feet, and sometimes the knees and hips. For five years he has suffered from dyspnoea on exertion, which has been worse of late. His habits are alcoholic.

Status Præsens. A powerfully built man, five feet nine inches tall, weighing 198 pounds. He is dyspnoic (respiration 32 per minute). The lungs are negative. The apex beat is diffuse and

felt indistinctly in the fifth space 11 cm. from the median line. The heart action is markedly irregular, but no murmurs or accentuated sounds are heard. The pulse is 142 per minute, very irregular in force and frequency, and of low tension. The radials and brachials are sclerosed. The liver is enlarged. He is passing twenty-four ounces of urine in the twenty-four hours, showing a specific gravity of 1026, a trace of albumin, and no casts.

Six of the patients of this group were between forty and fifty years of age. Seven gave a history of heavy drinking; 5 of infectious diseases, 1 of syphilis, 1 of lead poisoning, and in 3 there was nothing to account for the cardiac condition.

All of our patients were dyspnoeic on exertion, 3 had typical attacks of cardiac asthma, 6 had subjective cardiac disturbances (palpitation, oppression, etc., but no anginal pain), and 6 suffered from cough. The heart and pulse were irregular in 8 cases. One showed a bradycardia (58) and 2 attacks of tachycardia (pulses of 160 and 172).

One of the cases of tachycardia for two months showed a marked œdema of the legs, enlargement of the liver (his habits were excellent), emphysema of the lungs, and an apparently normal heart. The urine was quite normal, and an explanation of the œdema was not forthcoming until one day he appeared complaining of oppression in the chest, and we found that his heart was beating at the rate of 160 to the minute. He recovered on digitalis, and after this experience the muscular sounds of the apex always seemed weak and feeble.

I am indebted to Dr. Hastings, Dr. Roper, and Dr. Warren for the blood and urine examinations of these cases.

THE CLASSIFICATION OF THE CASES HERETOFORE CALLED RHEUMATOID ARTHRITIS.

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THIS is the first of a series of papers on the joint diseases heretofore classified as rheumatoid arthritis, chronic rheumatism, arthritis deformans, pseudo-rheumatism, etc., which I hope to publish at intervals in the near future. The first two or three papers are intended to be preliminary, and will in a broad way deal with the classifications of these diseases. In these preliminary papers it will be noticed that the facts which have lead to some of the conclusions are not mentioned in detail. These are purposely omitted because the plan is to start out with a comprehensive view of the whole subject and too many details by complicating it might, I