feeling of weakness, and fell to the floor in a fainting condition. When seen by Dr. Fotheringham a short time after he was extremely cyanosed, the heart's impulse diffused and fluttering, and the pulse imperceptible. After a few hours he was removed to his home. Evidence of dilatation of both ventricles was present, most markedly of the left. Every effort to overcome the condition was of no avail, death occurring in a week from the onset of the attack. Autopsy revealed marked hypertrophy of both sides of the heart with extreme dilatation of both ventricles. The cardiac muscle was pale, soft and flabby, and showed "tabby-cat" streaking beneath the endocardium. There was no valvular trouble. The fatal issue of this case was nearly a year and a half after his attack of typhoid fever. The splendid muscular development of the patient, with the impatience to restraint so often seen in those who have always enjoyed vigorous health, unfortunately became causes for overtaxing the myocardium, weakened out of proportion to the general strength.

These two cases, occurring in members of our profession, certainly suggest a lack of apprehension of the dangers, even at a remote period, from over-exertion following typhoid fever.

In convalescence from diphtheria, the occurrence of sudden death from cardiac failure, even in the most carefully-managed cases, is a common, unpleasant experience of every physician. In other cases, however, death or severe disability could in all probability have been averted by the exercise of care. The following case will illustrate the point:

CASE XI.

Miss P., aged 23, a nurse in a Brooklyn Hospital, had an attack of diphtheria in December, 1903. Until this time she had been quite strong and showed no signs of heart weakness. Antitoxin was administered, rapid improvement followed, and she was better within a week. Being anxious to return to Toronto for the Christmas holidays, though warned of the danger by her physician, she left the hospital in two weeks, went to New York, spent the afternoon shopping, and took the night train for Toronto. On her arrival the next morning she was cyanosed, suffered from extreme dyspnoea and precordial distress, and the pulse was rapid, irregular, and almost imperceptible. The feet, legs, and face were much swollen. The apex beat was weak and diffused, and the point of maximum impulse was out as far as the anterior axillary line in the fourth interspace. The urinalysis showed no evidence of renal trouble. No cardiac murmurs were present, though the first sound at the apex was weak and valvular in character, and the second pulmonic sound was greatly accentuated. The transverse cardiac dullness was much increased both to the right and left of the sternum. Under absolute rest and treatment, the oedema soon disappeared, the cyanosis and dyspnoea passed off, and the pulse became slower and more regular. After six weeks, however, the point of maximum impulse was in the fourth interspace, $1\frac{1}{2}$ in. outside the nipple, and was very weak. The pulse rate had fallen to between 80 and 90 but was increased on the least exertion or excitement. Owing to her urgent importunity, she was allowed to be gradually propped up higher in bed, and after some days to be lifted into a chair at the side of the bed, for a carefully-lengthened period each day. She was instructed of the danger of attempting any exertion beyond that specified. I had not visited her for some days, and as she felt well and apparently concluded that the dangers had been exaggerated, she finally dressed and went downstairs. As there was no apparent ill-effects from her venture, she undertook to call on some friends, and finally surprised me one day by calling at my office to inform me of her intention to return to her duties at the hospital. I impressed her with the extreme danger attendant on such a course, but she was insistent. The next day, however, I received an urgent message to visit her. She was very nervous, breathing rapidly, complained of distress over the lower part of the precordium, and the pulse was weak and rapid. The point of maximum impulse was again found out to the anterior axillary line. Three months of absolute rest followed, during which time the urgent symptoms had disappeared, and the apex beat had returned to about $1\frac{1}{2}$ in. outside the nipple. She was then allowed to gradually sit a little higher in bed, then to be placed in a wheeled chair, and after three months more to carefully begin walking about the room. The slightest over-exertion, however, would produce an anginal attack. At the present time, fifteen months from the beginning of her illness, she looks well, and is extremely careful no symptoms arise. The apex beat is still an inch outside the nipple, most evident in the fourth interspace, and the impulse is weak. No murmurs have developed.

In attempting to form an opinion as to the ultimate prognosis in this case, one is struck with the dearth of literature bearing on the subject. A cure must involve nerve and muscle regeneration, with efficient compensatory hypertrophy—a process, of necessity, prolonged if at all possible in an organ continually subjected to a degree of functional stress. Recovery much beyond that which she has attained at present is unlikely.*

During the acute stages of these infective diseases, while the patient is under the physician's supervision, the danger of cardiac trouble is generally apprehended, its manifestations

watched for and appropriate treatment carried out. But unfortunately this supervision is not extended over a sufficient length of time. The question, therefore, naturally arises, how long should this time be? The answer involves the question, how long does it require for the disappearance of the inflammatory exudation into the myocardium and for the regeneration of muscle, nerve, and other tissues? Until this has occurred the possibility of danger must be constantly kept in view and carefully guarded against. The data for a satisfactory answer to the question are difficult to obtain. Regeneration in such highly-organized tissues as muscle and nerve are known to be slow. In the muscular paralysis following peripheral neuritis a year or more may be necessary, and there is no reason for believing that in the heart, an organ functionally more unfavourably circumstanced, the processes of repair will be more rapid. In cases, of course, where the changes affect the muscle only, the nerves not being seriously involved, the outlook is better. After a severe illness, however, the opinion may be ventured that probably a year will be required in favourable cases before the organ has regained its former strength.

In all these cases of acute infection, it cannot be too strongly argued that a broad view of the pathology of the disease is the only safe guide to an appreciation of the myocardial condition, even in the absence of all symptoms and physical signs of its involvement.

There are other important phases of this subject, such as the influence of muscular stress in valvular disease, with which one might deal, but their importance will have been suggested by the consideration of the points already discussed.

BIBLIOGRAPHY.

Hypertrophy from Overstrain, Virchow's Archiv, 1857. Myers, Diseases of the Heart among Soldiers, 1870. Allbutt, St. George's Hospital Reports, vol. v, 1870, and System of Medicine, 1898, vol. v, pp. 841 to 855. Da Costa, Toner Lecture, 1874; Amer. Jour. Med. Sci., 1871. Morse K. Taylor, Heart Strain in the Military Service, Handbook of the Med. Sci., 1816, vol. iii. Roy and Adams, Remarks on Failure of the Heart from Overstrain, BRITISH MEDICAL JOURNAL, December 15th and December 22nd, 1888. Sansom, The Diagnosis of Diseases of the Heart and Thoracic Aorta, 1892, pp. 36 and 45. Strumpell, Textbook of Medicine, 1887, p. 292. Fagge, Principles and Practice of Medicine, vol. ii, p. 39, 1891. Weber, Hygiene of Hill Climbing, Lancet, October 28th, 1893. Wilson, Disordered Heart in Young Soldiers, Army Medical Reports, 1896. Broadbent, Heart Disease, 1897, p. 79. Powell, Treatment in Diseases and Disorders of the Heart, 1899, p. 96, and Allbutt's System, vol. v, pp. 885 and 901. McCarthy, Functional Diseases of the Heart among Soldiers, Thesis for M. B. Degree Cambridge, 1898 (quoted in Allbutt's System). Gibson, Diseases of the Heart and Aorta, 1898 pp. 624 and 697. Stengel, The Immediate and Remote Effects of Athletics upon the Heart and Circulation. Von Leyden, Myocarditis, International Clinics, 1898, vol. 1, p. 105. Munson, Military Hygiene, 1901, pp. 35 and 92. Snerrington, Allbutt's System, vol. v, p. 464. Gardiner, Aneurysm of the Aorta, Allbutt's System, 1898, vol. vi, p. 351. Larrabee, Heart Strain, Handbook of the Med. Sci., 1902, vol. iv, p. 614. Curschmann, Nothnagel's System, 1901, vol. 1, p. 150. Camac, Johns Hopkins Bull., vol. xv, February, 1904.

AN INQUIRY INTO THE CAUSE OF ANGINA PECTORIS.

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For many years I have been noting carefully the individual symptoms that are present in cases of angina pectoris, in order to find the immediate cause of the symptoms included in this term and the mechanism by which they are produced. In this paper I deal only with the train of reasoning that leads towards the discovery of the immediate cause of angina pectoris, and at the same time I illustrate the field of inquiry that is opened up by the study of heart failure from the point of view of impairment of the individual function of the heart muscle fibre.

Definition of the Term "Angina Pectoris."

The term "angina pectoris" includes a number of distinct symptoms that are evoked by certain conditions of the heart. The symptoms included in this term may vary in number, in intensity, and (as in the case of pain) in situation. For the purpose of this study I limit my remarks to those cases that exhibit three of the most characteristic symptoms, namely, (1) pain situated over the region of the heart, (2) a sensation of tightness or constriction of the front of the chest, (3) a sensation of exhaustion referred to the chest, amounting at times to a feeling of impending dissolution.

The reason for the limitation of the term "angina pectoris" in this paper is to enable the argument to be presented clearly and definitely, by showing that those well-defined symptoms

* June 1st, 1905. This patient returned to her hospital duties some two months ago. She has been very careful to avoid excessive exertion, and so far has developed no bad symptoms.

may be associated with widely-divergent gross pathological changes. To enter into the consideration of other phases of angina pectoris would lead me beyond the point I want to elucidate here. Suffice it to say that the underlying principles on which this argument is based is applicable to all forms of angina pectoris.

Conditions in which Angina Pectoris is Produced.

In looking over my notes of a very large number of cases I find that the three symptoms mentioned may arise in patients with most diverse forms of lesion, and even in patients without any evidence of cardiac disease. From among a number of conditions I select the following six: Cases of (1) aortic aneurysm, (2) aortic valvular disease, (3) atheroma of the coronary arteries, (4) myocardial degeneration (fatty and fibrous), (5) increased arterial pressure of the elderly (usually associated with 3 and 4), and (6) arterial spasm of the young (vasomotor angina pectoris of Nothnagel).

It is manifest that identical symptoms produced from conditions so diverse cannot be directly due to the organic lesion, to the aortic aneurysm, to the disease of the aortic valves, to the atheroma of the coronary artery, or to the increased peripheral resistance. We must therefore look for a cause common to all six conditions.

Conditions Giving Rise to an Attack of Angina Pectoris.

In order to find what this common cause may be, consideration of the conditions giving rise to an attack of angina pectoris will help materially. In the first place, it is to be noted that angina pectoris in the six conditions cited appears as a late symptom, after the heart has been struggling a long time against obstacles opposed to its efficient action, or after the nutrition of the muscle has been impaired by gross pathological changes in the coronary artery or when the muscle fibres have become impaired through slowly advancing degeneration of the fibres. We find, further, that many patients when at rest do not suffer from pain, but that any cause inducing increased work on the heart provokes an attack. Bodily exertion in any form, excitement, increased peripheral resistance (as in exposure to cold air, a period of high arterial pressure in the elderly, spasm of the arterioles in the young) may bring on directly an attack of angina pectoris. That is to say, in predisposed individuals all circumstances that throw more work on the left ventricle induce an attack of angina pectoris.

Association of Angina Pectoris with Exhaustion of the Muscle of the Heart.

Such considerations lead to the conclusion that angina pectoris arises from certain conditions of the muscular substance of the heart, when the contraction meets with a resistance greater than it can easily and efficiently overcome, whether a fairly strong muscle struggles against an increased resistance (as when there is great peripheral resistance or a narrowed aortic orifice) or when a weak or degenerated muscle has opposed to its contraction a normal or even a lowered pressure, but a pressure greater than the weakened muscle can readily overcome.

Association of Angina Pectoris with Impairment of the Function of Contractility.

If we inquire into the question of what function of the muscle fibres is concerned in the production of angina pectoris we shall find additional confirmation for the foregoing conclusion.

I have already called attention in the BRITISH MEDICAL JOURNAL to the fact that affections of the various functions of the muscle fibres (Gaskell's functions) can be demonstrated by means of graphic records of the cardiac movements. I have taken a large number of tracings from patients who have suffered from angina pectoris, during the attacks and when free from pain, and an analysis of these tracings enables me to say with confidence that angina pectoris can occur when the excitability, the conductivity, and the power to produce a rhythmical stimulus are unimpaired. There only remain now the functions of tonicity and contractility. The evidences of failure of the function of tonicity have not so far been analysed, and I am at present engaged on this subject. I have specially studied the matter in connexion with angina pectoris, and I can state with confidence that angina pectoris may occur without any evidence of the impairment of the function of tonicity. Seeing, then, that angina pectoris can occur in patients when four out of five functions of the heart muscle are demonstrably intact, we are forced to the con-

clusion that angina pectoris is an evidence of the impairment of the remaining function, that is, contractility. When we come to look for facts in support of this view we find a three-fold evidence: (a) An *a priori* evidence in the fact that this is the function directly concerned in supplying the motive force to the circulation of the blood, and that it is the function that will necessarily become exhausted when an excessive resistance is opposed to the contraction of the heart muscle; (b) the symptoms associated with and the conditions giving rise to the characteristic sign of depressed contractility (pulsus alternans) are of a similar nature to the symptoms in angina pectoris; (c) angina pectoris may be associated with the pulsus alternans.

The Pulsus Alternans a Sign of Depressed Contractility.

The form of irregularity to which the term "pulsus alternans" is applied is where a large beat regularly alternates with a small beat, the beats, however, appearing at regular intervals—that is, the pulse may be perfectly regular in rhythm, but every second beat varies in strength, as in Figs 1 and 2.

Fig. 1.—Simultaneous tracings of jugular and radial pulses during an attack of angina pectoris. The radial pulse shows a regular rhythm in regard to time, but there is an alternation in the sizes of the beats—a small beat regularly following a large—the pulsus alternans.

Fig. 2.—Tracing of the radial pulse showing the pulsus alternans. From a case of high arterial pressure during an attack of angina pectoris.

Sometimes there may be a slight delay in the appearance of the smaller beat. This regularity in the rhythm or with a slight delay in the appearance of the smaller beat distinguishes it sharply from a more common form of irregularity—the so-called pulsus bigeminus, where the smaller beat is due to an extra systole, and appears at a shorter interval after the large beat (Fig. 3).

Fig. 3.—Bigeminal pulse. The spaces between the larger and smaller beats is less than the spaces between the smaller and greater beats, in contrast with the regular rhythm shown in Figs. 1 and 2.

The alternating action of the heart which produces the pulsus alternans has been explained by F. B. Hoffmann as being due to an impairment of the function of contractility, and is thus described by Wenckebach: "When the contractility is lowered, the contraction is made more quickly than the normal one—that is, it does not last so long, and this is quite independent of the manner in which the contractility of the heart was reduced (say, by high frequency or by stimulation of the vagus). In this condition both systole and diastole are shorter; the cardiac muscle sooner reaches the maximum of its contraction, and sooner returns into a position of rest. Hence the pauses between each pair of beats are longer. If now one period becomes a very little longer from some cause or other (by changing the frequency once, for example, as in Hoffmann's experiments), the next systole will be a little larger by reason of the longer rest

which the heart obtained. But, on account of the greater length of this big contraction, it will be followed by a somewhat shorter pause, even if the heart returns again to its normal rhythm at once. The next systole will therefore be smaller; but as it lasts a shorter time, it is followed again by a longer pause, and the succeeding systole will again be larger."

Although the pulsus alternans has been demonstrated to be a symptom of impaired contractility, it does not follow that this function is intact in the absence of the pulsus alternans. Patients may die of depression of contractility, and this alternating action of the heart never appear. Its appearance may be very transient, due to some temporary cause. Fig. 1 was obtained from a patient immediately after exertion, and with the symptoms present of angina pectoris. As soon as I had taken the tracing the pulse became perfectly regular, though the other symptoms of cardiac exhaustion persisted. In the quotation from Wenckebach's book just given, it is stated that in experiments by changing once the frequency of the heart's action the alternating rhythm may be started. This is well seen in Figs. 4 and 5, where, after a long pause—due probably to a ventricular extra-systole—the alternating rhythm is started and continued for a brief period.

Fig. 4.—Tracing of radial pulse showing the appearance of the pulsus alternans after a long pause. From a patient suffering from angina pectoris.

Fig. 5.—Showing the same thing as Fig. 4. From a patient aged 72 suffering from great exhaustion after an attack of bronchitis.

It may be asserted that while the absence of the pulsus alternans must not be taken as evidence of the integrity of the function of contractility, its presence is, on the other hand, a distinct sign of the impairment of this function.

Symptoms Associated with the Pulsus Alternans.

All patients who exhibit this form of heart action invariably show a great limitation of the field of cardiac response; that is to say, while at rest the heart may seem to be doing its work efficiently, very slight exertion will at once produce symptoms of cardiac distress.

Conditions giving rise to the Pulsus Alternans.

When we inquire into the conditions in which this symptom appears, we find a striking resemblance to the conditions that provoke an attack of angina pectoris. Thus, I have found it appear for a short time in a patient with an enfeebled heart after walking hurriedly (Fig. 1). I have found it in several cases when the heart was exhausted after struggling against a very high arterial pressure (Figs. 2 and 4). I have found it in a patient after a long attack of paroxysmal tachycardia associated with profound exhaustion; and Dr. John Hay has shown me tracings of the pulsus alternans from a similar case. Wenckebach mentions that Huber had also found the pulsus alternans in the course of an attack of paroxysmal tachycardia. I have also found it in a youth with acute dilatation of the heart a few days before death, and in a female aged 72 after an attack of bronchitis (Fig. 5). In the record of two cases by Wenckebach and of a case by Volhardt the pulsus alternans was associated with other evidences of great exhaustion of the heart muscle.

Association of Angina Pectoris with the Pulsus Alternans.

I am confident that if tracings were systematically taken of the radial pulse in cases showing the symptoms of angina pectoris the pulsus alternans would be found fairly frequently.

It is not easy to detect it with the finger or by the heart sounds, though when the attention is specially directed to the matter it may be recognized. In this way further evidence of the association of angina pectoris with exhausted contractility will be found. In five of my cases of pulsus alternans the symptoms mentioned as characteristic of angina pectoris were present. In six other cases the pain was absent, but the symptoms of exhaustion, shortness of breath on exertion, and general distress, showed that practically the symptoms were identical with those of angina pectoris, minus the pain.

The Arterial Pressure is not a Guide to the Condition of the Contractility.

It might seem at first sight that the condition of the arterial pressure would be a guide to the state of the function of contractility. As a matter of fact it does not serve as a guide, as the pulsus alternans may be present in cases with an abnormally high pressure, as well as with a very low pressure. In the case of a man whose heart had been exhausted by lifting very heavy weights, and who suffered from attacks of angina pectoris with the alternating rhythm, the blood pressure was 120 mm. Hg, as measured by Martin's modification of the Riva-Rocci instrument. The pulse of the patient when Fig. 1 was taken was extremely soft and compressible. The pulse in the patient from whom Fig. 2 was taken was very hard and resistant, and the arterial pressure equalled 210 mm. Hg. When the pressure fell to 170 mm., the pulsus alternans and all anginal symptoms disappeared. In this case (and in that from whom Fig. 4 was taken) the association of the pulsus alternans and angina pectoris with the high arterial pressure, and the disappearance of both phenomena when the pressure was lowered, demonstrated the intimate connexion between the exhausted function of contractility and the symptom of angina pectoris.

Conclusion.

From the consideration of the fact that angina pectoris occurs in lesions of great diversity, some condition common to all must be the cause of the symptoms. The fact that angina pectoris appears only after the heart muscle has been long exposed to excessive strain points to the cause being situated in the muscle. All the functions of the muscle fibres save that of contractility can be shown to be intact in many cases that suffer from angina pectoris. The alternating action of the heart is a demonstrable sign of exhausted contractility, and its presence is always associated with symptoms that are included in the symptom complex of angina pectoris. The same exciting cause—extra strain on the heart—may provoke both the angina pectoris and the alternating action, and both may disappear with removal of the cause. The inference to be drawn from the consideration of these facts is that the symptoms that are included in the term "angina pectoris" are so closely associated with an impairment of the function of contractility of the muscle fibres of the heart that in all probability angina pectoris will be found to be an evidence of the impairment of the function of contractility.

A CASE OF SYNCOPAL BRADYCARDIA SHOWING THE INDEPENDENT ACTION OF THE TWO SIDES OF THE HEART.

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Cases of persistent bradycardia are still sufficiently rare to justify their being reported when an opportunity has occurred for their more or less continuous observation; in the following case, moreover, there were certain clinical signs which appear to invest it with additional interest:

History.

On November 24th, 1904, I was consulted by Mr. P., aged 57, whose occupation involved a good deal of standing, but without any violent exertion. He stated that two years before he had felt giddy, and fainted for a few seconds; he was seen by a medical man, who remarked on the slowness of his pulse, but said his heart was otherwise normal for his age; from that date till six weeks before he came to me he had no return of the symptoms, and there is no evidence as to the state of his pulse in the interval. For a month or six weeks he had been suffering from palpitation, shortness of breath and giddiness, and on the morning of