drugs in use for the treatment of virulent rheumatism in childhood are no more specific than the drugs that are used in the treatment of acute tuberculosis, and no more effectual. There is much to be done for a child with valvular disease, much that will help him. Yet it is at the best but a treatment of the third order of merit, for if the first is prevention, and the second cure, this is but palliative.

STRAIN AS A FACTOR IN CARDIO-AORTIC LESIONS.

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THE clearest conception of the influence of mechanical stress *per se* in the causation of cardio-aortic lesions is obviously to be obtained by the investigation of cases wherein all other factors can be eliminated. For this reason the effects of strain have been studied, especially in soldiers, athletes, and others, who, by reason of occupation or practice, are subjected to severe sudden exertion or to prolonged over-exertion at a time when the general health is good and the heart normal. Thus Larrabee defines heart-strain as "the untoward effects of over-exertion in the previously normal heart," and Allbutt says, "when we enter upon a discussion of strain of the heart, we are understood to refer to the effects of muscular exertion upon hearts which, before the strain, were either strictly or virtually sound." In other words, the influence of strain has been considered chiefly with reference to persons whose physical condition renders them least liable to show its ill-effects. From a systematic standpoint, the advantages of approaching the subject in this way are readily apparent. In fact, it seems the only way of appreciating its importance as a single, determining, etiological factor. It would also seem a very obvious conclusion that if over-exertion is capable of doing injury to normal structures, much more readily will its untoward effects be produced when these structures are unsound; yet, paradoxical as the statement may appear, the consideration of strain from the view point of the healthy and vigorous has undoubtedly led to an under-estimation of its power to do harm in cases where the myocardium or aorta is already weakened by age or disease. The morbid opportunity offered under these circumstances not only allows of the more ready production of injury by over-exertion, but, what is even more important to the clinician, degrees of strain borne with impunity by the healthy structures may here become factors of the most serious significance. It is the influence of these lesser degrees of strain acting on structures already weakened that is often not fully appreciated. For this reason, it would be better from the clinical standpoint to consider strain in a relative rather than in an absolute sense, to indicate an increase in intracardial or intra-aortic pressure, due to unusual or controllable conditions, out of proportion to the capacity of the tissues concerned, to withstand it. The qualifying phrase "unusual or controllable" is used in order to limit the term "strain" so as to exclude the pressure which is normally incident to the function of the heart and aorta under the most favourable circumstances. I wish to emphasize, however, that to grasp the practical significance of strain it is even more important to appreciate the condition of the tissues exposed than the absolute amount of violence to which they are subjected.

Perhaps another reason for the failure to fully apprehend the ill-effects of strain on the cardio-aortic tissue is due to the general tendency in the past to rely too much on the stethoscope for the detection of cardiac disease. Useful as the stethoscope may be in the recognition of valvular lesions, it is, unfortunately, of very limited service in detecting the much more important myocardial changes. Myocardial changes are rather to be suspected from a knowledge of their association with certain general diseases or of their being the result of certain causes. Therefore a broad view of the pathology of any particular case is a more reliable guide to the recognition of myocardial changes than stethoscopic or other form of physical examination, especially at an early stage, before hypertrophy or dilatation have occurred. This applies particularly to the appreciation of the myocardial trouble associated with the acute infective diseases.

The capacity of the cardio-vascular system, when sound, to withstand severe mechanical stress is very great. Thus Löwy has shown that the heart when exerted to its uttermost is capable of doing thirteen times as much work as when the

body is at rest; and Sherrington states that rupturing strains of the healthy arteries require nearly twenty times as much strain as the body can possibly exert upon them. I can find no experimental data giving any definite statement of the increase of pressure in the chambers of the heart or aorta during severe exertion. An investigation to ascertain this point, desirable as it is, would be beset with great difficulties. Clinical observation has abundantly shown, however, that severe or even irreparable damage may result; and bearing in mind the great reserve strength of these structures in health, the increase of pressure necessary to produce such damage must indeed be enormous. The effect of such degrees of stress on weakened structures may *a priori* be readily imagined.

But apart from this we need only recall how frequently we observe the insufficiency of the cardiac muscle to withstand the requirements of ordinary quiet life, that in consequence dilatation is a common, and rupture not a very rare event, to understand how powerful a cause of mischief severe exertion may be.

Having in mind these well-established facts, it seems remarkable that in special treatises on diseases of the heart, and in systems or textbooks of medicine, the influence of strain is frequently disposed of in a few words, merely mentioned incidentally or in some cases ignored. The relatively unimportant place thus often assigned to this factor in dealing systematically with etiology of cardio-vascular diseases, does not indicate an adequate appreciation of the potency of strain to cause cardiac mischief in the previously healthy, as shown by many contributions by competent observers, to be mentioned later; nor is it in keeping with the frequency with which its influence is seen in the routine of practice. The unrestrained athletics often permitted in schools and colleges, contests or undertakings requiring strength and endurance, entered into by the young, weakly, or untrained, or what is even worse, misguided exercise, laborious holidays, or over-taxation incident to occupation (occasional or continuous) in the sedentary and out-of-trained at a later period of life, or after convalescence from acute infections, all go to show that the possibilities of harm from muscular stress are not generally appreciated. I wish, therefore, within the compass of this paper to deal—of necessity briefly—with the physiological and pathological phenomena of severe exertion with reference to the heart and aorta, to review in a fragmentary way the literature of the subject, to relate a number of illustrative cases which have come within my personal experience or that of some of my colleagues, and to attempt to outline some special circumstances wherein it appears to me the importance of mechanical stress is not kept definitely enough in view in the guidance of the healthy, nor in the management of the sick and convalescent.

The work of the heart is represented by the amount of blood expelled from its chambers during contraction and the resistance to be overcome in forcing it through the peripheral circulation. The energy exerted by the heart during contraction is in part changed into kinetic energy, represented by the momentum imparted to the blood stream, and in part into potential energy stored up as tension by the dilatation of the elastic walls of the arteries. The amount of the latter will depend on the elasticity of the vessel walls. At the beginning of exertion there is a rise of arterial pressure due to compression of the intramuscular vessels by the contracting muscles and to an increased output by the left ventricle. The left ventricle, therefore, has to expel a larger amount of blood against increased pressure. Under the stress of severe exertion the ventricles are unable to empty themselves as completely as under ordinary conditions, so there is a resulting increase of residual blood within them which tends to over-distension, and from this cause also there is increased stress thrown on the muscular walls. The initial effect of exertion is, therefore, to throw increased work on the left ventricle for three reasons, namely, (1) from increased amount of blood to be expelled, (2) from distension of the cavity, and (3) from increased peripheral resistance. It will, therefore, be readily understood that the brunt of sudden exertion and the primary effect of prolonged exertion is borne by the left side of the heart and arterial system. The initial rise of pressure is of short duration. Owing to reflex influences from the muscles, and possibly to the action of fatigue products on the vaso-dilators, dilatation of the intramuscular vessels soon takes place; the cutaneous vessels also dilate, as shown by reddening of the surface and sweating; and the more active respiratory movements increase

the suction action of the right side of the heart. These factors all tend to a fall of arterial pressure and a flooding of the veins and right side of the heart. In all cases, therefore, of prolonged exertion increased strain is secondarily thrown on the right side of the heart, which, especially if emphysema or other cause of obstruction to the pulmonary circulation exist, may be seriously or hopelessly strained in its efforts to maintain the onward flow of the blood. On account of its thinner walls the right ventricle is less able to accommodate itself to the increased stress.

The effect of repeated or habitual exertion, within the limits of the heart's resistance, as in proper athletic training and occupations attended by muscular exertion, is to produce a compensatory hypertrophy of both sides of the organ, with some enlargement of its cavities. This must be regarded as a perfectly physiological and conservative process, analogous to the hypertrophy of the skeletal muscles under systematic exercise, and it affords a ready explanation of the value of proper training in increasing the staying power of the heart in severe or prolonged exertion.

It is also important to note that the observations of Allbutt and others go to show that while the initial effect of exertion is to raise the arterial blood pressure, habitual exercise lowers the mean arterial pressure. Consequently, when at rest, the blood pressure of athletes and others habituated to proper exertion is found to be lower than that of persons leading sedentary lives. Athletic and other exercises, therefore, when properly regulated, are beneficial to the heart by producing physiological hypertrophy of the organ and lowering the average of peripheral resistance.

Having dealt briefly with the physiological mechanisms by which the cardio-vascular system reacts to strain, we are now in a position to deal with cases where, owing to excess of effort or defect in the tissues, the reserve strength or regulative mechanisms are unable to adapt themselves to demands out of proportion to the capacity of the myocardium to bear them without suffering injury.

From the experimental side, Roy and Adami have shown that overstrain, produced artificially by narrowing the ascending aorta by ligature, is capable of causing not only valvular incompetence but organic changes in the flaps. The latter consisted of oedematous swelling, subendothelial haemorrhages, opacities, etc., occurring at the margins of the valves. These experimental findings, especially their situation, are suggestive of the results on the valves of the high blood-pressure incident to many cases of arterio-sclerosis, and of the influence of repeated mechanical stress in initiating slowly-developing organic mischief, as in sclerotic changes in the valves.

From the clinical side, we are indebted to the military surgeons for first directing attention to and carefully investigating the effects of over-exertion on the heart. As late as 1871 the lack of literature on the subject was referred to by Allbutt in the statement "that the mechanical causation of heart disease is either omitted or treated in a way so meagre, by both English and foreign writers, as to be worthless." During the early part of the American Civil War so many cases presenting symptoms of cardiac trouble developed in the otherwise healthy soldiers, that an investigation of the subject was undertaken by Morse K. Taylor and Da Costa, and their observations were published from 1864 to 1871. Myers, of the Coldstream Guards, published in 1870 an article on Diseases of the Heart among Soldiers from observations made in the British service; while Frantzel, in 1873, reported the results of some of his observations during the Franco-Prussian War. Morgan, and especially Allbutt, were the earliest investigators of this factor as seen in civil life, the latter publishing his observations in *St. George's Hospital Reports* in 1871. Since that time the subject has been studied in the military service and civil life by Herz, Wilson, McCarthy, Treadwell, Stengel, Larrabee, Darling, Williams and Arnold, Balfour, Powell, Weber, Schott, and others.

Among soldiers, certain contributory factors appear to increase the liability to injury from overstrain. In this connexion the wearing of closely-fitting uniforms and bearing heavy accoutrements, by interfering with respiratory movements and enforcing strained positions; the mental stress incident to battles; the proneness of soldiers to excesses in alcohol and tobacco; and the frequency of syphilitic infection, have all been mentioned. Both Da Costa and Taylor emphasized the influence of diarrhoea, dysentery, and typhoid fever as predisposing causes. Apart from such possible modifying conditions in the soldiers' surroundings, the influence of over-exertion in civil and military life is to

produce similar effects on the cardio-vascular system. Different observers have noted that exercises in which the legs were employed were especially likely to be associated with cardiac trouble. The explanation of this is stated to be the greater formation of fatigue products where the legs are used owing to the greater bulk of muscular tissue involved.

The symptoms of overstrain described by different observers, as well as their conclusions, are remarkably in accord. Cases in which definite symptoms arise may be divided into acute and chronic. The acute form is most often seen as the result of severe or prolonged exertion in the young, weakly, or untrained. The subject shows symptoms of dyspnoea, precordial or epigastric distress, is pale or cyanosed, dizzy and faint, and may fall to the ground. Vomiting may occur. The pulse is rapid, weak, and may be irregular or intermittent. The transverse cardiac dullness is increased, the impulse faint, and the apex beat somewhat out. The termination of these cases varies. Usually the symptoms soon pass off, without evidence of any permanent injury. As already explained, the brunt of the injury is borne by the right side of the heart; and Taylor states that the damage done in the first instance will depend on the degree of dilatation and the length of time the organ is subjected to the distending force. "In some instances, sudden death takes place, and autopsy reveals marked dilatation of the right side of the heart and engorgement of the venous system." In other cases a condition termed "irritable heart" remains, sometimes permanently. Allbutt classifies the "irritable heart" among the functional cardiac disorders, though as seen in soldiers and in the later years of life the difference between it and the more definite organic disability of heart-strain is only one of degree. It is more commonly the result of repeated over-exertion, coming on gradually, than the sequence of the acute dilatation just described. The form appearing in the young, due to the restless activity of youth, Allbutt says is a very recoverable condition.

In the irritable heart there is an incapacity for exertion, necessitating the avoidance of all but the mildest forms of exercise. In the more pronounced cases the patient's "staying power" is permanently gone. Under favourable conditions, in the cases just mentioned, compensatory hypertrophy develops after some time, which may enable the patient to attend to the ordinary affairs of life without discomfort.

Chronic heart strain presents in an aggravated degree the symptoms already described in the unrecoverable cases of irritable heart, with more definite evidence of myocardial changes, with failing compensation, and perhaps valvular lesions eventually supervening. Myers says that recovery never takes place in these cases, and Taylor takes practically the same view. The latter states that if compensatory hypertrophy develops in from four to six months, it is a good recovery—that is, a recovery so far as to enable the patient to attend to the ordinary affairs of life without discomfort. Da Costa, in 300 cases of all degrees of severity from the irritable heart to the more pronounced condition, had 38 per cent. of permanent recoveries. The subsequent history of the recovered cases, however, is not given, or perhaps they would show a considerable percentage of cases of cardiac breakdown at a later period.

In addition to injury to the myocardium, mechanical stress is a well recognized cause of aortic valvular lesions. While more likely to cause injury where the valves are already diseased, still it may damage these structures when sound. The influence of muscular stress in this connexion was noted by Morgagni, and more recently by Peacock, Barr, Seitz, Allbutt, and many others. Peacock in his Croonian Lectures related 17 cases in which rupture of an aortic segment resulted from muscular strain. The following cases are instructive examples of such results.

CASE I.

For the notes of the first case I am indebted to Dr. H. A. McCullough of Toronto.

C. R., aged 19, a healthy, robust farmer's son, at the end of a day's threshing, as a feat of strength, undertook to lift the wheel of the threshing engine. Suddenly he felt something snap in his chest. He had to be carried home, and for six weeks was confined to bed, being quite unable to walk, from breathlessness and weakness. A double aortic murmur developed after the injury, with collapsing arteries and other evidences of aortic incompetency. He never regained strength. After some time, dropsy and other signs of cardiac failure developed, from which he died in three years. No autopsy was made, but the history of the case points definitely to rupture of an aortic segment with subsequent regurgitation and failure of compensation.

CASE II

The second case occurred in the practice of Dr. J. L. Davison

Toronto. The patient was a vigorous man, of about 55 years of age, a captain on a lake steamer. During the fall of 1900, while his vessel was being loaded with lumber at Manitowaning, he became enraged at the slowness of the men engaged at the work. In order to encourage them by example, he picked up a scantling himself, placed it on his shoulder and began to walk through the sand towards the vessel. He had only gone a short distance when he experienced a severe pain in the chest, with the sensation of something having given way. He fell to the ground in a faint, and had to be carried to his room. In a few days he was brought to Toronto, where he placed himself under Dr. Davison's care. He was quite unable to endure any exertion, which would immediately produce extreme rapidity of the heart and dyspnoea. During the winter, notwithstanding the greatest care, he had recurring attacks of syncope, with dyspnoea, tachycardia, and precordial distress. In the following spring, eight months after the injury, he died during one of these attacks. At the autopsy, I found a rupture of one of the segments of the aortic valve, with the formation of a small aneurysmal dilatation in the sinus of Valsalva behind it (Fig. 1). There was no marked

Fig. 1.—Ruptured aortic segment with protrusion of sinus of Valsalva.

evidence of arterial sclerosis or other valvular disease. The patient had never suffered from syphilis, nor was he given to alcoholic excess, and up to the time of the accident was in good health.

This is a striking example of an acute rupture in one previously healthy. Ailbutt mentions two other classes of cases in which forcing of the aortic segments results from over-strain—namely, first, “chronic forcing of a valve previously weakened by some constitutional poison such as syphilis,” and second, “chronic forcing of the valves by the importunity of repeated strains, none of which was sufficient to break down a healthy valve, but all of which, by molecular rather than massive strain, contributed gradually to increase valvulitis and to break down the resistance of the part.” An example of the former recently came under my observation in the Toronto General Hospital. A longshoreman, of excellent physique, and previously healthy, had been infected with syphilis a few months before. After a night's debauch, on attempting to work the next day, he was taken ill and brought to the hospital, with the most pronounced signs of aortic incompetency, which has since quite incapacitated him from work. He had never had rheumatism or other acute disease.

Two other cases which I shall relate show that parts of the aorta other than the valves may suffer.

CASE III.

This case I am permitted to report through the courtesy of Dr. N. A. Powell of Toronto. A young man, aged 24 years, of rather delicate habit, a photographer, had suffered from syphilis a few years before. He had shown no previous evidence of cardiac trouble. One evening he walked briskly about half a mile to call on a young woman. Arriving at the house he felt faint, and asked for a glass of water. As it was being handed to him, he fell forward on the floor and died instantly. Autopsy revealed an irregular Y-shaped rupture of the ascending aorta, a short distance above the aortic orifice (Fig. 2). The vessel appears of about normal thickness, but shows slight irregularity of the intima, probably from degenerative changes due to the syphilis. The early age of the patient and the slight strain, if such, indeed, it may be called, due to the walking, are interesting features of this case.

CASE IV.

This case occurred in a financier, of about 58 years of age. One of his brothers had died from aneurysm. The patient was short in stature and rather obese. He had been in usual health, and on the day of his death had come from Montreal to visit his brother in Toronto. Arriving at his brother's residence, he had ascended the steps, entered the house

and was just in the act of taking off his overcoat, when he was suddenly seized with pain in the chest and faintness. He was put to bed and Drs. Peters and Davison were sent for. When they arrived, they found him in a collapsed condition, pale and with a weak and rapid pulse. They diagnosed rupture of an aneurysm. Restoratives were used without

Fig. 2.—Y-shaped rupture of aorta. Patient aged 24 years.

effect beyond slightly rallying. About four hours after the onset of symptoms, the patient's condition having remained about the same, the nurse had gone out of the room for a moment. Hearing a noise, she immediately returned to find him dead. I made an autopsy next day. The body was that of a large, well-developed man. There was a great deal of subcutaneous, omental and mesenteric fat. The arteries showed well-marked diffuse sclerosis, the kidneys showed interstitial nephritis. Death was due to an irregular rupture of the aorta, $1\frac{1}{2}$ in. in length, 3 in. above the valve (Fig. 3). The rent, which was irregularly trans-

Fig. 3.—Rupture of aorta, with formation of dissecting aneurysm.

verse in direction, had gone through the intima and media and this was followed by the formation of a dissecting aneurysm, which had separated the media from the adventitia as far as the iliac vessels below, and backwards towards the heart, beneath the reflection of the pericardium over the first part of the aorta. The aneurysm had then ruptured through the adventitia into the pericardial cavity, which was found distended with blood. The secondary rupture had evidently been the immediate cause of death. The blood between the adventitia and media had pressed the latter inwards so as to indent it into the lumen of the vessel and so greatly reduced its calibre. The aorta was abnormally thin, and the intima showed extensive degenerative changes. The vessel was not pouched at the seat of rupture, nor was the site of the rent apparently more diseased than other parts of the vessel. The patient had been a moderate drinker; he had never had syphilis.

In this case the important factor was obviously the advanced degenerative change in the walls of the vessel. The over-exertion was so slight as to scarcely warrant the term strain. The general arterio-sclerosis and marked obesity, however, were undoubtedly causes of chronic strain.

CASE V.

Another case, for the notes of which I am indebted to Dr. A. H. Garratt of Toronto, occurred in a stockbroker, 54 years of age. No history of rheumatism, syphilis, or other serious disease; no previous cardiac trouble. Patient was in his usual health, and was coming down in an elevator. As he was getting out the boy suddenly started the elevator, and the man was forced to make a sudden violent effort to escape being caught between the cage and the walls of the shaft. He felt faint and ill, and the doctor was immediately sent for. The pulse was rapid and weak, and continued so throughout his illness. No cardiac murmurs were present at any time. Obscure cardiac symptoms continued, and he died in four weeks. Autopsy showed a moderate degree of atheroma and a sacculated protrusion of the wall at the base of an atheromatous ulcer $\frac{1}{4}$ in. above the valve.

I shall not here enter upon a discussion of the generally-recognized influence of strain in the etiology of atheroma of the aorta, nor of the part which it plays in the causation of aneurysms, by producing rents in the media, with consequent weakening of the wall, tending to protrusion. In these cases strain acts in association with other causes, and it is difficult to apportion proper relative weight to each.

In lower animals severe exertion at times is a cause of cardio-aortic lesions, in some cases leading to broken wind, in others to rupture. Two cases of rupture of the heart have come under my notice—one in a valuable young stallion, while mounting a mare, where the right heart ruptured, and one in a cavalry horse at the Niagara military camp during the past summer, when the right heart ruptured during the exercises.

In youth and old age the influence of strain presents such important differences from those which occur during the vigorous period of early manhood and middle life as to call for special consideration.

In youth the processes of waste and repair are more rapid, the vessels more elastic, and the nervous mechanism governing them more sensitive. Youths consequently bear severe stress of short duration better than prolonged exertion. The latter, by reason of the rapid waste and the influence of fatigue products, soon leads to exhaustion. The nervous apparatus readily influences the elastic vessels, so that the initial rise of pressure is very brief, consequently the heart is soon relieved of strain, and therefore seldom suffers. Where the exertion is more prolonged, however, the stress falls on the abnormally thin walls of the right heart, as already explained, and this may be severely damaged. The hearts of youths are, therefore, ill adapted to withstand trials of endurance or prolonged stress. Sir Douglas Powell has called special attention to the unsuitability of paper chases and similar strenuous sports calling for staying power beyond the strength of early life, and warns of the irreparable cardiac damage which may result from unwise indulgence in them.

In old age we have to bear in mind not only the gradual lessening of general nutrition and the degenerations in the heart and vessels normally incident to that period but also the frequent occurrence of premature advanced arterio-sclerosis with secondary myocardial involvement. The heart is weaker, the arteries more rigid, and the nervous mechanism perhaps less sensitive than normal. As before stated, the work of the heart is expended partly as kinetic energy in imparting momentum to the blood stream and partly as potential energy, stored up in the elastic walls of the vessels. As the elasticity of the vessels is lessened there is a corresponding loss of potential energy to assist in maintaining the arterial flow. Strain under these circumstances, therefore, means that a weakened heart, with part of its energy (potential) lost, has to work against increased peripheral resistance, and that, owing to the rigidity of the vessel walls and reduced sensitiveness of the vaso-motor nerves, the initial rise of pressure due to exertion is longer maintained. It will therefore be readily understood why the left heart and aorta, under such unfavourable conditions, are especially exposed to injury from sudden strain or at the beginning of prolonged strain. Clinical observations substantiate these statements. Dilated left heart and ruptured valves of aorta are not uncommon results of misguided exertion in the old. Some striking examples of this have come under my observation during the past few years.

CASE VI.

In one case, a patient of Dr. Bowles of Woodhill, a vigorous farmer, 50 years of age, who had never been seriously ill, in January, 1904, spent some hours turning a sausage machine. He stated that he had

worked very hard, sweat freely, and when through was very tired. He was referred to me some three months and a half later. He complained of feeling weak, with numbness in various parts, dizziness, and was unable to withstand exertion. He suffered from indigestion, with occasional nausea, had lost considerable weight, and felt miserable and depressed. He also complained of pain in the chest. These symptoms he definitely fixed as following the work before mentioned. Physical examination showed considerable sclerosis of the peripheral vessels; the apex beat was in the fifth space, three-quarters of an inch outside the nipple, and was weak and diffuse. There were no murmurs. The pulse was regular, and only slightly rapid. Examination of the urine showed increased quantity, specific gravity 1009, urea 6 gr. to the oz., phosphates reduced one-half, a trace of albumen, and excess of indican. A few hyaline casts and cylindroids were found. I considered the case to be one of heart strain, due to severe exertion in a man suffering from arterio-sclerosis with interstitial nephritis. With careful regulation of diet and exercises, baths, and cardiac tonics, all his urgent symptoms have disappeared, and he feels very well, but he has to avoid all severe exertion.

CASE VII.

Another case occurred in a farmer, aged 70, who had never shown any evidence of cardiac trouble, and was unusually vigorous for his time of life. One day in April, 1903, he chopped down a tree, and, with the assistance of a neighbour, sawed it into blocks. He then felt sufficiently well to undertake splitting the blocks, but before he had finished he was obliged to give up from exhaustion. He was weak, faint, and complained of pain in the chest. He went to bed, oedema of the extremities soon developed, and he died in less than a month with all the evidences of dilated heart.

This was an example of severe muscular exertion overtaxing and producing dilatation in a senile heart, which until the time of the overstrain had been able to meet the requirements of ordinary life.

CASE VIII.

Another instance was in a gentleman, aged 63 years, who had always taken good care of himself, was never used to severe exertion, and had never suffered from any severe illness. For the year and a half previous to the occurrence now to be related he had been subjected to unusual worry. In April, 1899, he left Toronto in good health to undertake some business in the country. In order to reach home on a certain Saturday, he hurriedly walked 10 miles on Friday afternoon and drove 30 miles to the railway station (Peterborough) the next morning. When he reached the town he felt so weak and breathless that he consulted a physician, Dr. Scott, who prescribed for him and put him on the train for Toronto. He was unable to walk up the steps to the doctor's office. When he reached Toronto, some 80 miles, he was weak, breathless and only semi-conscious. A fellow-traveller helped him to a carriage, and he was driven to his home. I saw him shortly afterwards and found him panting for breath, deeply cyanosed, voice weak and tremulous, and he was unable to express himself intelligibly. The pulse was running about 200, and was weak, irregular and intermittent. The cardiac impulse was weak and fluttering, and the whole precordium appeared throbbing weakly and irregularly. The transverse diameter of cardiac dullness was increased both to the right and left. No murmurs were present, but the first sound at the apex was valvular and the second pulmonic greatly accentuated. He became profoundly unconscious, Cheyne-Stokes breathing and occasional attacks of "goose-respiration" with a peculiar hissing expiration, developed, and continued for weeks. No oedema ever developed. Urinalysis excluded any definite renal trouble. In this condition he hovered between life and death for weeks. Thrombosis occurred in the right brachial artery, the pulse disappearing, and the extremity becoming cold. After some days, however, collateral circulation was re-established and the arm became warmer, though the pulse never returned. In a couple of months he began to show marked loss of flesh, and this continued, the weight dropping from 180 to 120 lb. After about two months he began to show slight signs of improvement, and in six months he was able to be about a little. He was, however, weak, nervous, very emotional and depressed, and was unable to withstand the slightest exertion or excitement. The pulse continued irregular and intermittent, was rarely below 120, and on the slightest provocation would become uncountable. After a year he had improved sufficiently to enable him to enjoy the quietest sort of life. He lived on the narrowest margin for four and a half years, and finally died from cardiac failure following a slight attack of bronchitis.

There seemed no human reason why this man might not have lived for years had a senile heart not been overtaxed by unwarranted exertion. These cases, to my mind, show the extreme danger in advancing years from exertion much beyond the ordinary, especially if prolonged, even though, considered in an absolute sense, the exertion may not have been very severe, but such as would have been borne in early manhood without the slightest harm.

The specimen of ruptured left ventricle shown in Fig. 4 illustrates, so far as I can learn, a unique though quite intelligible result of the strain thrown on the heart during the passage of a stomach tube. The patient was an extremely anaemic woman of 60 years of age, who had consulted a physician in reference to some stomach trouble. During the passage of a tube, while investigating the case, the patient was noticed to become slightly cyanosed, then pale, and before the tube was withdrawn she was dead. I was asked to make

an autopsy by the coroner, Dr. W. J. Greig. Examination showed a rupture of the left ventricle, near the ventricular septum, about midway between the base and the apex. The myocardium was very pale, soft, friable, and fatty. There was no valvular disease, but slight atheroma of the aorta and the orifices of the coronary arteries were not involved, though there is a fair amount of sclerosis along their course and some thrombosis near the beginning of the right coronary.

I have already mentioned incidentally, in connexion with the case of dissecting aneurysm, the tendency to chronic heart strain in cases of obesity, but wish now to deal with the subject a little more in detail. The occurrence of breathlessness in the corpulent, on slight exertion, is an everyday experience. In the absence of any valvular lesion this is commonly attributed to degenerative changes in the myocardium, or to the mechanical interference with the action of the cardiac muscle, owing to the excess of fat deposited about the heart and between the fibres. Recent investigations go to show that the influence of fatty infiltration and fatty degeneration, so-called, in producing cardiac symptoms have been greatly exaggerated. Pratt states that the size of the

Fig. 4.—Rupture of left ventricle during passage of stomach tube.

heart is proportionate to the size of the skeletal muscles, but the work of the heart is proportionate to the bulk of the body. In the corpulent, owing to the sedentary life and other causes, the muscles are usually small and flabby, although the bulk of the body is greatly increased. Hence there is a relative disproportion between the size of the heart and the work it has to perform—in other words, there is a condition of chronic overstrain. The hearts of the corpulent are therefore poorly adapted to withstand the additional increase of work attendant on over-exertion.

Gout is another disease in which the heart is usually small in proportion to the body weight, though I do not know of any observations on the capacity of gouty subjects to bear exertion.

Other conditions, wherein unwarranted exertion may be followed by the most serious consequences, are the myocardial changes following acute diseases. The toxæmia of the acute infective diseases—typhoid fever, diphtheria, rheumatism, pneumonia, influenza etc.—as is well known, produces serious damage to the myocardium, the effects of which are shown both in the parenchymatous and interstitial tissues. Of the parenchymatous changes, cloudy swelling, fatty and hyaline degeneration, vacuolation and segmentation of the cells, and fragmentation of their nuclei have been described. The interstitial changes include swelling of the intima of the vessels, hyaline degeneration of the media, and leucocytic infiltration of the adventitia. Numerous capillary hæmorrhages occur at times, and especially important is the formation of hyaline thrombi in the small vessels in some instances. Degenerative changes have also been described in the cardiac ganglia and vagus nerve. Curschmann believes that the overwhelmingly preponderating rôle in the morbid process is played by the interstitial changes, which account for most of the clinical manifestations.

The anatomical relations of certain diseases may account for variations in the gravity of the prognosis in cases exhibiting similar myocardial changes. Thus in pneumonia or in acute infections in persons suffering from emphysema or

other causes of obstruction to the pulmonary circulation, the resulting increase of work thereby thrown on the right side of the heart is a danger superadded to the myocardial degeneration. In diphtheria especially, in addition to the direct influence of the toxins on the muscle, there are reasons for believing that the cardiac ganglia and vagus nerve may also be involved, in a manner similar to that which occurs in peripheral neuritis. The cardiac manifestations of this disease would therefore be in part analogous to the paralysis occurring in the skeletal muscles from peripheral neuritis, with this important difference—that the latter may obtain physiological rest until the comparatively slow process of nerve and muscle regeneration takes place. There is little wonder, therefore, that the damaged heart, forced to bear the pressure of the circulation, sometimes fails in its task, even when this has been reduced to the fullest possible extent. Much less wonder is there that failure occurs in a case where over-exertion has been allowed. A consideration of these facts will explain the greater severity, more prolonged duration of symptoms, and the greater frequency of sudden death in the myocardial trouble following diphtheria.

In rheumatism, the frequent coincidence of endocarditis and pericarditis, with possibly valvular defects, are factors to be borne in mind in forming an estimate of the gravity of the myocardial changes. In addition to all these diseases, we must not lose sight of the effect of the toxins on the central nervous mechanism controlling the circulatory apparatus and blood pressure which, though ill understood, are no doubt of importance. In rheumatism the use of the salicylates also possesses elements of danger. The pain attending this disease to some extent is conservative in its action by enforcing bodily rest. The salicylates, themselves depressing to the cardio-vascular system, relieve the pain without preventing or ameliorating the myocardial mischief. This enables the patient to get about at an earlier period than would otherwise be possible, with consequent danger to the weakened heart before sufficient time has elapsed for myocardial repair.

In typhoid fever, acute cardiac manifestations may occur at the height of the fever or during convalescence. The former usually disappear with the decline of the fever, while the latter, according to Curschmann, may persist for two months or more. He adds, however, that "it is comforting to know that the condition is almost invariably attended by a favourable prognosis." He states, moreover, that he has never seen a case of myocarditis, with or without dilatation, following typhoid, though he notes the mention of isolated cases in the experience of other observers. My own limited experience does not accord with Curschmann's favourable view, as illustrated by the following two cases, in which death from cardiac failure occurred at a late period following the disease:

CASE IX.

In the first, a previously robust man of 23 years of age, a physician, had a rather severe attack of typhoid fever in January, 1901. He was confined to bed for two months, and apparently made a good recovery. Two months more were spent in recuperation, when he felt sufficiently well to resume practice. His practice was in the country, and the following autumn, seven months after getting out of bed, there was a gathering of the farmers of the neighbourhood at a barn-raising, which the doctor also attended. Not to be outdone in feats of strength by his rural friends, he undertook his share of the heavy lifting required on the occasion. He complained of no immediate ill effects, but the next morning, while sitting in his chair talking to his housekeeper, he suddenly died. No autopsy was made, but death was attributed to cardiac dilatation following on the severe exertion of the previous day.

CASE X.

For the notes of the following case I have to thank my colleague, Dr. J. T. Fotheringham, of Toronto. The patient was a physician and professor in the Ontario College of Pharmacy. He was 45 years of age, of excellent physique and athletic tendencies. He had never suffered from any serious illness until the midsummer of 1901, when he contracted a mild and irregular form of typhoid fever while at his summer home on the Muskoka Lakes. Although feeling very miserable he continued to go about for about a month, when he came to Toronto, and entered the General Hospital under Dr. Fotheringham's care. His temperature at this time did not exceed 101°. Defervescence soon occurred, and within two weeks he insisted on leaving the hospital, about the end of August. At the middle of September he undertook his academic duties, and continued them throughout the winter. Early the following summer he attended the military camp at Niagara in charge of the field hospital, and afterwards went to Muskoka for his holidays. Ever after his attack of typhoid he was subject to syncopal attacks, especially on suddenly arising from bed at night. He also found himself unable to undergo exertion, such as hurrying for a car. His pulse was slow and irregular. No valvular lesion was present. While at the college one Saturday during the Christmas holidays of 1902, after ascending a flight of stairs, he was suddenly seized with a

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.

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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.