CONSIDERATIONS AS TO THE ETIOLOGY AND SIGNIFICANCE OF DILATED HEART.

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The object of this paper is not so much to adduce fresh facts respecting the causation of dilatation of the heart as to review the more important factors concerned in its production, with especial reference to their relation to one another, to the essentially symptomatic nature of the condition, and to demonstrate that its significance in relation to prognosis and treatment depends at least as much upon its precise causation as upon its degree. Before considering cardiac dilatation in its ordinary pathological sense it is necessary to recall what modern observations have made clear as to dilatation of the heart under physiological conditions of the circulation. In the case of the healthy heart temporary dilatation may very readily be induced by increased work and within certain narrow limits a considerable increase in the strain which has to be met. By so doing, others, be a mechanical advantage in relation to the ventricular output. These limits are, however, very readily passed and when this is the case the dilatation is an evidence of fatigue—it means that the heart is becoming overstrained. As the intensity of the mechanical strain increases the muscular force of the heart will manifest itself early or late according to the severity of the strain which has to be met and the condition of the heart at the time. If the strain is well developed and in a vigorous condition it is capable of responding within considerable limits to the call for increased work, and conversely if the strain is from any cause enfeebled or out of condition a comparatively slight additional strain will induce dilatation.

It is broadly true of the heart as of the voluntary muscles that the liability to fatigue is dependent not only on the severity and duration of the strain alone, or on the condition of the muscle alone, but on the inter-action of the time being between the mechanical strain and the ability of the muscle to cope with the strain. In other words, in the causation of dilatation of the healthy heart which results from fatigue the two essential factors—factors of varying relative importance—are (1) the element of mechanical strain and (2) the condition of the myocardium.

To any who have not given consideration to the subject it may not be obvious how important a bearing the temporary dilatation of the healthy heart under the stress of increased work has upon pathological dilatation. It is, however, easy to trace a gradual transition from the fugitive temporary dilatation of the vigorous heart, the result of some violent exertion—e.g., running or bicycling—to acute dilatation which is unquestionably pathological. If the strain has not been excessive and the heart muscle is healthy the disturbance of equilibrium in the heart, it may be almost immediately, is temporary, whereas if the strain has been excessive the fatigue is more lasting and it remains dilated for hours or days or weeks, as the case may be. Again, if the heart muscle is out of condition, whether from lack of training, as a result of disease, or from some other debilitative condition, a comparatively slight increase of work will cause a disturbance of equilibrium—the heart is sooner fatigued than it should be and the liability to definite pathological dilatation is great. In the former case it is only the excessive work that is mainly responsible for the overstrain of the heart; in the latter it is the inability of the heart muscle to withstand the increased strain that mainly determines it. It is, however, when the two factors are combined that the abnormal condition of the circulation would be the temporary dilatation of fatigue passes straightway into definite and more or less lasting pathological dilatation. The importance of both factors, which we may for brevity term the mechanical and the myocardial, is most forcibly brought out in those cases which are, as is well known, mainly a matter of the heart.

By judicious exercise the heart is gradually strengthened so that the force of its contraction is capable of effectively counter-balancing the abnormal increase of strain, and which is thrown upon it during exertion, and if the increased work is sufficiently severe and long-sustained some degree of physiological hypertrophy of the heart ensues. In passing it must, however, be remarked, as emphasised by Roy, that this physiological hypertrophy is not at all in the same physiological as that of the muscles of the athlete or as the "blacksmith's arm," it is more liable to undergo retrogressive metamorphosis than are unhypertrophied muscles.

But training, whilst it pays such attention to this gradual hardening and strengthening of the heart, and the favours it should come into play. My object in referring to these matters respecting the behaviour of the heart under physiological conditions, or under conditions which deviate comparatively little from physiological limits, is to illustrate the essential importance of my first point, that the ability of the heart to withstand an increased strain is not only a matter of extra work—or what amounts to very much the same thing, the liability of the heart to suffer from the effects of increased strain—is dependent upon the inter-relation that exists at the time being between the element of mechanical strain acting upon the heart and the condition of the myocardium which has in large measure to bear the strain, and that whilst on the one hand excessive mechanical strain without any important variation in the myocardial factor, and on the other primary myocardial weakness without any increase of work has the physiological mean, may, acting alone, determine a disturbance of equilibrium or definite cardiac breakdown, it is when both factors are combined that the heart is especially liable to pass from a condition of temporary heart fatigue to one of definite pathological dilatation.

From which there follows as a necessary corollary the conclusion that, given a disturbance of one factor, a very slight modification of the other may suffice to turn the scale against the heart, a conclusion which underlies the fact familiar to clinicians that when the heart and circulation are in a condition of unstable equilibrium marked variations in the size and behaviour of the heart are very readily induced by comparatively trivial causes.

Reverting to the clinical etiology of pathologically dilated heart we find that when we go behind the numerous clinical antecedents of the condition the same principle still holds good; the two essential factors in its causation are: (1) increased work; and (2) myocardial weakness. These two essential factors may be operative alone or combined; it is, however, clear that it is the rule, if indeed it is not invariable, for the element of myocardial weakness to play a part sooner or later even in cases in which disturbance of the usual pathological conditions of the circulation was the primary cause of the heart lesion.

INCREASED MECHANICAL STRAIN AS A FACTOR IN DILATATION.

It is evident that increased intra-ventricular pressure must throw more work upon a hollow muscular organ like the heart, and the three most important proximate causes of intra-ventricular pressure so far as the left side of the heart is concerned are: (1) hypertension; (2) hypertrophy; (3) abnormal high blood-pressure in the systemic circuit, and certain valvular lesions, obstructive or regurgitant, but notably aortic lesions; and for the right side of the heart sustained high blood-pressure in the pulmonic circuit, the lesion being usually one of secondary pulmonary hypertension associated with compensatory dilatation of the right ventricle. That such affections as these are potential causes of dilatation is an elementary fact in cardiac pathology, but we know that in fairly small doses both abnormally increased right and left is usually viewed as associated with coronary occlusion. That such affections as these are potential causes of dilatation is an elementary fact in cardiac pathology, but we know that in fairly small doses both abnormally increased right and left

1 A paper read before the Harveian Society of London on May 18th, 1899.
2 Contributions to the Physiology and Pathology of the Mammalian Heart, Philosophical Transactions of the Royal Society, 1892.
3 A paper read before the Harveian Society of London, 1892.
4 It is well known that the heart is especially liable to pass from a condition of temporary fatigue to one of definite pathological dilatation.


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but this, except in the case of aortic regurgitation, is relatively unimportant. If, however, the strain to which the heart is subjected should be excessive or the heart muscle be unable to respond to the physiological stimulus of increased work it is primary dilatation—dilatation in the definite pathological sense—whi-ch ensues. Now it is when there is a definite tendency to dilatation that other physical factors connected with intra-ventricular pressure come into play and materially aggravate the effects of the primary dilatation. The heart muscle has to contend. I refer to two, the importance of which was so ably demonstrated by the late Professor Roy in conjunction with Professor Adami, though hardly done justice to by clinicians. 1. That the vents are not normally equal to that the aortic pressure is a great contributor to this, and that some residual blood remains behind, its amount being readily affected in either direction by variations in the aortic pressure. A moderate increase in the amount of residual blood does not cause an increased strain on the myocardium. It is true, but it is otherwise when the heart is already overworked; then the amount tends to increase and per se adds to the difficulties of the heart. Now this point is surely one of much practical importance; it means that a relatively slight increase of aortic pressure, especially so if accompanied by a very slight degree of pathological dilatation of the heart, and confirms what clinical experience teaches—that the part played by separate etiological factors must be carefully sifted out and their influence as far as possible be eliminated before we attempt to establish the exact condition of the myocardium. 2. The second fact that is very formidable. In this connexion the recent observations of my colleague, Dr. Lees, respecting dilatation of the left ventricle in acute specific diseases with or without actual myocarditis, fatty and other forms of degeneration, fibrosis, stenocardial myocardial weakness, the dilated heart of severe anemia, of nervous or general debility, and that associated with toxic causes. Clinically careful examination and inquiry usually enable us to decide as to the cases which belong to the category of primary myocardial weakness, for they are generally characterised by an absence of systemic fever and by a relative importance of dilatation in the causes of heart failure. This consideration, taken in conjunction with what has been said respecting the relation of increase in the residual blood, leads to the conclusion that a dilated ventricle is per se unfavourably placed for mechanical reasons. In every connexion dealing with the element of mechanical strain in the causation of dilatation and in gauging at the bedside its importance in any given case we have to do with other factors besides the primary increase of intra-ventricular pressure to consider. As an instance of how that sometimes the dilatation is connected with the element of mechanical strain, the rise of systemic blood-pressure to the condition of the heart the relationship between the general blood-pressure in the systemic circuit and the local blood-pressure in the coronary circulation must be taken into account.

MYOCARDIAL WEAKNESS AS A FACTOR IN DILATATION.

Inasmuch as in every case of dilatation of sufficient degree there is some measure of myocardial weakness which is the direct consequence of the dilatation it is necessary to remark that we are here dealing with the myocardial weakness as the primary and predominant factor in the actual causation of the dilatation. The examples of acute specific diseases with or without actual myocarditis, fatty and other forms of degeneration, fibrosis, stenocardial myocardial weakness, the dilated heart of severe anemia, of nervous or general debility, and that associated with toxic causes. Clinically careful examination and inquiry usually enable us to decide as to the cases which belong to the category of primary myocardial weakness, for they are generally characterised by an absence of systemic fever and by a relative importance of dilatation in the causes of heart failure. This consideration, taken in conjunction with what has been said respecting the relation of increase in the residual blood, leads to the conclusion that a dilated ventricle is per se unfavourably placed for mechanical reasons. In every connexion dealing with the element of mechanical strain in the causation of dilatation and in gauging at the bedside its importance in any given case we have to do with other factors besides the primary increase of intra-ventricular pressure to consider. As an instance of how that sometimes the dilatation is connected with the element of mechanical strain, the rise of systemic blood-pressure to the condition of the heart the relationship between the general blood-pressure in the systemic circuit and the local blood-pressure in the coronary circulation must be taken into account.

Before passing on to the consideration of the myocardial factor in the causation of cardiac dilatation it may be well to refer to the fact that even in cases where the element of dilatation has been most pronounced the condition of the myocardium has very much to do with later developments. Thus the dilated heart of chronic high arterial tension is partly due to the rise of intra-ventricular pressure and partly to changes in the myocardium, degenerative in nature and in degree of severity. It is evident that the latter connexion the precise condition of the coronary circulation is of much importance—an importance which appears to be considerably underestimated to judge by current observations. It is well known that one of the important conditions influencing the vigour of the heart beat is the blood-flow in the coronary arteries; a rise of blood-pressure at the root of the aorta with a corresponding improvement in the coronary circulation is important. Conversely a fall of blood-pressure in the aorta leading to a lessened blood-flow in the coronary arteries causes diminution in the force of the heart beat. So important is this relation of the coronary to the general circulation that a general rise of blood-pressure in the systemic circuit may by increased vigour of the heart more than counterbalance the effects of the increased strain on the heart. It is not unusual for us to observe at the bedside that in certain pathological states—notably chronic Bright's disease—the condition is comparatively satisfactory while the general blood-pressure remains high, whereas coincidently with the fall of blood-pressure below a certain limit the general condition is very bad and the heart is commonly explained as being due to the heart muscle giving out in consequence of the prolonged strain to which it has been subjected, and no doubt this is true; but it appears to me extremely probable that once this diminution of general blood-pressure does supervene from commencing heart failure the progress of the latter may be greatly accelerated by a consequent fall of blood-pressure in the coronary arteries leading to imperfect nutrition of the heart muscle. Then, again, it is not probable that the marked liability to sudden heart failure in cases of advanced aortic regurgitation is in part due to the abnormally low blood-pressure in the aorta and consequent interference with the coronary circulation. Other instances might be given, but it is enough to illustrate the point that in considering the relation of variations in the general blood-pressure to the condition of the heart the relationship between the general blood-pressure in the systemic circuit and the local blood-pressure in the coronary area must be taken into account.

4 Transactions of the Royal Medical and Chirurgical Society vol. lxxxi.
accompanied by very little variation in the size of the heart, whereas a heart with senile myocardial weakness may remain much enlarged for years with but very little in the way of severe symptoms. So important is this lack of correspondence between the amount of actual enlargement of the heart and the degree of muscular feebleness or degeneration that clinical observation is in the majority of cases insufficient to balance the degree of dilatation as upon its cause and the amount of contractile vigour possessed by the heart muscle as evidenced by the pulse, the characters of the cardiac impulse, and of the heart sounds.

In alcoholic indiscretion is especially important when the heart is a direct depressant to the cardiac muscle. It causes weaken- enough dose induces dilatation, which may be accompanied by relative incompetence of the auriculo-ventricular valves. If we recall in this connexion that given slight dilatation any degree of superadded myocardial weakness will seriously handicap the heart we have no difficulty in understanding the pathological conditions of the hearts seen in hospital-outpatient practice. Alcohol, given in sufficient quantity under experimental conditions, acts as a direct depressant to the cardiac muscle. It causes weaken- ing of the vascular contractions, and if given in large enough dose induces dilatation, which may be accomplished by relative incompetence of the auriculo-ventricular valves. With ordinary drinkers there are other factors besides the direct toxic action of the alcohol on the heart muscle; the large amount of fluids ingested is in itself sufficient to increase the pressure in the great vessels, and the cardiac output is intensified by the high arterial tension so often present in alcoholics. From extended and critical observation it appears to me that it is difficult to over-estimate the importance of alcoholic excess in causing or precipitating cardiac lesions. The question of whether in the majority of cases the disease is due to the alcohol itself, or to other toxic agents, such as nicotine and possibly other constituents of tea. The abuse of tobacco, as is well known, causes various functional disturbances of the respiratory tract, such as asthma, bronchitis, tachycardia, irregularity, and the latter may seem to react unfavourably on the mechanics of the circulation; but what is of most importance in connexion with dilated heart is the direct toxic action of nicotine on the heart muscle similar to that produced by alcohol, if present in sufficient amount. I do not think that there is much clinical evidence that tobacco is a relatively important factor by itself in causing dilatation, but I am con- vinced from clinical observation that when the heart is already overtaxed by increased mechanical strain, and when this is the case an amount of alcohol which under normal cir- culatory conditions would not injure the heart is sufficient to cause irreparable damage. There are many instances of this in everyday practice, such as chronic high tension and valvular lesions affecting the left side of the heart and emphysema affecting the right ventricle, in all of which failure of compensation is very liable to be prematurely induced by indiscipline in this respect.

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of heart failure. But what concerns us from the point of view of prognosis and treatment is the special significance in an extremely delicate case of a degree of dilatation that is not only a virtual or a statistical demonstration of the cardiac weakness that is present. This we can best ascertain by attention to the following points.

1. The degree of dilatation. — Now, as Boy and Adrain pointed out, dilatation in the heart is a special type of failure of equivalent of fatigue, so that the measure of the dilatation is a reliable indication of the degree of fatigue. From this the deduction is drawn that in "a diseased heart the degree of dilatation is in inverse ratio to its power of doing its work."

2. The condition of the myocardium with which the dilatation is associated. — It is well to remember that a man with fatty degeneration or syphilitic fibrosis or gumma of the heart walls, or with disease of the coronary arteries, may be on the brink of a catastrophe although the heart is but little dilated, whilst another with much smaller dilatation, or without any pathological condition, may have years of life and usefulness before him.

3. The condition of the heart in relation to the general circulation is affected. — Apart from physical signs we may take note of other factors and the next one to be considered is:

4. The reaction of the heart to treatment. — A broad view on lines such as these is essential if we are to avoid the extremes of making too much or too little of any given cardiac dilatation, and perhaps at the present time the former is as important as the latter.

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ANEURYSM OF THE SUBCLAVIAN ARTERY; LIGATION OF THE FIRST PORTION OF THE SUBCLAVIAN, FOLLOWED IN THIRTY-EIGHT DAYS BY REMOVAL OF THE ANEURYSM.

By Herbert W. Allingham, F.R.C.S. Eng., Surgeon to the Household of H.R.H. The Prince of Wales; Assistant Surgeon and Lecturer on Operative Surgery at St. George's Hospital.

A seaman invalided from the Royal Navy, aged 25 years, was admitted into St. George's Hospital on Feb. 20th, 1899, and gave the following history. Three years previously he had noticed a swelling above the right clavicle which increased gradually in size. This increase had been especially marked during the 12 months previously, and had been accompanied at any time by swelling below the tumour. The patient's general health had always been good and syphilis was denied. On examination of the tumour it was evident, but otherwise there was no symptom except the pulse. Some slight tenderness on pressure over the tumour was still present, but it was decided to ligature the subclavian artery and to await some change in its size which might be expected during its spontaneous regression.

On March 1st a V-shaped incision was made on the right supra-clavicular region, filling the interval between the sterno-mastoid and trapezius muscles, and in size of about that of a Tangerine orange. There was a characteristic systolic bruit with delay and diminution of the right radial pulse. Some slight tenderness on pressure over the tumour was evident, but otherwise there was no symptom except the pain passing down the inner side of the arm to the elbow. A consultation with my colleagues was held and it was decided to ligature the subclavian artery on the proximal side of the tumour. This was done on March 1st through a V-shaped incision, one arm of the V being down the anterior border of the sterno-mastoid and the other arm along the clavicle. The flap of skin was turned up. The sterno-mastoid muscle was divided and the first part of the subclavian artery was exposed and a kangaroo tendon passed round the vessel immediately external to the origin of the vertebral artery and just to the inner border of the scalenus anticus. The recovery from this operation was complete and it was thought that there was no indication of secondary haemorrhage. No pulsation was felt in the tumour or in the brachial or radial arteries. On the 4th some slight beating was detected in the aneurysm, which continued being felt day by day. The sac meanwhile was apparently smaller and harder. On the 17th, as the pulsation was still decided, the patient was again anaesthetised and a further consultation was held. It was found that the tumour although pulsating did not expand with the beat of the heart and it was therefore considered advisable to further watch its progress before attempting more radical measures. On the 26th it was noted that the aneurysm was increasing in an upward direction and that there was more pulsation which was both visible and palpable. On April 8th, my colleagues, Mr. Holmes, Mr. Dent, and Mr. Turner, to whom I am indebted for their valuable advice, being present, and assisted by my colleague Mr. Jaffrey and Mr. Collard, the difficult and severe expedient of removal was resorted to five and a half weeks after the preliminary ligation. Here it may be remarked that the hardening and, as one may so put it, the better definition of the tumour brought about by a previous ligation of the subclavian rendered the complete removal easier and more expeditious than if a soft, thin-walled sac had had to be dealt with, in which case the amount of blood lost would have been less excessive owing to the small calibre of the channels to become active that fear of gangrene would arise. The operation occupied two hours. The after-history was unimportant, healing of the large wound taking place after some slight local skin sloughing. A section of the aneurysm shows that its cavity is largely occupied by firm clot, the blood passing into it from the subclavian, which was partly patent, and out posteriorly by way of what appeared to be the profunda cervicalis. In shape it resembled a pear with the stalk external and the base upwards. The axillary artery, as far as it was exposed in the wound, was found to be obliterated. A careful examination of the arm was made on May 5th, and it was found that pulsation was present in the brachial artery, and was followed by decidedly good results, limited though these were to a hardening of the aneurysm and diminution in its pulsation. It is interesting, also, that the procedure was followed by decidedly good results, limited though these were to a hardening of the aneurysm and diminution in its pulsation. It is interesting, also, that the procedure was not followed by secondary haemorrhage as is supposed to follow on ligation of the first part of the subclavian artery. As there was no prospect that the pulsation would cease altogether, the difficult and severe expedient of removal was resorted to five and a half weeks after the preliminary operation. Here it may be remarked that the hardening and, if one may so put it, the better definition of the tumour brought about by a previous ligation of the subclavian rendered the complete removal easier and more expedi- tion. However, it is not always necessary to follow the sac the tumour would cease altogether, the difficult and severe expedient of removal was resorted to five and a half weeks after the preliminary operation. Here it may be remarked that the hardening and, if one may so put it, the better definition of the tumour brought about by a previous ligation of the subclavian rendered the complete removal easier and more expeditious than if a soft, thin-walled sac had had to be dealt with, in addition to the probability that if rupture occurred during manipulation loss of blood would have been less excessive owing to the small calibre of the channel through the aneurysm. Second, the preliminary ligation had caused the collateral anastomosing channels to become active that fear of gangrene was at no time entertained. Thirdly, removal of the greater part of the clavicle was necessary in this case, access to the sac and to the vessel below being impossible without this operation. Even now, so soon after the operation, the patient can perform all of the usual movements without difficulty, although it can hardly be expected that he will retain full...