An Address

ON

CARDIAC PROBLEMS IN ADOLESCENT LIFE.

DELIVERED TO THE BUCKINGHAMSHIRE DIVISION OF THE
BRITISH MEDICAL ASSOCIATION

BY

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THE problems in connexion with disease and disturbance of the heart vary to some extent with the period of life. During childhood and youth we find the foundation laid of organic heart disease, which in many cases causes no inconvenience to the patient until later on in life. On the other hand, the disease may have been so severe in childhood, especially in cases untreated, that the whole adolescent life is seriously crippled. Advancing years bring their degenerative changes, cardiac and vascular, of which not a trace is present in early life. Acute illnesses in the young are not accompanied by those serious cardiac complications which so often lead to cardiac failure in later life. The problems which I bring before you to-day are largely the ones which have confronted me in my ordinary work and which I had to try first to understand and then to solve. You will find that the ordinary textbooks rather leave you in the air as regards these problems because it is not their function to deal with the actual experiences of medical life and work. I propose, then, to give some hints as to how to approach these problems of cardiac disturbance and cardiac disease, so as to enable you to deal with the prognosis and treatment of the individual cases you are called on to deal with in practice.

1. CONGENITAL HEART DISEASE.

I do not refer here to cases which have been recognized early and watched through infancy and childhood, but to the unexpected discovery of a cardiac lesion, usually through the presence of a cardiac murmur. The boy or girl may be quite well and happy, the parents may have had no cause to suspect hidden trouble, but the doctor is often pained and puzzled and anxious as to what he ought to do. The murmur is often loud—in fact some of the loudest murmurs are found in connexion with congenital lesions. It is usually systolic in time, loudest at the base of the heart or at the left side of the sternum, and often quite audible in the back. On a systolic murmur alone it is not wise to make a diagnosis of the exact lesion—nor is it necessary. If a double murmur is heard at the base, running through systole and diastole—the so-called mill-wheel murmur—one can with confidence diagnose a patent ductus arteriosus. This is sometimes taken to be the double murmur of aortic disease—obstruction and regurgitation—but to anyone who is familiar with the characteristic sound of an aortic regurgitant murmur the difference ought to be manifest. The changes in the heart and in the circulation which follow from a double aortic lesion are quite different from those in a patent ductus arteriosus case.

We return to our patient with the evidence of a congenital heart lesion and his puzzled doctor. The first comforting fact is that if the boy has reached the age of 12 or 15 years, the congenital lesion cannot have been very severe, for if it had the boy would have died in early life. The mortality associated with congenital heart disease is very high during the first five years of life, either from cardiac failure or defective circulation. So that the fact that the patient has passed through childhood successfully is a hopeful sign.

The doctor will next make a general survey of his patient from the point of view of his physique and development. If he finds that the boy has grown normally and has the development suitable to his years, he may also be reassured as to the degree of congenital defect. It may be laid down as a general law that if any vital organ has had its functional capacity impaired from the time of birth there will follow a corresponding defect in the normal general development of the body. This condition, known as infantilism or dwarfism, is seen as the result of congenital disease (with impaired function) of the liver, the intestines, the pancreas, the kidneys, the lungs, and the heart. Hence if we find our young patient normally developed we have an indication not to form a gloomy prognosis.

Another question to be settled is, What sort of life has the patient been able to lead? Has he led the ordinary life of childhood, or has he been unable to engage in the games and exercises of his companions? If the parents are unaware of any disability, or if the boy has been leading an ordinary life, then we have most important evidence as to the small significance of the congenital lesion. You may count as of no importance the presence of a murmur, however loud it may be, if the boy holds his own at cricket and football. If there has been no disability of the heart from the functional point of view, you will probably find no evidence of dilatation or hypertrophy in the ventricles, or any physical change in the heart other than the murmur.

On the other hand, there may have been noticed a certain limitation in the patient's ability to undergo any active or prolonged exertion within the normal powers of his years. In such a case you will probably find some change in the size of the heart, usually in the shape of hypertrophy of the right ventricle, sometimes of both ventricles. When there is a difficulty in supplying oxygen in sufficient amount to the tissues as the result of a congenital lesion there will be some compensatory change in the heart, manifested as hypertrophy, or in the blood, manifested as polycythaemia. When the compensation is complete, as sometimes happens, there will probably be no symptoms of functional disability. When the compensation is not quite complete, as more commonly happens, there will be a corresponding limitation of the patient's powers owing to functional inefficiency of the heart.

If these views be accepted the indications as to the lines of treatment will be clear. The boy who has no symptoms and who has been leading an ordinary life will be allowed and encouraged to continue doing so. The boy who has found certain limitations to the amount of exercise he can take and strain he can undergo will be advised to keep well within those limits. More especially are competitive struggles with healthy boys to be discouraged. His life race must be run at a slower pace and with a certain time allowance. Record breaking is not for him. If we are guided by the symptomatology in these cases our treatment will probably be much more beneficial to the patient than if we concentrate on the problems presented by the cardiac murmurs.

2. THE ASTHENIC HEART.

I have applied the term "asthenic heart" to a very common condition in adolescence. It is not a disease, but a developmental form of general asthenia which may manifest itself by symptoms ascribed to a weak heart.

My typical example of this condition is that of a boy or girl for whom medical advice is sought because of fainting, or palpitation, or temporary loss of consciousness, or sudden changes in colour—turning white or flushing suddenly—or pain about the precordial region. Alarm has been caused by the presence of one or more of these symptoms, and the home diagnosis has been some form of heart trouble.

Now we have here a serious problem—to say whether a heart is sound or not. All of us can detect well marked disease of the heart with great ease, but when it comes to the decision as to the absence of any disease the problem is much more difficult. One fact may be kept in mind here—that while in advanced life we often find symptoms of organic disease without marked physical changes in the heart, during adolescence we are not likely to meet with serious heart disease which is not evidenced by clearly defined physical changes in the heart. Before dealing with the examination of our patient's heart let us consider the symptoms which have excited alarm.

In adolescent life organic heart disease may or may not be accompanied by symptoms. The symptoms when present are usually definite and traceable to the heart. In patients who are going about they are—shortness of breath on exertion, a disinclination to make the exertions which are a pleasure to healthy companions of their own age, and a feeling of tiredness after moderate exertion. These symptoms are quite compatible with heart disease, and to the medical mind they suggest it. You will note, however, that our type patient here has not been brought because of these symptoms. Faintness and fainting, loss of consciousness, sudden changes of colour, and chest pains—these are not suggestive of heart disease in young people who have been leading an ordinary life. I go farther and say that so far as my own experience goes a history of one or more of these symptoms is *prima facie* evidence that the boy has not heart disease. You are all familiar with the type of boy or girl who faints at school when standing up. The vasomotor tone is low and the cardiac

tonus is not fully developed. This is the type of case we are dealing with, and the other symptoms are to be similarly explained. So that in dealing with symptoms which have caused alarm in the family circle and led to a diagnosis of heart disease do not be misled, but consider carefully whether the symptoms are really cardiac and whether they cannot be explained as having another origin.

The patient's heart must be examined, and here it must be admitted difficulties often arise and differences of opinion appear. There are certain changes in the heart which some will pronounce to be pathological and which others—including myself—will declare to be within physiological limits. The common changes noted are: (1) an increase of the heart rate; (2) an irregular action of the heart; (3) the presence of dilatation, usually of the left ventricle; (4) the presence of a systolic murmur at the apex.

(1) *Increased Frequency of the Heart Rate.*—While no one will be alarmed by an increase of rate up to 100 beats a minute, when one finds a rate of 120, or 140, or even 160, as I have found it, there is apt to be suspicion as to serious disease. The rapid heart in those cases is, however, the result of nervous disturbance and instability. The owner of the heart which runs off at the rate of 140 will show other evidences of nervous instability, more especially on the sympathetic and emotional side. Endocrinologists would no doubt supply information as to the exact hormone which is wanting or is in excess. You will find that the heart rate varies much from time to time, but when the patient is asleep it is always normal. This does not happen in hearts which are toxic or suffering from active organic disease.

(2) *Irregular Action of the Heart.*—A sinus irregularity of the heart, if the action is slow, is very common and is dependent on respiration. Extra-systoles may occur at times. When there is a combination of these two irregularities—a compound irregularity, as I have called it—the result is rather puzzling to one unaccustomed to the irregularity. It should always be kept in mind that the serious irregularities of the heart beat, such as those associated with auricular fibrillation, auricular flutter, heart-block, etc., are extremely rare during adolescence. On the other hand, the above forms are quite common and can usually be recognized by making the patient hold his breath. During the period of breath-holding the sinus irregularity will disappear entirely, and the extra-systoles will present their characteristic features, which are easily recognized. It may be stated definitely that neither of these irregularities is of any significance or importance, as they occur quite commonly in the absence of any organic disease of the heart.

(3) *Dilatation.*—If a medical man believes that the youthful heart must lie in a certain position and within the prescribed geographical limitations of the textbooks, he will find pathological dilatation common in these patients. Having found it on one occasion he will probably order some months' rest in bed—irrespective of the fact that a little exercise would probably have brought it within his normal standards. If, on the other hand, he recognizes—as is the case—that the youthful heart lies in many different positions, and that the apex is a very variable point, he will not be much concerned at finding the apex beat in the fourth intercostal space and half an inch outside the nipple line. He will examine the patient standing up and lying down; after rest and after exercise; and he will note many changes in the shape of the heart. In the type of case we are dealing with there is no doubt that these physiological variations are strongly marked. The displacement of the apex outwards and to the left is often very striking. Equally striking is the way it may come in again as the result of any strain thrown on the heart, such as a smart run or drawing deep breaths. I cannot help thinking that in such patients full inflation of the lungs has a striking effect in bringing the heart back to "normal" limits. If, on the other hand, one is dealing with dilatation due to organic heart disease, it will be found that the position of the apex does not change in the same way as the result of exercise or full respirations.

(4) *The Presence of a Systolic Murmur.*—A soft systolic murmur is often audible at the apex and may give rise to unwarranted suspicions of heart disease. In this connexion it is well to bear in mind that a slight degree of mitral regurgitation is never the cause of cardiac symptoms in a young person. The murmur, like the dilatation, is subject to great variation from time to time, and under changing conditions of rest and exercise. Both are probably dependent on the atonic condition of the heart. The ventricles are not contracting and the valves are not closing with their usual

smartness and efficiency. Hence at times a slight reflux takes place through the mitral valve, and a systolic murmur becomes audible. The important point is that we have no clear evidence here of valvular disease, and systolic murmurs at the apex, apart from definite valvular disease, may be safely ignored in the case of young people.

We have thus failed to find any convincing evidence of the presence of serious heart lesion. At most we have made out signs of cardiac flabbiness and instability—a very different condition from organic disease. If we extend our examination to the rest of the circulatory system we shall probably discover evidences of a lowered vasomotor tone. The extremities are often cold and dusky, and chilblains are commonly present in the winter months. The pulse is soft and weak and of low tension. If we feel it at the wrist with the arm pendent it may appear to be fairly good, but if the hand be raised above the head the pulse falls away or disappears entirely. This is in marked contrast to the pulse in a healthy youth, which maintains its good tone in any position and irrespective of gravity. Functional or postural albuminuria is often present as the result of vasomotor instability.

If we extend our examination of these patients still farther we shall find that it is not only the heart and blood vessels which are asthenic, but that other muscles in the body are lacking in tone. The stomach and intestines are atonic. Splashing is often elicited in the stomach many hours after a meal, due to atonic dilatation. The muscles of the abdominal wall are flabby and atonic. So general are these evidences of muscular weakness that we are led to assume a nervous cause rather than a purely muscular condition. There is as the basis of the varied symptomatology an underlying state of nervous instability and immaturity. Whether time will show that this is also traceable to some endocrine defect or not I cannot say definitely—it is quite possible. But there is no doubt, from the period of life at which the symptoms show themselves, that the condition is largely a developmental one, tending to come on at the age of puberty and to pass off when the patient has reached adult development. In some cases it does not pass off, and if the subject has joined up and been accepted for the army, after a period of strain he will be discharged as suffering from "soldier's heart."

As regards treatment, if the symptoms complained of are not cardiac in origin, and if signs of organic disease of the heart are absent, do not acquire the reputation of being a careful doctor at the expense of the patient's future—by treating him as suffering from a weak heart. If you accept the views given above you will agree that the very worst policy to pursue is to make invalids of these subjects by guarding and treating their hearts. Such a policy has in the past cut short many a promising career. Patients of this type are to be encouraged to lead an active open-air life, and their ordinary routine is only to be regulated so far as to protect the undeveloped powers from undue stress and strain.

3. THE ATHLETIC HEART.

A somewhat allied condition is described and known as an "athletic heart." After some more than usually severe game or other exertion a boy has fainted or complained of severe precordial pain. He is said to be suffering from a strained heart. Now I do not wish to deny the possibility of the heart of a healthy boy being strained as the result of exercise, but I do say that I have never met with a case. I have met with many cases in which the heart was said to have been strained, but in all cases some other explanation presented itself. In some cases a damaged heart was present, the result of organic disease, which had been latent as regards symptoms until a breakdown occurred. I recall the case of a young miner who was said to have injured his heart as the result of lifting a heavy piece of coal. He became breathless and suffered from palpitation, and the doctor who examined him found that he had strained his heart. He drew full compensation for this for twelve months, at the end of which time I saw him. There was well marked mitral stenosis with hypertrophy of the right ventricle. By no stretch of imagination can mitral stenosis be induced by physical strain. This young fellow had certainly had rheumatic valvulitis some ten years previously, with the gradual development of stenosis in the mitral valve, but with compensation fully maintained until a severe muscular effort broke it down. This is one type of the so-called strained heart to be met with. In other cases so described I have failed to find any evidence of injury or strain. The diagnosis has been made either on the symptoms described in the last section—

palpitation, faintness, etc.—or in some slight deviation from normal on examination of the heart, such as dilatation, rapid action, etc. Perhaps it is dilatation of the heart that crops up most frequently as the result of this so-called heart strain. Now dilatation of the heart is an evidence not of strength but of weakness. It is never found to occur without some definite cause, such as organic heart disease or severe general illness. But heart strain is said to occur when neither of these conditions is present—in a presumably healthy lad undergoing the ordinary exercises suitable to his age. If you can imagine the Oxford and Cambridge boat crews being set to compete against each other twice a day for weeks on end, you might find that cases of dilatation from cardiac debility appeared. But no one would think of suggesting such a strain—one race a year suffices. In the case of schoolboys, a competition may lead to one entrant falling out exhausted before the race is finished, but he does not strain his heart. Why? Because the heart is very well able to protect itself, makes its own adjustments under conditions of physical stress, and when it has had enough produces in its owner sensations of breathlessness, precordial pain, or exhaustion, which make him pull up. I do not deny the possibility of a healthy youth producing a condition of injurious strain in his own heart, but I think all the chances are very much against it, and I have not so far met with a case. Consequently I do not find that the problems of a strained heart in youth are very difficult of solution.

4. ORGANIC HEART DISEASE.

It was suggested to me that I might give you an address to-day on cardiac murmurs. Heaven forbid! I hope that none of you has to mourn over years misspent in the unprofitable study of cardiac murmurs, as I have. Who was it who gave the death blow to this futile pursuit? It was James Mackenzie, a general practitioner in Burnley, who evolved the real methods of cardiac examination, discovered many hidden secrets of the cardiac mechanism, and pointed out the lines of further and progressive investigation. Mackenzie has always held that murmurs have to be considered in any examination of the heart, but he has also shown that they do not hold that position of importance which had been accorded them.

For practical purposes we may divide murmurs into two classes: first, those that count, and secondly, those that do not count. This applies very well to the adolescent period of life.

By the term "murmurs which count" I mean those that are indicative of serious valvular disease, and most commonly of lesions which lead to progressive cardiac change and cardiac disability. The murmur in itself is nothing. The questions it raises—Has the underlying lesion produced any changes in the heart? and Are those changes serious and likely to be progressive?—are everything. A murmur is a clue which has to be followed up, if it bears evidence on the face of it that there is something behind. The two chief murmurs falling into this category are the presystolic murmur of mitral stenosis and the diastolic murmur of aortic regurgitation. They claim notice because of their frequency and their importance. During adolescence a presystolic murmur tends to alter and become louder because the initial lesion of childhood, mitral valvulitis, has induced a progressive narrowing of the mitral valve. As the youth grows, the heart should also increase in size, and the mitral valve should become larger. But the process of stenosis which has begun prevents this enlargement of the mitral opening, and thus the work of the right heart is increased. The diastolic murmur of aortic regurgitation is quite common, much commoner than is usually stated and believed. This is because it is easily overlooked unless one is always on the watch for it. Either of these murmurs may be present at this age without any symptoms—that is to say, the patient may be leading an ordinary life and unconscious of any cardiac disability. This is all to the good, but a careful examination of the heart for secondary changes is none the less necessary. It is on the symptoms present and on the secondary changes in the heart that our immediate prognosis and treatment are based, and not on the mere presence of the murmur. These, then, are the two chief murmurs which count. Expressed in other words, it may be said that it is the murmurs which are diastolic in time that count. Compared with these two in frequency, the other murmurs which are of importance, such as an aortic systolic murmur, are so rare as not to call for consideration here.

By the term "murmurs that do not count" I mean the systolic murmurs common at the apex and the base, often

suggestive of mitral incompetence or of a lesion of the pulmonary valve; often soft and indeterminate as to whether they are produced inside or outside the heart; and often puzzling as to whether they signify anything or nothing. That is why I call them murmurs that do not count. They are not worth worrying about. However exact the diagnosis, one learns nothing from the murmur as to prognosis, which will be decided by the presence or absence of other changes about the heart, indicative of a valvular lesion.

In the solution of our cardiac problems I think one simple method of examination is too much neglected—namely, palpation. At the age we are considering it is much more applicable than in later years when fat breasts and thick chests make precordial palpation of little use. In adolescents we can make out the cardiac boundaries by palpation and percussion with great accuracy and we can determine definitely the presence of two most important conditions—namely, dilatation and hypertrophy. Personally I learn much more about the condition of the heart from palpation than I do from auscultation. By auscultation one learns what noise a valvular lesion is producing; by palpation one learns what effect the lesion is producing on the heart. Some doctors, beginning with auscultation, receive such a shock on hearing a murmur that they make no further examination. Let me illustrate my point.

A young patient was found to have a heaving apex beat, outside the nipple line, and indicative of hypertrophied action. There was no murmur audible and no evidence of heart disease apart from the hypertrophy of the left ventricle. The radial artery was hard and thickened, and the blood pressure was 160 mm. The urine was of low specific gravity and polyuria was present. Further examination showed the presence of bilateral cystic kidneys, which explained the vascular changes, and the ventricular hypertrophy noted on palpation.

A child suffered from a succession of attacks of precordial pain, cyanosis, rapid heart action, and collapse. These attacks were looked on as cardiac in origin, but on routine examination nothing abnormal was detected. There was a soft systolic murmur audible at the apex and the base but no evidence of valvular disease. Careful palpation showed an area of pulsation at the base of the heart, strong and suggestive of aneurysm, and on percussion the basal dullness was found to extend up to the clavicle. A diagnosis of aneurysm was made—there seemed no other possible diagnosis—and at the autopsy a large aneurysm of the pulmonary artery was found.

Palpation does not always settle cardiac problems, but it may do good in raising them. For instance, suppose that on palpation a definitely hypertrophied action of the left ventricle, with extension outwards of the apex, is felt. It is clearly our duty to search for some cause in the heart or in the circulation for this hypertrophy. If on auscultation a murmur, systolic in time, is heard at the apex, some are content to accept this as a sufficient explanation and to label the case one of mitral incompetence. This may be correct in some cases, but in my experience it is not common in the case of these young people to find mitral regurgitation leading to great hypertrophy of the left ventricle. Before accepting this as a complete explanation I would ask you to examine carefully for two other cardiac lesions. They will not probably both be present, but one or other will serve as a sufficient explanation. The one is aortic regurgitation, manifested by a diastolic murmur, heard either at the apex or along the left side of the sternum. The other is pericardial adhesions, usually between the pericardium and the chest wall, and tying down the ventricle to such an extent that it can only carry on the circulation by hypertrophy and dilatation. Either of those conditions will produce an amount of hypertrophy which is seldom met with in pure mitral regurgitation at this age, and one or other of them is usually present in such cases, although very easily overlooked. In the same way when we find overaction of the right heart, with heaving ensiform, and dullness and pulsation well to the right of the sternum, we must try to find some lesion which will explain this abnormality. It may be the result of mitral regurgitation but it usually is not. The commonest causes here are mitral stenosis or pulmonary stenosis, which should be carefully looked for. You will find that auscultation and palpation are mutually helpful—auscultation in that it leads one to seek for changes in the heart which result from the lesions which produce murmurs; and palpation in that it shows changes in the heart which lead one to seek out the lesions causing them.

TREATMENT.

The treatment of heart disease during adolescence has certain problems of its own. Perhaps the chief is the prevention of further rheumatic infection. Acquired and chronic

heart disease in early life is the result of acute rheumatic infection—for all practical purposes other causes may be left out. The danger of fresh infection or the recrudescence of latent infection persists as a real one all through adolescence. Sometimes this will take the form of an acute flare-up—acute rheumatic fever; in other cases it will be manifested by slight pains in joints or tendons or muscles, symptoms which do not incapacitate the patient but are unfortunately often accompanied by fresh endocardial and myocardial inflammation. Consequently my own practice is to treat those patients all through adolescent life with sodium salicylate. This need not be given continuously, but for a week or ten days in every month the patient should take from 40 to 60 grains of sodium salicylate daily. This is the best preventive treatment of further infection which I know.

Those patients who have a cardiac lesion but who present no symptoms under the ordinary routine of life had better be left alone so far as any heart treatment is concerned. In giving advice about their future life and work one will naturally select some occupation which does not involve great physical strain.

Those patients who have a cardiac lesion and who find that any extra exertion induces symptoms such as breathlessness had better be advised to observe their own limitations. Games and exercises should be regulated so as not to tire out the patient or produce cyanosis and breathlessness. In selecting a suitable occupation for their adult life it will probably be best for them to take up work of a sedentary nature so that the exercise they take may be independent of their work and not part and parcel of it.

Those patients who have a cardiac lesion accompanied by breathlessness and distress on any exertion do not require to be specially restricted. Nature will teach them to go slow and not to do themselves harm. They have permanently damaged and disabled hearts.

During adolescent life there is less call for direct treatment of the heart than either before or after. This is fortunate, for it spares me the trouble of going into the nature of efficient and direct cardiac treatment. I am afraid I should only have exposed my own want of knowledge of the whole subject of cardiac tonics and my utter scepticism as regards most of them. No one has yet discovered a tonic which will improve the action of a heart which has hypertrophied in order to overcome some permanent difficulty. No one has yet discovered a tonic which will restore that heart when once the muscular power has given out. The more one studies the action of the heart in health and in disease the more one is struck by its wonderful power in accommodating itself to meet the ever-varying calls of the circulation, and in overcoming the difficulties and new problems introduced as the result of disease. Of all the self-regulating mechanisms in the human economy there is none which surpasses the heart in the power of meeting difficulties in health and developing compensatory changes in disease. These inherent powers of producing compensatory changes in the heart, largely through hypertrophy of the cardiac muscle, are specially manifested during adolescence and are not to be relied on to the same extent in adult life.

We can ease the work of a diseased and labouring heart by giving it partial rest. We can remove all physical strain by keeping the patient in bed. We can cut off the sources of nervous disturbance in the brain by sedatives and narcotics. We can deal to some extent with difficulties in the circulation owing to the engorgement of other organs or the presence of oedema and effusion. These things are possible, but when it comes to be a question of direct action on the heart, treatment directed to improve the action of a failing heart, we are apt to discover our therapeutic helplessness.

Yet there is one condition in which the heart is disturbed and its action is impaired, not through the valvular lesion and not through myocardial exhaustion primarily, but as the result of an abnormally rapid rate. The cardiac force, as regards its pumping function, may be sufficient and the compensatory changes may be in working order, but the heart muscle shows signs of failure owing to the rapid rate present. If the heart rate is much increased the resting time of diastole is curtailed, and in the course of time exhaustion of the heart muscle follows. Many rheumatic hearts become disturbed at times during adolescence by the development of this rapid rate, which may be a regular action with a normal rhythm or an irregular action with an abnormal rhythm. In both of these cases it is the rapidity of the heart which is causing the trouble and which is calling for treatment. Under these

conditions we can employ digitalis with great confidence, giving full doses for a few days, and we shall often find that when the heart is slowed the patient is much relieved as regards all the signs and symptoms of cardiac failure. I do not say that digitalis will act in all cases. Unfortunately it will not, and we cannot at present tell why it slows the heart in some cases while in others it fails to do so. But it remains as our great sheet-anchor in cases of cardiac failure associated with a rapid action of the ventricles.

Hunterian Lectures

ON

MAN'S POSTURE: ITS EVOLUTION AND DISORDERS.

GIVEN AT THE ROYAL COLLEGE OF SURGEONS OF ENGLAND
BY

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[Abstracts.]

LECTURE II.—THE EVOLUTION OF THE ORTHOGRADE SPINE.

THE human spine is a mechanism of the utmost complexity; between the sacrum and the skull are incorporated twenty-four vertebrae, each provided with three short levers—the spinous and transverse processes; each lever is furnished with, not a single muscle, but a group of them. As we pass from sacrum to skull we find the levers change in length, strength, shape, and direction. The muscles, too, which move and balance the levers alter in disposition and strength as we pass from vertebrae to vertebrae. The twelve dorsal vertebrae, besides their three short processes, are provided with very long and powerful levers—the ribs. The student is so apt to concentrate his attention on the respiratory action of the ribs that he forgets that they are the most powerful of the spinal levers. All the muscles attached to ribs—the intercostals, the rectus abdominis, and oblique muscles of the belly wall—are prime movers and balancers of the spine. Here, then, is a very complex team for Nature to drive along a straight evolutionary course. By what process or processes did she succeed in transforming the multitude of spinal levers and muscles, which serve the needs of a pronograde monkey, into the forms which answer to the needs of man? Such a transformation seems highly probable if we suppose that, in the course of its evolution, the human stock has passed through two intermediate stages: (1) a *hylobatian* stage, represented by the small orthograde apes—the gibbons; (2) a *trogodytian* stage, now represented by great anthropoids such as the gorilla or chimpanzee.

THE SACRALIZATION OF BODY SEGMENTS.

With the evolution of the upright or orthograde spine there took place a shortening of the lumbar part of the spinal column, and my first effort will be to show you the means which Nature used to attain this end. In most pronograde monkeys, both of the old world and of the new, the three vertebrae* which become modified in the course of development to form the sacrum are the twenty-seventh, twenty-eighth, and twenty-ninth of the spinal series; Professor Wingate Todd¹ has given reasons for believing that the twenty-seventh vertebra formed the first of the sacral series in the earliest mammals. If one examines a number of monkeys of the same species it will be found that in some the twenty-seventh vertebra, in place of being purely sacral in its characters, shows some admixture of lumbar traits; in others, the twenty-sixth vertebra shows signs of forsaking some of its lumbar traits and adopting the characters of the first sacral. There is a tendency in some for the process of sacralization to spread in a tailward direction; in others in a headward direction. In small orthograde apes, represented by the gibbons, sacralization has spread headwards, so that the twenty-sixth now forms the first of the sacral series. In this manner a lumbar vertebra has been added to the sacrum of gibbons, giving them four sacral vertebrae in place of the pronograde three, but reducing their lumbar

* See a paper I contributed to the *Journal of Anatomy and Physiology*, 1902, vol. 37, p. 18.