A British Medical Association Lecture
ON
THE PASSIVE MECHANICAL FACTOR
IN HEART DISEASE:
ITS INFLUENCE AND MANAGEMENT.*

BY

ALEXANDER BLACKHALL-MORISON, M.D.,

F.R.C.P. LOND. AND EDIN.,

CONSULTING PHYSICIAN TO THE GREAT NORTHERN CENTRAL HOSPITAL;
CARDIOLOGIST TO MOUNT VERNON HOSPITAL.

(With Special Plate.)

By the "passive mechanical factor in heart disease" I understand the conditions which promote or impede the effective exercise of the active factor—the cardiac muscle. The matter so defined is, obviously, both physiological and pathological, and its adequate treatment might well occupy several lectures instead of one, but I shall endeavour so to deal with it that its practical importance may be apparent without claiming for it more than its due weight.

In his interesting Linacre Lecture on the "Law of the Heart," delivered in 1915, Dr. Starling has endeavoured to show the great adaptability of the heart in dealing with a wide variation in the quantity of blood it receives, in spite of much difference in the arterial pressure it has to overcome, and he experimentally tries also to demonstrate that the physiological law applies in disease when, in addition to variations of arterial resistance, mechanical disabilities, such as destruction of the aortic valves, is brought about. The output of the ventricle, within certain wide limits, remains the same provided the inflow be constant. Although under these circumstances altering the arterial pressure has no influence, a great effect is produced by varying the inflow. Increased inflow is associated with increased outflow, and there is no damming up of blood on the venous side. These conditions Dr. Starling illustrates by an ingenious heart-lung preparation, as he terms it, which he described in his lecture.

It must, however, be noted that, although Starling's detached or eviscerated heart disposes of large or small amounts of blood, with the same *sang-froid*, its rate is in no ways varied. It slogs at the same monotonous pace whatever work it has to do. This is not so *in corpore*. While, therefore, the heart may show, by some augmentation of total chemical changes, that it does more active work the larger the amount of blood it has to dispose of, there is evidently a passive factor in the situation which must be recognized.

Hill and Barnard, whose work on gravitation in the circulation is well known, maintain that the principle of the siphon is not applicable to the vascular system, as the arteries on the one hand and the veins on the other are of such different distensibility and elasticity.¹

But does this conclusion, even if correct, apply to the lesser or pulmonary circulation? Hill² prefers the analogy of the "rheochord" to the siphon as affecting, in any case, the systemic circulation, on account of the innervation of the vascular system. In the case of the cerebral circulation, however, even after the demonstration of its rich innervation, which I gave in 1898,³ he maintained the Monro doctrine, and, so far as I know, does so still. Bradford has also shown that the pulmonary circulation responds to nervous stimulation, but there is little doubt that both the pulmonic and cerebral circulation have, for physical reasons, a large passive element in their conduction.

In an exhaustive consideration of asphyxia neonatorum which was published in the *Lancet* of 1894, like Dr. Starling, I also constructed a heart-lung apparatus, but with the dead heart of a sheep, a system of tubes, and a lung bottle, which convinced me of the important rôle of the passive force of gravitation in the circulation as distinguished from the active forces of aspiration and propulsion. Still earlier, in 1878,⁴ an anatomical comparison of the heart and vascular system of the quadruped

and man appeared to me to argue the greater ease in the conduction of the erect as compared with the horizontal circulation.

But the investigation of the conditions of the pulmonary circulation is one of the most difficult tasks the physiologist can set himself, as is acknowledged in Tigerstedt's exhaustive article on the subject.⁵ It has nevertheless been reasonably concluded that the pressure variation in the pulmonary circuit is much less than in the systemic circulation and the velocity of the blood greater.⁶

In the supply from the right to the left heart a passive siphonage appears to play a large part, and, while a chemical estimation of cardiac energy may show, as I have stated, greater work on the part of the heart, the passive force referred to probably makes the efficiency of that organ under the circumstances possible. What, however, the experimental physiologist, with all his ingenuity and skill, fails to accomplish is at times brought about by what I have termed the vivisection of disease. In an address given to the Æsculapian Society of London, on cardiac motion as revealed by the vivisection of disease,⁷ I gave full particulars of the clinical history and necropsy of a case of sarcoma of the heart, which placed the right auricle and ventricle out of action, and in which the circulation was for long maintained by the left side of the heart alone. The patient ultimately died without giving any evidence of the anasarca or effusion into serous cavities which so limited an amount of cardiac activity would *a priori* have suggested as probable. The only portions of the right ventricle which could by any conception be regarded as effective were the right ventricular aspect of the septum ventriculorum and a small portion of the posterior wall.

That the circulation was effectively carried on thus for a considerable period is evident from the degree in which the heart was involved in the morbid process. It was not merely pushed aside by or adherent to the growth, as frequently happens, but was involved in it and replaced by it, a process requiring time. The aspiratory and propulsive aid of the right chambers of the heart may, therefore, be regarded as having been extinguished, and the process of their extinction was clinically marked by the gradual silencing of a rough tricuspid bruit—a bruit of tricuspid fixation—which was a marked feature of the case in its first period. The *cadaver* showed no oedema of limb or excess of fluid in the peritoneal cavity; 13 oz. of blood-stained fluid found in the right pleural sac could be accounted for by local conditions. Both lungs were adherent to the ribs and the base of the left to the diaphragm. No more striking evidence, in my opinion, of the passive force in the transmission of the blood from the right to the left heart could be conceived, and sections of the growth show that both the conus arteriosus and the aorta were held by it as in a vice, encroached upon but, of course, not occluded.

In this connexion I may also mention the conditions of the fetal circulation and of cases of dextro-cardiac malformation which survive in some cases to middle and even old age, and in others are so malformed as to render any length of life impossible. Communication with the left heart may be free, but active propulsion remains the rôle of the right heart. The passive siphon of the pulmonary circulation fails to be established or to be only inadequately established, and the length of life of the bearer of the lesion is in proportion to the dextro-cardiac activity thus imposed, although the most cardiacally malformed fetus, under the hydraulics of its circulation *in utero*, may come into the world well nourished and generally well developed. The maternal circulation, while anatomically distinct from that of the fetus, is so related to it, through placental pressure, as to transmit to it not only oxygen, but also nutriment and hydraulic force. In this case, whether the placenta be at a higher level than the fetal heart or below it, the passive pressure from a higher source remains, and the circulation is well maintained until pulmonic siphonage becomes due.

It has frequently been stated that there is a marked difference between the development of the dextral chambers in the fetal heart before and after birth, but at term the relative muscularity of the right and of the left chambers of the heart is very much that which it is in extrauterine life. I show a specimen to demonstrate this fact, obtained from a child 7 days old, who died of atelectasis pulmonum, and in whom the heart was normal.

* Delivered before the Edinburgh Branch of the Association on February 27th, 1920.

The fetal communications will be seen to be still pervious. On this subject Dr. George Alexander Gibson wrote interestingly, and it is a pleasure to me to recall his genial personality and his valuable work.⁸

The Oliver-Sharpes lecturer of 1911 and the Lumleian lecturer of 1917 both made merry in their orations over what they were pleased to consider "back pressure," an ancient and erroneous conception as a cause of cardiac failure discarded by the greater acumen of that new cardiology, of which they regarded themselves as exponents. The back pressure which they triumphantly demolished was, however, a creation of their own imagination. For no one ever called in question loss of power in cardiac muscle as being the cause of its failure. That waning power is, however, unquestionably the outcome in many cases of the additional work imposed upon it by mechanical passive defects. The Lumleian lecturer, however, remarked, "We do not fully understand the underlying causes of oliguria in cardiac cases, any more than we know the exact mechanism of cardiac oedema," and, unless the principle of the passive force in the lesser circulation be grasped and that of stagnation or back pressure be appreciated, it will be difficult, in my opinion, for anyone to find a satisfactory solution to these questions. By its aid the matter becomes clear.

One of the earliest signs of failure in the easy current from the right to the left heart is the occurrence of a reversible crepitation at the base, usually in the first instance, of the left lung. There are sufficient anatomical reasons, as I long ago pointed out,⁹ for the selection of this site. The patient manifesting it on left or right decubitus, as the case may be, by a single revolution on his own axis loses it in the uppermost lung.¹⁰

The French with their customary happiness of expression have termed this sign "*oedème à bascule*," a see-saw oedema. It is the first evidence of stagnation in the left horn of the pulmonary siphon. Normally this siphon carries the blood to the left ventricle, where, like a well-judged leg hit, the haemic ball is easily lifted into the far field of the arterial stream. But when the cricketer, the left ventricle, is fatigued, there is a degree of impotence in the stroke which declares failure, and the evidence of this is noted by the sign in question. It is the first stage in a stagnation which gradually extends or may extend to the whole venous inflow as ventricular effort wanes, and may, as we shall presently learn, even develop into a powerful retropulsion into the superior caval venous system and also into the abdominal venous cistern, of which the inferior vena cava is the mouth. Both the cardiac lung and the cardiac kidney are explicable by a recognition of lessened arterial impulse and increased venous sluggishness.

The heart, suspended in the thorax within its pericardial sac, has a certain relation to lungs, diaphragm, and thoracic parietes. Under normal circumstances these relations are perfectly adapted to allow, without friction, the limited changes in position and dimensions which occur in systole and diastole. These perfect relations may be disturbed by the genesis of states in which the normal territory of the heart is invaded or encroached upon or in which the heart itself oversteps its boundaries. By such encroachment or excursion the free and unhampered systole and diastole in the upper and lower chambers of the heart are interfered with, and, to use an engineer's phrase, which has also been used in this connexion by that master of the English language, our President, Sir Clifford Allbutt, the "sweet run" of the mechanism is replaced by friction, effort, impediment, and disadvantage. Even the beneficial action of agents which would otherwise prove successful in removing disability may from such passive causes be rendered nugatory.

The *exocardial* conditions which aid or interfere with this sweet run of the machinery, may be conditions of negative or positive pressure within the chest, intrathoracic inflammations of various kinds and in various phases, neoplasms, distortions of the cage in which the heart is contained and works, and certain developmental states, in which the viscera are transposed.

Hypermyosis, excessive hypertrophy or *cor bovinum*, is usually of endocardial origin or of this combined with exocardial tethering, but, as *cor bovinum*, its bulk alone in relation to the thoracic contents and wall may be regarded as an exocardial excursion beyond the normal boundaries of the heart. From mere bulk it may be one of the factors

deranging the smooth working of the organ and provoking signs of cardiac difficulty or distress.

Intrathoracic pressure under normal circumstances is always negative, but is at its maximum in inspiration and at its minimum in expiration. Inspiration is aspiratory from the venous cistern into the chest, and expiration is propulsive towards the left heart. The greater the inspiration the more the retention of blood in the chest by the thoracic vacuum, and, in dyspnoeic conditions, inspiration gains the upper hand, tending to dilate the auricles and to a less extent the ventricles, but the thin-walled upper chambers naturally feel this force most. This is well shown on a model devised by Herman.¹¹

The asthmatic, with his prolonged expiratory snore or wheeze, acquires a fixity of chest which impedes the venous inflow, and experience teaches us that this condition may be most promptly dealt with by a rhythmical compression of the chest in the expiratory phase of respiration. In some cases I have prescribed nothing else, when the chest has retained resiliency.¹²

What we may at any time demonstrate in the dyspnoea of the asthmatic we also see in that of the cardiopath, and here, when lung and heart are fixed by adhesions, impeding the rhythmical play of the former and the contraction or systole of the latter, we have to deal with conditions which induce in many cases a hypertrophy of heart which causes that organ, by its mere increase in muscle and its greater blood content, still farther to disturb the balance between the respiratory and circulatory pumps.

In these circumstances we may struggle in vain to relieve the burdened and tethered heart by cardiac tonics or by any other means, but may, by affording the organ more room to act in, transform in some cases a hopeless condition of embarrassed circulation into one of comfort and convalescence by cardiolysis or thoracotomy. To this point I wish to draw particular attention because of its great practical importance.

Brauer of Heidelberg first practised cardiolysis for symphysis cordis in 1902,¹³ and I used the same method for hypermyosis without symphysis in 1907.¹⁴ I have now had considerable experience in precordial thoracotomy, but a detailed discussion of the measure is not possible in this place. I wish, however, to state with all the emphasis in my power that the surgeon may here at times, under the guidance of the physician, very effectively come to the aid of the latter, and shall relate one experience on this point which will speak for itself.

Just as the indication for most fruitful venesection is a degree of venous turgidity which is associated with cyanosis, so the best guide to precordial thoracotomy is an amount of systolic movement in the costal or sterno-costal thorax which is the necessary consequence of an enlarged and usually adherent heart. For obvious reasons this is best seen in the young, in whom also a permanent precordial bulging may indicate the adaptation of a mouldable cage to its enlarged cardiac content. With such adaptation there may be an absence of signs warranting thoracotomy. The *modus operandi* of the measure is simply the relief of intrathoracic pressure resulting in greater mobility of heart and improved siphonage, the more resilient the chest the better the result as a rule, which is not, however, without exceptions.

The question of an anaesthetic has given me much concern in these cases, and I have known one to die on the table under general anaesthesia before operation could be undertaken. But that anxiety is now a matter of the past, for I have found that local anaesthesia serves every purpose and is safe. In the following case the measure indicated definitely saved life.

F. P., aged 10 years, was admitted under my care to the Great Northern Central Hospital on February 11th, 1919, with a history of having suffered six weeks previously from rheumatic fever, and of having been confined to bed since. On admission he merely complained of pain in his arms and legs, but his face had begun to swell. The temperature varied from 98° in the morning to 99° in the evening; the respirations ranged from 24 to 26, and the pulse from 96 to 120. It had evidently not been his first attack of rheumatism. The area of cardiac dullness was large, and he had a well developed apical systolic bruit. The lungs were, however, clear, and the urine free from albumin. There was visible and palpable pulsation from the fourth to the sixth interspace, and the cardiac impulse heaved the thorax. A rough systolic bruit was audible at the apex, traceable to the left and heard in the back. The second sound was accentuated in the area of the pulmonary arterial cusps,

and in the aortic area was closed. He improved under treatment, and a month later was sent to a relief hospital in connexion with the Great Northern Central, but was readmitted on April 23rd, 1919, with a history of having exhibited swelling of the face and abdomen two weeks previously. He then showed enlargement of the liver with effusion into the peritoneal cavity, and crepitation and rhonchi on auscultation at both pulmonary bases.

By radiography the cardiac area was seen to be much enlarged (Plate, Fig. 1) and free fluid was removed from the abdomen by paracentesis on April 24th. The patient, who was all this time confined to bed, was taking digitalis and continued to do so, fruitlessly, for the following month. During this time his condition became steadily worse and his legs and body generally anasarctous, the liver enlarged in size, the peritoneal effusion reaccumulated; there was effusion also in the left pleural cavity, the urine became albuminous, and I regarded the condition of the patient as hopeless.

Believing I had to deal with an enlarged and adherent heart, I advised cardiomyolysis as the only hope of altering the situation, but expressed a cautious prognosis as to even temporary benefit, stating that, so far as I could judge, death was otherwise inevitable. After some days' reflection the parents consented to operation, and I asked my colleague, Mr. Barrington-Ward, to remove ribs and cartilages in the area of maximum impulse to the left of the sternum. I stipulated, however, that the operation should be performed under local anaesthesia, as I did not consider that the patient could survive a general anaesthetic.

Operation.

On May 23rd, 1919, portions of the sixth and seventh ribs with their cartilages were removed. The periosteum was not peeled off the pleura, but was divided to relieve tension. The left pleural cavity was accidentally punctured and fluid escaped, air at the same time being sucked in on inspiration. The puncture was closed by suture. The patient cried a good deal during the operation, but confessed afterwards that he had been more frightened than hurt. I confess that, even after the operation, I was not sanguine of any striking benefit, and was, consequently, astonished by the result, which was chronicled, to my dictation, in the notes of my house-surgeon, Captain Huxtable, M.C., as follows:—"29/5/19 (that is, six days after the operation): pulse 108, regular; respirations 36; anasarca entirely disappeared; no free fluid in peritoneum; no swelling of face; subjectively quite comfortable; sleeps quietly, which was not the case previously."

The use of digitalis was continued after the operation and later abandoned. When it had been left off for a time there was some return of peritoneal effusion, the liver remaining enlarged. The fluid was drawn off, the digitalis resumed, and the effusion did not recur. The pulse throughout was regular, but of rather high rate (112-120). The patient was later removed to Mount Vernon Hospital, to be under my care, and is there now, more as a convalescent than as a patient (Figs. 4 and 5). He travelled to London recently for the purpose of radiography, and I exhibit the radiograms made. The chief

FIG. 5.—A, Cardiogram. This case having been a low thoracotomy (sixth and seventh ribs) and the periosteum not having been removed, the systolic excursion of the ventricular systole is not so pronounced as in a higher thoracotomy. B, Radial pulse of low tension.

difference these exhibit is in the diminished distension of the right heart and a clearer distinction between it and the diaphragm on the right side in those taken after the operation as compared with the skiagram taken before operation, in which an inclined plane runs from the right heart directly into the liver shadow (Plate, Figs. 2 and 3).

Present Condition.—He has gained 9 lb. in weight since admission; pulse regular, rate 84 and of good force. There is heaving pulsation in the precordium, including the lower half of the sternum. The area of cardiac dullness is 5 in. by 7 in., the right border of dullness extending 2 in. to the right of the mid-sternal line. The apex beat is in the fifth space $3\frac{1}{2}$ in. from mid-sternum. A systolic bruit of musical pitch is traceable to the left posterior axillary line and as far as mid-sternum to the right. The bruit is audible also in the back and loudest in the left paravertebral groove. The second sound is accentuated over the pulmonary arterial valves and is normal in the aortic area, the sigmoid valves closing. The respiratory rate is 30, the pulmonary auscultatory signs are quite normal, as are the dimensions of the liver. There is no abnormal effusion into any tissue or cavity. The systolic blood pressure is 95 to 100 mm. of mercury and the diastolic 65 to 70 mm. He takes moderate exercise without any discomfort.

Nothing further need be said of the other *exocardial* conditions mentioned than that their effect is to crowd intrathoracic space and disturb the balance of pressure within the thorax, as well as to hamper the action of the heart, and deprive the blood stream of the full force of the

passive element in its transit which we have discussed, and incidentally to render more difficult the chemical interchanges of the respiratory function. The removal or diminution of such positive intrathoracic pressure when possible is obviously indicated, and it is usually possible, except in developmental states, by well-recognized means which need not be further considered.

The *endocardial* passive factor in heart disease is mainly valvular. In the period immediately prior to the war a certain school of thought, already referred to, covered with scorn both the undue importance attributed by some to valvular aid in the circulation, and the old and well known conception of what was and is termed "compensation" in the cardiac mechanism, for conditions rendering the circulation more difficult. In itself the term, properly understood, offers no serious objection. To the establishment of compensation the slow onset of difficulty is necessary. The physiologist may destroy valves in the laboratory, and, notwithstanding, for a time register a satisfactory arterial pressure, in such an apparatus as that of Starling already mentioned, but the clinician knows that rupture of a valve in man—a rare event—is frequently very rapidly fatal even in the right heart. The organ has not time afforded it to establish what is quite intelligibly termed compensation, that is, sufficient muscular force behind the suddenly disabled valve to enable the circulation to be efficiently maintained.

I know no more striking proof of the value of the passive valvular factor than is afforded by observation of the circulation in the heart and vessels before the valves have come into being. This is best observed in the transparent embryo of the fish, in which the powerful ventricle void of valve may be seen to be associated at the aortic end with propulsion in the systolic phase and with reflux in the diastolic, and at the sinistral end with reflux in the systolic and heart-ward aspiration in the diastolic phase. In the dorsal aorta the blood stream may also be seen to be not steadily progressive but swinging to and fro in a see-saw manner, in obedience to the systole and diastole of the ventricle. In short, in the embryo fish may be seen the best demonstration possible of the Corrigan pulse of well-marked aortic insufficiency. After the formation of the valves and increase of rate in the heart's action the stream becomes purely progressive, and in high rates of heart-beat as continuous as the venous inflow.

In this connexion I may be permitted a few words on methods of examination for the purpose of diagnosis, prognosis, and treatment. While recognizing the general interest, in some cases the utility and in others the necessity for examining a patient by graphic methods mechanical and electrical, and adding radiospection to the employment of our eyes and fingers, it will not surprise you if I express the hope that there may be no abatement in the assiduous cultivation of the old and classical methods. Every method must be used to determine not only the condition of the cardiac muscle, but also which of and to what extent the passive mechanical factors are involved in any given case.

In practice the *degree*, moreover, of the valvular deficiency or obstruction in any case is important. As I have endeavoured to show, the largely passive siphonage from the right to the left heart is effective up to the left ventricle. The featureless left auricle with the undefended pulmonary veins show how much is expected from the powerful mitral valve, and here also the *degree* in which the auricle is defended is important, as also is the *ease* with which the entrance of the blood into the ventricle is secured.

The depreciation of the importance of the passive valvular factor before the war, under the influence of the school of thought mentioned, was responsible for the passing into the army of a good many men who, from valvular defect, were incapable of standing the strain of war conditions. The lesson drawn from experience in the war was the revision on the part of these optimists of their views, certainly as regards the influence of aortic and mitral stenotic lesions; but they still retain some impenitence as regards mitral reflux. It is their last ditch, and they fight valiantly in it.¹⁵

But it may be said, cases of both aortic and mitral disease may be adduced which passed through the strenuous experience of war without disability. Such certainly occur, but not frequently, and here again the

degree of defect is as important as the nature of it. How can this be gauged? He would be a bold, not to say an imprudent, man who claimed precision in his estimate of this condition—in the answer, that is, to this question. But it may be affirmed with reason that the determination of the systolic and diastolic blood pressure by the sphygmomanometer is an aid not to be despised. The low diastolic figures of aortic regurgitant disease frequently reveal a considerable degree of this lesion, even when auditory and other signs would lead to a more favourable estimate, and the value of the aortic platform is nowhere better shown than in the normal ventricle behind a large aneurysm of the aorta when the valves are perfect, and the hypertrophied chamber behind valves which leak under these circumstances.

Similarly the height of the systolic blood pressure and the size of the pulse form a useful gauge of the freedom of entry of blood into the left ventricle through a stenosed mitral orifice. With pronounced muscular failure stenosis of the aortic orifice may reach such a degree as to render the radial pulses imperceptible and the diagnosis of any valvular lesion whatever impossible. This was so in a case from which I show a specimen.

On the other hand, obstruction at the mitral orifice may so engorge the right heart as to cause the right ventricle to throw a powerful systolic wave upwards into the superior caval venous system, and downwards into the inferior cava and hepatic sinuses. This may be seen in cases both with and without associated auricular fibrillation. It must be remembered that right ventricular systole occurs during right auricular diastole, and that the auricle may contract for a time more or less efficiently during the subsequent ventricular diastole, even when right ventricular retro-pulsion through a defective tricuspid valve has for some time been well marked. In one case, the heart from which is shown—that of severe mitral stenosis—there was present at the same time heart-block, probably of the left limb of the auriculo-ventricular bundle, according to electro-cardiography, for I postponed the anatomical investigation of the point until I had shown the specimen here, so as not to damage the aortic orifice.* In another case, the heart from which is also shown—that of aortic valvular disease and stenosis both of the mitral and tricuspid orifices, and all due to rheumatic endocarditis—the powerful right ventricular retro-pulsion, as exhibited in the phlebogram shown, and which was grossly visible to the naked eye, was associated with a regular pulse, indicating the absence of auricular fibrillation. At a later period, however, after a severe attack of paroxysmal tachycardia lasting for some days, a persistent irregularity of heart's action (auricular fibrillation) became established. This was also the sequence in a case of persistent *foramen primum* with hypertrophied right cardiac chambers, of which I recently published an account.¹⁶

But paroxysmal tachycardia may be followed by ventricular failure with the development of well-marked mitral reflux and death, but without any auricular fibrillation. In such a case observed by myself, the paroxysmal tachycardia was sudden in onset and as sudden in cessation, and of about twenty-four hours' duration. As, however, is often observed, such tachycardias may recur with some frequency, without entailing either auricular fibrillation or ventricular failure as a consequence. This condition was wont to be regarded as a cardiac neurosis, a term less discredited now than it was prior to the demonstration of the full innervation of the excitatory system of the heart.¹⁷

In these attacks we have also to remember, as Martius¹⁸ and others have pointed out, that auricular repletion and distension is a late, not early, condition in a valvularly sound heart. The tachycardial ventricle, unequal to sufficient output, accumulates blood which gradually distends and disables the auricle, which may collapse in fine tremor and systolic impotence—conditions synonymous with fibrillation of that chamber.

Similarly, there is no doubt that the ventricle, accelerated by exertion or emotion, or by both these states combined, may likewise distend and disable the auricle, even when the general condition of its musculature is healthy. This

is especially prone to occur if there be at the same time some local passive mechanical impediment to easy blood flow through the heart. I show a specimen from such a case which occurred in a youth during a football match, in whom *post-mortem* examination, some years later, showed only a slight degree of mitral stenosis. In this case thoracotomy had been performed, and I show a photograph and some tracings. I have observed the same sequence in another case, in which an active workman who helped to extinguish a fire broke down in auricular fibrillation immediately afterwards. In him also, some years later, I found a definitely stenosed mitral valve, due, so far as could be ascertained, to an almost forgotten chorea in very early life. Why, then, is auricular fibrillation not more frequently observed in cases of cardiac failure with aortic valvular disease? Of the failure in such a case I show an instructive series of tracings, together with a photograph of the heart and an exhibition of the effect upon it of sufficient doses of digitalis. The comparative rarity of auricular fibrillation in aortic valvular failure, as well as the rarity of dropsies in such cases, is, I believe, mainly due to the protection of the pulmonary circulation by the powerful mitral valve and the delaying of that much criticized back pressure or venous stagnation which is favoured by an undefended or obstructed left auricle. It is a matter of experience that few aortic valvular cases die waterlogged. As Dr. George Balfour states, "It is only rarely that dropsy and other secondary diseases of a serious character are established in such cases."¹⁹

It might, indeed, be argued in a case such as that to which I have referred, in which a degree of heart-block was present in association with auricular fibrillation, that the blockage was advantageous, rather than otherwise, by reducing the ventricular rate independently of the normal relation between the lower and upper chambers of the heart. This case is at once an example of that vivisection by disease to which I have already referred, and of that series of interstitial passive and pathological factors which it is impossible within the limits of our time to discuss to-day. It also showed a positive Wassermann reaction and had a cirrhotic liver, which I exhibit, and died waterlogged, but very slowly. This liver, as well as that from another case with powerful venous retro-pulsion, which I also show, demonstrates, in the wide and gaping inferior caval entrance and dilated hepatic sinuses entering it, the effects of the powerful venous retro-pulsion which both exhibited and phlebograms of which I have shown.

Again, when we consider the *duration* of some cases of pronounced auricular fibrillation of the tachycardial type, compatible more or less with corporeal activity and often with little subjective discomfort, we may reasonably attribute this to the presence in such cases of a sound valvular apparatus. I show a radiogram from such a case of eleven years' standing, in which it will be observed that there is very little evidence of dilatation of any of the chambers of the heart.

I submit that the continuance for any length of time of auricular fibrillation, with ventricular action of high rate, in cases of valvular disease of the heart is a very rare event. In auricular fibrillation with tachycardia in these circumstances, either the rate must be reduced more or less promptly or the patient dies. No better evidence of the value of valvular integrity need be sought than this fact, which all who have had much clinical experience will, I feel certain, be in a position to confirm.

While, therefore, from the time of Harvey to the present moment, no one has disputed the pre-eminence of the muscular factor in maintaining the circulation of the blood, the importance, likewise, of valvular integrity and freedom in movement of the heart is undeniable.

The combination of exocardial and endocardial passive mechanical factors finds a place in many cases of heart disease—in some the one class and in others the other preponderating. The management, therefore, of the problems involved is, like the factors themselves, one which combines dealing with both. Thus the thoracotomy or other method used for the removal of exocardial impediment may be necessary to the successful treatment, by appropriate agents, of disorders due to or aggravated by defects of passive endocardial factors. In considering the passive exocardial factors, we found that to influence beneficially the action of the cardiac muscle we had to deal with impediments to its action outside the heart

* Anatomical examination since made showed that the branch of the bundle involved was the right, not the left. For confirmation I submitted sections to Professor Arthur Keith, who kindly examined them with me.—A. B. M.

itself. In treating the endocardial passive factor we can only influence it by action upon the cardiac muscle and the blood content of the heart.

In the management of the passive mechanical factor in heart disease, a maintenance of efficient respiration is of the first importance. One of the earliest evidences of imperfect circulation through the heart, it has long been recognized, is some degree of embarrassment of respiration, of what has conventionally been called breathlessness and technically dyspnoea. It may be very slight, transient, and only evoked by circumstances which make an additional call on the heart by exertion, not only of the organ but of the organism. Or it may be more urgent and a consequence of failure of the organ itself to do its particular work, even when the organism as a whole is resting. In any case, the condition has to be dealt with on three lines—namely, by posture; in certain cases by rhythmical manual expiratory pressure and it may be also by manipulatory inspiration, in short, by artificial respiration; and thirdly, by strychnine.

Strychnine is to the distressed pulmonary function as needful as digitalis is to the disordered cardiac muscle. So much so is this the case that it has gained a deservedly high reputation as a cardiac tonic.

The laboratory experimentalist, and even some clinicians, are inclined to believe that strychnine has no effect on the heart. Experience, however, has shown incontestably that the dyspnoea of cardiac failure is relieved by the use of strychnine; and as such dyspnoea is a result of failure of the heart, the latter must be regarded as also eased by strychnine indirectly. This indirect benefit is due to improved respiration and a consequent promotion of better siphonage in the cardio-pulmonary circulation. The powerful action of strychnine on the respiratory centre and upon the spinal cord in raising its reflex irritability is not denied, even by the laboratory therapist.

The so-called Nauheim treatment owed, in my opinion, much of the good produced in the cases benefited by it as largely to the effect of the exercises practised on the respiratory function as it did to its effect upon the heart itself, or to the balneological element introduced. So, too, in so-called Oerfelism, with its hill-climbing and its restricted imbibition of fluids, the improvement of respiratory capacity with the more free cardio-pulmonary siphonage resulting, played no small part in the general relief experienced in cases suitable for that mode of treatment. That some who were submitted to it died on the hillsides they climbed, was a regrettable detail.

As regards the blood content factor, no one can have removed a large clot from one of the chambers of the heart *post mortem*, and witnessed its immediate shrinkage from elasticity, without being struck with the importance of this mechanical factor in dealing with the engorged heart. This effect, naturally, is most evident in the thinner and more distensible auricle, in which the shrinkage reminds one of the contraction of the uterus after removal of the placenta or of blood clot.

With such cardiac dilatation during life valvular incompetency frequently occurs, although even this has been denied by some. Sir Charles Ballance, in his recent lectures on the surgery of the heart,²⁰ properly regards any direct cardio-puncture for this state as a "freak operation," and one not to be undertaken lightly, but quotes Dr. Sloan's well known case, in which life was saved by puncture of the heart. In this case the needle of an aspirator was plunged into the heart in the fourth space half an inch to the left of the sternum. It must, therefore, have entered the left ventricle, and while ten ounces of "pure blood" were withdrawn, it is probable that direct stimulation of the muscle must have played a part in restoring its activity.²¹

But while such an heroic measure may not be frequently justifiable, venesection, which our ancestors practised too indiscriminately, still has a place in the rational treatment of heart disease.

By the use of a model of the heart-lung type²² it may be shown that any interruption to the venous inflow at once facilitates cardio-pulmonary siphonage and shrinkage of the dextral chambers of the heart. This limitation of inflow the distressed cardiopath seeks by the assumption of the orthopnoeic position, in which gravity retards dextro-cardiac repletion. Dr. George Balfour, whose memory is still green among Edinburgh men, laid much stress on securing recumbency in aortic valvular reflux, and advised the use even of an anaesthetic to lay the orthopnoeic flat.²³

But, according to the views expressed in this lecture regarding the passive factors in the circulation, the effect of gravity on the inferior caval column and the siphonage of the pulmonic circuit favoured by the posture in orthopnoea, call in question the desirability of changing the upright position for one in which, whatever its effect in diminishing aortic reflux, there is no question as to its favouring dextro-cardiac repletion and increasing pulmonary embarrassment. Were it indeed possible to compress the inferior vena cava without at the same time obstructing the aorta, which unfortunately it is not, such caval compression would have the same effect upon the cardio-pulmonary circulation as a free venesection and without the effusion of blood.

In the presence of cyanosis venesection is certainly indicated, and I have practised it myself with benefit under these circumstances. In aortic valvular cases Hodgkin²⁴ found bleeding of little service, and the reason for this is evident from the above remarks. To appreciate fully the benefit to be derived from venesection in suitable cases one must, however, consult the experience and practice of our predecessors of a past generation. Thus, we read that in a case of tricuspid and pulmonary obstruction Mr. Kinglake bled the patient during two years upon 312 occasions, to the average amount of 4 oz.—that is, to a heartful—and states that to describe the benefit gained by each bleeding would be to express the difference between the most affecting pain and comparative ease.²⁵

Venesection, like cardiolysis, may be a necessary preliminary to the effective use of that agent, indispensable to the cardiologist for action upon the passive endocardial factor—namely, digitalis—concerning which he is prone to wax eloquent.

"Dotted all over our upland pastures," writes Dr. George Balfour, "there is no nobler plant of our indigenous flora than the *Digitalis purpurea*, and there is no more potent benefactor of mankind among the many constituents of our materia medica."²⁶ There are also few agents concerning the *modus operandi* of which there has been more controversy. To discuss this question in any detail would require a lecture to itself. What, however, every clinician has convinced himself of is that in digitalis he has a trusty means whereby he has on numberless occasions benefited a disordered heart and with which he has at times positively saved life.

If we believe that all cardiac muscle, excitatory and executive, is under the control of the inhibitory, accelerant, and augmentative nervous system, and we succeed by medicinal agents in inhibiting, accelerating, or augmenting cardiac action, it seems reasonable to assume that we do so by the effect of the agents in question upon the nervous system proper for the control or execution of these actions. The only alternative would be to suppose that the use of certain agents renders the cardiac muscle refractory or resistant to their action, or, on the other hand, impulsively obedient to them.

But, leaving speculative disquisition and betaking ourselves to the important field of clinical observation, we can take firm ground on certain facts of experience. If such an attitude be regarded as empirical, so be it. From our empiricism emerges benefit. The most striking results of digitalis are unquestionably observed in the control by it of the paroxysmal tachycardial irregularities of the heart's action found in association with mitral valvular disease, whether obstructive or regurgitant. The heart slows, the tumult is stilled, and quiet reigns. Older writers therefore regarded digitalis as the "opium of the heart," and one of the most recent²⁷ considers it hypnotic to that organ. But it is not only in tumult with irregularity, but in tumult—that is acceleration—with regular action, that this drug is found to still the storm. The regular failing aortic valvular heart may be succoured, as well as the irregular failing mitral heart, and the failing heart which is neither aortic nor mitral, but has a sound valvular apparatus. These facts are undeniable by clinicians of any experience, and the result is an improvement of the mechanical efficiency of the passive valvular factor when this is defective, and an entire restitution in some cases of its action when the valves are without organic disease.

Cardiac action has always been known to be auriculo-ventricular in sequence, from the upper to the lower chambers, but it is only in our own day that the channel

for this normal sequence has been discovered. We have, moreover, learnt still more recently that this channel, like all muscle throughout the body, is under nerve control. The retardant and accelerant nerves must therefore act through this channel in an important measure. In this connexion one of the cases I have mentioned appears to be instructive—namely, that in which there was at the same time auricular fibrillation and ventricular retardation. The auricular storm continued, but the ventricular action remained slow, though somewhat irregular, because the storm—for we may regard the quiver of fibrillation as storm—in the upper chamber could not spread to the lower on account of anatomical blockage. When digitalis lays the general auriculo-ventricular storm, it lays wholly or in great measure both the auricular and ventricular tumult. Which does it still first and how does it still it? It is conceivable that it may act by interrupting, as in pathological block, the passage of impulses from the auricle to the ventricle, so quieting the latter, and, with a subsidence of tumult in it, allowing the impotent quiver of the former to recover. This view would be favoured by Thomas Lewis, to whom we owe the view that auricular fibrillation is the cause of ventricular irregularity. Or, as Professor Cushing has suggested, digitalis may by some undefined direct action on the ventricle restore its tone, and with this, necessarily, a restoration of efficiency of the auriculo-ventricular valves would follow, and so protect the auricles from distension as to enable them to recover. Between the latter view and older conceptions of the restoration of ventricular elasticity or tone there does not appear to be much difference. In any case the slowed ventricle, with increased contractile power and restored elastic recoil, implies both better closure of the valvular apparatus and improved aspirative action, which promote an easier siphonage in the cardio-pulmonary circuit. What I have said of digitalis applies, of course, to the whole group in which it is *facile princeps*, *strophanthus* alone approaching it in power and reliability.

Did time permit, other agents, such as atropine, camphor, and ether, etc., might in this connexion be fruitfully considered from the clinician's point of view, but I must desist.

I have thus endeavoured to call attention afresh to the passive mechanical factors in heart disease, which have in recent years, in my opinion, been unduly and unwisely ignored or minimized, but I trust I have done so without claiming for them more than their due weight. I have also pointed out that the removal when possible of passive impediment to the heart's action, and the remedy or modification, also when possible, of defect in the action of the passive mechanism within the organ, as well as placing the patient in a position in which he may derive the greatest benefit from the passive as distinguished from the active forces in the blood flow through the heart, are essential to dealing successfully with ineffective action on the part of that all-important organ.

REFERENCES.

- ¹ *Journ. of Physiol.*, 1897, vol. xxi, p. 322. ² *Journ. of Physiol.*, 1895. ³ *Edinburgh Med. Journ.*. ⁴ Dextral Valvular Disease, *Edinburgh Med. Journ.*. ⁵ *Ergotisme et Physiol.*, vol. ii, part 2, 1903. ⁶ Howell, *Textbook of Physiology*, ed. 1915, p. 518. ⁷ *Lancet*, January 9th, 1909. ⁸ *Edinburgh Med. Journ.*, 1892. ⁹ *Practitioner*, 1892. ¹⁰ *Lancet*, 1911, vol. i, p. 1202. ¹¹ *Physiology*, transl. by Ganguee, p. 160. ¹² *Lancet*, November 25th, 1905. ¹³ *Arch. f. Klin. Chir.*, vol. lxxi. ¹⁴ *Lancet*, 1907, vol. ii. ¹⁵ Medical Research Committee Report. ¹⁶ *Anatomical Journal*, vol. liv, part 1, October, 1919. ¹⁷ *Journ. of Anat. and Physiol.*, 1912. ¹⁸ *Die Tachycardie*, 1895. ¹⁹ *Diseases of the Heart*, p. 55. ²⁰ *Lancet*, 1920, vol. i, p. 74. ²¹ *Trans. Med.-Chir. Soc. Edin.*, 1894-95. ²² *Lancet*, 1904, vol. i. ²³ *Diseases of the Heart*, ed. 1898, p. 103. ²⁴ *Lond. Med. Gaz.*, vol. iii, p. 442. ²⁵ *Lond. Med. Journ.*, vol. x, part 4, 1789. ²⁶ *Op. cit.*, p. 359. ²⁷ *BRITISH MEDICAL JOURNAL*, 1919, vol. ii, p. 621.

ACCORDING to the report of the South Travancore Medical Mission for 1919, the mission hospitals, by inducing larger numbers of well-to-do patients to pay for their treatment, have made further progress towards self-support. During the year 657 major and 6,165 minor operations were performed. The major operations included 37 gastro-enterostomies and 6 operations for amoebic abscess of the liver; the latter was treated by puncture by a large trocar and drainage by a tube subsequently introduced through the cannula. The medical mission, which is administered by the London Missionary Society, also has charge of homes for leprosy men, women, and children. All the hospitals are greatly in need of linen and nursing appliances; gifts and subscriptions may be sent to Dr. S. H. Pugh, Neyoor Hospital, South Travancore.

DEFICIENCY DISEASE:

WITH SPECIAL REFERENCE TO GASTRO-INTESTINAL DISORDERS.

A BRITISH MEDICAL ASSOCIATION LECTURE DELIVERED TO THE SOUTH WALES AND MONMOUTHSHIRE BRANCH.

BY

ROBERT McCARRISON, M.D., D.Sc., LL.D.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; BREVET LIEUTENANT-COLONEL, INDIAN MEDICAL SERVICE.

[With Special Plate.]

CUSTOM has sanctioned the application of the term "deficiency disease" to a group of maladies the chief causal factor of which is deficiency in the food of certain substances other than proteins, carbohydrates, fats, and salts. These substances are spoken of as "accessory food factors," or more commonly "vitamines";* they are not foods in the sense of tissue builders or producers of energy; they aid the body to utilize food material sufficiently and its cells to perform their functions. Their true nature is unknown, but probably they are of the nature of organized catalysts or enzymes. They are found in the germs and peripheral layers of such seeds as wheat and rice, and throughout the whole seed in peas, beans, and other pulses. They occur abundantly in the cells of yeast, the yolks of eggs, and in varying proportions in the different parts of plants and in the organs and tissues of the animal body. They are more plentiful in the brain, liver and kidney, than in the muscles. Animals derive them directly from the plants they eat, or indirectly from fresh animal foods. It does not appear that the animal body is capable of manufacturing them itself. Some are soluble in oils and fat, and are contained in the majority of animal fats. Lard is said to be an exception, but though this is no doubt true of stall-fed pigs, I doubt whether it is true of pigs living on raw vegetable foods. These fat-soluble or A-vitamines readily undergo decomposition; they do not exist, or but sparingly, in vegetable oils; consequently artificial substitutes for butter, when made from vegetable oils, do not contain them. They occur in abundance in the green leaves and growing parts of plants, but are deficient in roots and root vegetables. Butter made from the milk of cows fed on green fodder contains a substance which tends to prevent oedema in pigeons fed on autoclaved rice, while that made from the milk of cows fed on dry fodder does not do so to the same extent. There is reason to believe that the absence of these fat-soluble or A-vitamines from the dietary, or of vitamins allied to them, is the actual or predisposing cause of rickets. Other vitamins are soluble in water and are known as B-vitamins. The chief sources of them is grain, fruit, and egg yolk. As they are soluble in water they are liable to be dissolved out of grains and fruit in the process of boiling; as they are concentrated in the germs of seeds they are frequently removed in the milling process. They resist considerable degrees of heat. They are not destroyed by boiling for a short time, but exposure to temperatures up to 130° C. for an hour and a half destroys them. They are commonly referred to as "antineuritic vitamins," since their absence from the food is the actual or predisposing cause of beri-beri.

Vitamins of a third group, the "antiscorbutic" or C-factors, are also soluble in water; they are found in the green parts of plants and vegetables and in fruit. They are unstable, and are very susceptible to the action of heat and alkalis. They are thus readily destroyed in the process of cooking, especially—as often happens—when sodium bicarbonate is added to the water in which vegetables are cooked. Dry grain does not contain them; they develop when the seeds germinate. Tinned or dried vegetables and fruit may contain them, but in reduced quantity and quality, and not in such perfection as the fresh raw products. Experiment shows that among the food materials lemon juice and cabbage juice are richest in them; fresh milk contains them in small amount only.

* The terms "deficiency disease" and "vitamine" are used in this lecture because both are in current use, but neither is satisfactory. Professor H. E. Armstrong, F.R.S., has proposed to replace the term "vitamine" by "adivant," indicating that the substance is necessary to life.

A. BLACKHALL-MORISON: THE PASSIVE MECHANICAL FACTOR IN HEART DISEASE.

FIG. 1.—Before operation. April, 1919. Skiagraphed by Dr. John Morison. Obtuse right cardio-phrenic angle. Transverse measurement of thorax $2\frac{1}{2}$ in.; of heart $1\frac{1}{2}$ in.

FIG. 2.—After operation. August, 1919. Skiagraphed by Dr. John Morison. Acute right cardio-phrenic angle. Transverse measurement of thorax $2\frac{1}{2}$ in.; of heart $1\frac{1}{2}$ in. Sixth and seventh left ribs seen deoasted. Focus the same in both figures. Blood entering and leaving the heart more easily. Respiration improved. Note narrowing of cavo-aortic zone and increase of auriculo-ventricular diameter.

FIG. 3.—After operation. February, 1920. Skiagraphed by Dr. Martin Berry. Conditions the same as in Fig. 2. Focus longer. Object slightly more magnified.

FIG. 4.—F. P., aged 11. Mitral regurgitation, with symphysis cordis and cardiac hypertrophy. The patient has now gained weight, and the enlargement of the liver has disappeared. In the photograph the liver is still enlarged. Note the total absence of all anasarca, which was marked when the case was operated upon. Note also the cicatrix of the wound over the site of the removed ribs and cartilages.