REMARKS

ON

THE FORM AND MECHANISM OF THE HEART.

Notes of a Lecture given in St. John's College, Cambridge.

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WHEN Harvey (of Caius) first attempted to follow with his eye the movements of the heart, as he saw them taking place in newly opened animals, he tells us that their suddenness and complexity baffled him; *ita ut modo hinc systolen illinc diastolen, modo e contra, modo varios, modo confusos fieri motus ne existimarem cernere (De Motu Cordis)*. This indeed is hardly to be wondered at. The entire cycle of the heart's movements may occupy one-half to two-thirds of a second. In this time all the phases of auricular and ventricular diastole and systole are passed through, the heart rotates and tilts, the great vessels in close relation with its cavities pulsate, coil, and uncoil. It is not wonderful if the details of so complex and so sudden a series of movements are scarcely to be made out by the eye alone. The paces of the horse have been watched more widely and more often than the motions of the heart. But simple eye-observation has always been as inadequate in one case as in the other. Veterinary authorities have differed as widely over the nature and sequence of the horse's paces, as physiologists over the visible motions of the heart. Two indirect methods of observation have lately been applied to determine the truth in the case of the horse. One is Marey's—the graphic method. In this, by special mechanism, the animal is made to leave on a recording surface permanent traces of his movements, or rather of the movements of some of his members. These traces are then deciphered and interpreted. From the tracing, the movements can be inferred and synthetised.

The other method, most recently and most completely carried out by Muybridge, might be called the static method. In it, a series of pictures of the animal in successive instantaneous positions are obtained. We get an idea of how he would look if he were suddenly fixed in any one of his positions. From this discontinuous series of phases, we can readily infer by what movements the one phase passed into the other.

These two methods are thus complementary. The graphic method gives us the positions of selected parts of the animal at each instant: the static method gives us the positions of each part at selected instants.

Analogous methods may be applied to analyse the movements of the heart. Marey's cardiograph (which we shall consider in our next lecture) gives us continuous tracings, from which the motions of selected points of the heart have been clearly and certainly inferred. Thus the motions of the apex, or rather the rhythm and character of its impulse, are completely determinable in any given case. But, applied to the heart as a whole, the cardiographic method is scarcely adequate. It can at best only give us a clue to the displacements of a limited number of its points. In a simple mechanism like that of a limb, which has only one or two degrees of freedom, such partial data would be enough to determine the motions of the whole. But the heart has many parts, it has no rigid attachments, its points are not simply connected, it has interior as well as exterior movements. To settle even a few of the questions that arise, we should need a multitude of cardiograms, and the greatest of skill to interpret them.

The static method seems more promising. If we could obtain fixed pictures of the instantaneous figure of the heart in various phases of its cycle, we should be able, if the phases were well chosen, to study and educe at leisure the movements by which the cyclical changes of form are effected. This method has been applied with exquisite skill, and, in view of the great difficulties in the way, with much success, by Ludwig and Hesse at Leipzig ("Beiträge zur Mechanik der Herzbewegung", *Arch. für Anat. und Phys.*, 1880). The results have not yet found their way into your physiological text-books; but, as I have had the opportunity of making myself personally familiar with them, and as I think they will throw light on several of the questions of physical diagnosis which it is our business to study together, I do not hesitate to ask your attention to them to-day.

We have, then, to find a means of fixing the form of the heart in two or more typical configurations of its cycle. In a first attempt, at least, we should naturally choose for fixation the states of complete systole and complete diastole, under conditions agreeing with the normal. The form of the heart as you see it in the *post mortem* room, and as it is seen in this specimen, cannot be regarded as representing any one of the normal configurations. The muscle may be relaxed as here, but it is not distended as in natural diastole; or it may be rigid, and yet not emptied of its contents, as in systole. The internal resistances and supports afforded by the liquid blood are altogether diverse from those due to cavities that are empty, or contain only clots. The exact study of form in dead hearts is thus almost as hard as the exact study of motion in living ones. To be true to nature, we must find means to fix the form of natural diastole in a heart whose muscle still retains all its vital properties. We must also have the power of producing in this heart a contraction whose process shall imitate exactly the natural contraction of systole, and of fixing that.

What are the factors which determine the dilatation-form of the heart in diastole? Its muscle is relaxed; there is, therefore, no contractile element. The chief factors must be of a passive kind; they are (1) the elasticity of the muscular walls; (2) the pressures to which these walls are subjected. Suppose, then, we have here suspended a heart still possessing all its vital properties unchanged: let us gradually fill it with blood until the pressure is equal to the mean blood-pressure during life. It will dilate, and assume a definite form. Consideration will show you that this form can only differ to a slight and negligible degree, if at all, from that of true diastole. When the heart is in its place in the thorax, its outer surface is exposed to slight and variable pressures depending on respiration, etc. These may affect somewhat its changes of position; they may guide its movement as a whole; but, in the case of a heart lying loose in the smooth pericardial sac, the factors which control and determine its typical diastolic form must, after all, be these main ones of intrinsic elasticity on the one hand, and internal blood-pressure on the other.

Here is a plaster-cast of a dog's heart, or rather of its ventricular part, in what I have called typical diastole. You will ask how the mechanical difficulties in the way of modelling a cast of the still-living heart have been overcome. The heart must be still living; the elastic properties of a muscle in which death or rigor has set in are altogether different from those of a live muscle. And the process we use must be such as not in any way to destroy the heart; for we want to obtain a cast of it in contraction. The difficulties, then, are not slight; but they have been overcome at Leipzig in the following way.

A dog is rapidly bled from the carotids; the chest is opened; the auricular vessels are ligatured; straight graduated glass tubes are tied into the pulmonary artery and the aorta; and the heart is then removed from the body, and suspended by the tubes, which are kept vertical. The blood of the animal has been meanwhile freed from fibrin, and it is now poured into the vertical tubes so as to dilate the ventricles, until the pressure is equal to the mean blood-pressure during life (150 millimètres). The auricles at once begin to pulsate rhythmically, and continue to do so for nearly an hour. The ventricles remain soft, pliable, and irritable all this time. The heart-muscle, in contact with nutrient blood and prevented from cooling or drying, is in fact alive. Certainly in the first few minutes of this hour no change in its vital properties can have occurred. Little pins are quickly inserted into the muscle at certain spots to serve as reference marks; and then a thin layer of very quickly setting plaster is lightly applied. A minute or two is enough to let this harden sufficiently, and then it is broken off by a skilled hand, who is able to set together the pieces again, and so to form a mould from which casts may be taken. The weight of the plaster does not alter the form of the ventricles, for the level of the blood in the vertical tubes remains unchanged during the manipulations. All this of course requires great skill and quickness; many unsuccessful experiments were made before these were attained; but I am describing the course of an experiment supposed in every way successful. It was in this way, then, that this cast of the diastolic heart was made. Here is another cast of the same heart in typical systole. To obtain this, the heart, still living and uninjured (for the auricles still pulsate), is emptied of blood, and then plunged quickly into a hot saturated solution of bichromate of potassium at 50° Cent. (122° Fahr.). One rapid and final contraction of the ventricular muscles takes place; this is permanent, for the muscle passes into *rigor caloris* (Foster, vol. i, p. 2; Hermann, vol. i, pp. 99, 147); and in this state it is fixed, and its textures hardened, without shrinkage or alteration by the bichromate. We can let it stay in the solution till the hardening is complete, and take a cast of its outward form at leisure.

We have, then, before us the form of the heart in diastole, and the form of the same heart in systole; corresponding points being marked

by the little reference-pins. Let us first consider the external differences. You see that in diastole—(1) The shape is roughly hemispheroidal, the pointing of the apex being scarcely marked: in the plane of the auriculo-ventricular openings (the *ostia venosa*) the greatest dimension is from right to left; the antero-posterior is less; from this plane to the apex is the shortest of all. (2) The conus arteriosus arches over the plane of the ostia. (3) The posterior surface, bisected by the longitudinal interventricular furrow, is much flatter than the anterior. In systole, on the other hand—(1) The shape is conical, the apex distinct; the diameters drawn in the plane of the base are notably shortened, but in equal degree; the length of the cone from base to apex remains unaltered; it is now greater than either diameter of the base. (2) The conus has sunk down towards the plane of the base. (3) The difference in curvature between the anterior and posterior surfaces has disappeared. The three diagrams (Figs. 1, 2, 3) bring out these points; they represent vertical projections of a pair of casts superimposed for the sake of exact comparison. They are taken from Ludwig's *Arbeiten*, 1880.

1.—Posterior surface.

The fact that the length of the ventricle remains unchanged, is very striking. It is true, in strictness, of the left ventricle only, for the apex is part of this ventricle. We shall see later what slight modifications apply to the right ventricle. Again, the reference-pins inserted along a meridian of the left side in diastole, maintain their vertical distances

2.—Left lateral surface.

almost unaltered in systole. The length of the ventricle is, in fact, constant; not merely as a whole, but also in its parts. This settles, while it perhaps accounts for, a dispute that has lasted for centuries. Galen and Vesalius taught that the heart lengthened in systole; Harvey that it shortened. In the seventeenth century, the famous medical schools of Montpellier and Paris fought fiercely, but chiefly with dialectic weapons, on Galen's side and on Harvey's respectively. At length, Bassuel asked triumphantly, "How, if the ventricle lengthens, can the auricular valves, anchored to its walls by the tendinous cords, ever succeed in closing?" No answer was forthcoming from the south,

and so victory remained with Paris. Since then, the text-books tell you that the heart shortens when it contracts. It is not hard to see how Harvey, and other observers since, have got this impression from

3.—Anterior surface.

watching the still acting heart *in situ*. Kürschner's authority is often cited: let us see what he says (Wagner's *Handwörterbuch*; *Herzthätigkeit*). "By means of the ribs, the increase in the heart's length during diastole may readily be demonstrated. In rabbits, the apex is deeper by a whole interspace in diastole than in systole. The question may also be easily settled by inserting a pin opposite the point occupied by the apex in diastole: it will then be observed that at every contraction a free space is left between the apex and the pin." A moment's reflection will show you that this does not settle the question. To prove that the length alters, we must not merely prove that the apex moves: we must show that it approaches the base. I can move about freely the tip of this oblique India-rubber cone which rests on the table, but I do not change the length of the cone; the tip moves parallel to the base. So the apex of the heart may twist and tilt, while it maintains unchanged its distance from the base. There is another source of fallacy. The conus arteriosus lies anteriorly, and catches the eye first. We have seen (Fig. 3) that this sinks down towards the base with each contraction. The distance between the prominent parts of the conus and the apex does, therefore, diminish in systole; but this is not the length of the heart in any useful sense of the term. I have said enough, perhaps, to fix in your minds, as a fundamental proposition, that the length of the ventricle does not alter as the heart contracts. We shall see again and again what valuable corollaries follow from this.

The next point is the very considerable difference between the area of the base in diastole and that in systole. (Fig. 4.) This is so marked,

Fig. 4.—Projections of the base in systole and diastole of the same heart. The disposition of the ostia in systole is indicated.

and at first sight so astonishing, that careful measurements have been made so as to check and control the experiments in various ways. But from all of them it still comes out that, in systole, the area of the base (in the plane of the ostia) is little more than half what it is in diastole. Moreover, this marked diminution of area falls almost entirely on the ostia venosa; the arterial orifices are scarcely, if at all, affected. Let me fix this in your memory, as I tried to fix the former proposition regarding the length of the heart—by citing an apparent contradiction. In Foster (i, 4), you read: "According to Kürschner, the circumference of the base of the ventricle is absolutely increased during the systole; a tape placed round the base becomes tense at the commencement of systole, while the cavity is still full of blood." A few lines before, we are told that, during the systole, the right-to-left and antero-posterior diameters of the base both shorten. How can the circumference be increased, if the diameters both diminish? you may very naturally ask. If you turn to Kürschner, you will find that he almost reluctantly admits the tape experiment, first performed by some one else, I suppose, and proceeds to explain it away. He insists that the tightening is only momentary, and at the very beginning of systole—that is, before the contraction of the heart, as a whole, is completely set up. An instant afterwards the tape is loose. He gives an untenable explanation of this momentary effect, as I think; and you may easily find another more satisfying. Looking at these casts, you will see that it is impossible to put a string round the base of the ventricles, without including the conus arteriosus. Now, you know that, as systole begins, the conus descends: it may very well carry the encircling string with it on the anterior aspect, and the string which did go straight round in a circle is now pulled down into an oblique ellipse, as it were. It will thus be

Fig. 5.—Sections through the upper third of the ventricles.

tightened for a moment, until the diminution of the girth from the advancing contraction more than balances the increase due to obliquity. There are other factors which come in; but all I wish to do here is to clear up what I know to have been a puzzle to some. The string experiment refers to the very beginning of systole, and does not strictly bear on the question of the area of the base (seeing the string includes the conus at least). The fact on which I am insisting is, that in complete systole the area of the base itself is diminished by nearly a half.

Putting now the two data together, we see that the inevitable inference is, that the systolic form is derived from the diastolic by diminution of the heart's transverse section everywhere, without change of its length.

Let us see how this is borne out by a study of the internal cavities of the ventricle. You will see at once that our method for getting a cast of the outward form will not do for the form of the cavities. We

might pour plaster into the dilated cavities, but we could not set free the mould without destroying the heart. Another method was chosen. Two young dogs were taken of the same litter, make, and size. They were similarly fed, and grew together. It might be presumed that their hearts would be as similar as they were themselves. Several such pairs of dogs were taken, and preliminary experiments showed that the presumption was justified. The pairs of hearts were alike in development, size, and weight. One heart of a pair was dilated as before with its own blood, and then lowered into cool bichromate solution; it was thus slowly hardened in its diastolic form. The other was similarly brought to contract by hot bichromate solution, and hardened in systole. When the hardening was complete, casts of the cavities could be made in plaster, or fusible metal; or corresponding transverse sections could be cut, to show the relations of the walls to the lumen.

Here (Fig. 5) are two pairs of such cross-sections, made in the middle and upper thirds of the length respectively. They will illustrate the points brought out by examination of the casts of the cavities. The cavity of the left ventricle in diastole is nearly cylindrical. It is rounded off below, and above divides into two necks set at an angle. These, of course, correspond with the auricular and aortic openings; they are separated by the great anterior flap of the mitral valve. The cast shows three shallow grooves, corresponding to the two great papillary muscles, springing from the outer wall of the ventricle, and to a fleshy prominence at the anterior angle between the wall and septum. The septum is singularly smooth, as it passes up to the root of the aorta. You will see in this hardened specimen, and in the diagrams, that the three longitudinal prominences do not extend higher than the middle third of the cavity. So the lumen, above their tips and just below the valves, is free from their encroachment. The cross section here is concave all round. In systole, the cavity has wonderfully altered. The cast shows the suprapapillary space as a rounded

Fig. 5.—Sections through the middle third of the ventricles.

solid mass. From this springs a short vertical axis, and round it twist four sharp flanges or wings, like the threads of a right-handed screw, ending in a ragged point. The deep hollows between the flanges correspond to the three longitudinal prominences and the septum. The septum has bulged into the cavities to make up for its anterior-posterior shrinkage. The cross-section of the lumen below appears then as a four-rayed star; near the base, it becomes smooth and rounded.

Of the right ventricle I will not say much at present. The casts and diagrams show that in diastole its cavity has the shape of the bowl of a short thick spoon, the long axis being oblique to that of the left ventricle. The right ventricle, in fact, twists round the left somewhat, so that the sections in the diagrams are rather oblique for the right ventricle, though they are transverse for the left. In systole, the lumen becomes narrowed to a fine slit; it forms a larger arc of a smaller

circle than in systole. In this ventricle, as in the other, there is something of a free suprapapillary space under the valves.

We shall presently return to the question of what the suprapapillary spaces and the papillary prominences really mean. At present I ask you once more to look at the cross-sections (Fig. 5). A great many similar sections have been made, and their areas (that is, the area of surface occupied by muscle, the shaded area) have been carefully measured with the planimeter. It is found that the muscle-area is the same in systole as in diastole. Any change in the contents of the outer circumference is entirely at the expense of the lumina. Now this confirms, in a striking way, what we said above, that the systole is effected by diminution of the cross-section everywhere without change of the length. When a muscle contracts, you know that its volume is perceptibly unchanged. If its length do not alter, its average cross-section must also be constant. In a hollow muscle like the heart, if under contraction the length on the whole do not alter, the average cross-section of muscle must also be constant. The process of contraction must, therefore, be such that the elements, originally in each transverse plane, must remain in that plane throughout, neither approaching the base nor receding from it on the whole. The sole effect of the contraction will thus be to diminish the lumen. The walls thicken, but they thicken only at the expense of the cavities. There cannot be an increase in the circumference when contraction begins, as the tape experiment might suggest.

Thus all the facts we have hitherto noted are in agreement as to the mode in which the heart changes its form. Can we get any idea as to the "why" of all this? By what mechanism is this peculiar mode of contraction brought about? It would be natural to expect, with Harvey, that the heart in contracting should shorten all its dimensions—*rationi enim consentaneum est, cor eo quo movetur tempore undique constringi, et secundum parietes incrassare secundum ventriculos coarctari*. As a fact, it shortens all its dimensions but one. By what process of compensation is the length kept constant? The arrangement of the muscular fibres of the heart is extremely complex. I need only refer you to your Quain, or to this book of Pettigrew's (*Physiology of the Circulation*), to remind you of the layer upon layer, the spirals and the vortices, into which the ventricular walls have been anatomised. But if you examine a cross section like this, you will see that in effect we can represent the various layers by dividing the wall into three zones (fig. 6). Inside, you have a zone of longitudinal fibres

Fig. 6.—Cross section at middle of ventricle in systole.

composing papillary muscles and columns. Outside, you have a thin surface layer of spiral fibres running obliquely back to the left. Between, there is a broader band of fibres, taking on the whole a circular course round each ventricle, and enclosing the longitudinal masses as in a sphincter.

Imagine this circular band to contract. It will drive the included masses towards the centre; their surface will be too great for their contents, and so they will be thrown into wrinkles and folds. They serve thus a kind of secondary purpose: they are utilised by the sphincter ring to obliterate the cavity by crowding it out, as it were. Their own contraction serves little to fill up the cavity. If they increase in thickness, they shorten in length, and so take up no more space than before; their volume remains constant. But you must not think that these central longitudinal masses have nothing active to do. If the heart were made up of the circular fibres alone, it is plain that, as they contracted so that the circumference of each ring shortened,

its thickness must at the same time increase. The result would be, that the heart would lengthen on the whole. If the heart were mainly made of longitudinal fibres, when they contracted they would thicken and shorten simultaneously, and the heart would shorten on the whole. In the actual heart both sets are present, and you see that they conspire as to diminishing the cavity; but they conflict as to changing the length of the heart. The result is, on the one hand, increased efficiency in expelling the blood; on the other, a balance between the opposing forces which keeps the length of the heart unchanged. This latter balance is not an idle refinement. The mutual checking and restraining of each other's play calls out the elasticity as well as the contractility of the muscles. In systole, they are in strained equilibrium. The result is that, as soon as ever the effort of contraction ceases, the elastic action will set up a spring-like reaction, and the ventricles begin to dilate actively. Now, Marey and Fick, Goltz and Gaule (*Arch. für Phys.* xviii), have shown, beyond question, that, at the beginning of diastole, there is such an active element in the dilatation. They found that the negative or suctional pressure in the left ventricle might be as much as 23 millimetres of mercury; and that independently of the respiration.

This common injection-syringe represents the heart's action very fairly. It is a thick-walled India-rubber globe, with two tubes furnished with valves. The inlet-tube conveys water, under slight pressure, from the vessel above. My encircling fingers represent the circular fibres of the ventricle-wall. I contract them, and drive the India-rubber, against its elastic resistance, into the cavity of the globe, forcing water through the outlet-valve. I relax my fingers, and the India-rubber actively dilates by its own springiness, and so the water is partly sucked in, and partly flows in by its own pressure. This active suction of the left ventricle partly explains why the power of the right may very well be much less than that of the left. The force that overcomes the resistance of the lung capillaries is not entirely a *vis à tergo*. The left ventricle does some of the work of the right. To produce this elastic straining in its walls, of course requires a greater muscular effort in the contraction; but the energy is not lost or wasted. When diastole begins, it is restored—for the benefit of the pulmonary circulation. There are several other factors which go to help the right ventricle to diminish its cavity, without calling for active contraction from its walls; some of them you will be able to think out for yourselves. There is a heart in the museum, whose right ventricle is adherent to the pericardium, and forms with it a firm, gristly, partly calcified case; it looks quite incapable of contracting at all. Yet the left ventricle did the work of both so well, that the patient died without any serious signs of pulmonary or venous congestion.

Let us, lastly, see how we can apply our principles to explain the action of the auriculo-ventricular valves. We shall, for brevity, speak only of the mitral. Here is a perfectly flaccid and relaxed sheep's heart. I have tied a glass tube into the aorta, through which I now fill the ventricle with water. You see the valve-segments floating up; they come together, but they do not close the ostium quite perfectly; a jet of water regurgitates; the valves are so far incompetent. In a similar heart, which is contracted and stiff in *rigor mortis*, the valves are quite competent: I can put on a very considerable pressure, and yet they do not let a drop escape. This is not a new experiment. The difference between the hearts has been often noticed, and it has been attributed in some obscure manner to the action of the papillary muscles. But I can make the valves of the flaccid heart competent in a very simple way. I simply encircle the base with an ordinary elastic band, so that its area is somewhat contracted. The valves are now as good as in the other heart; yet the papillary muscles are in the same state as before. The fact is, we were in the first instance expecting the valves to close orifices that were too big and too round for them. In normal circumstances nature does for them what I did, when I put the elastic band round the base. As systole begins, the muscles surrounding the ostia contract; and presently, instead of the round gaping orifices of diastole, the valves have to close oval and compressed ones. When systole is complete, the area of the orifices is not much more than half of what it is in diastole. Thus the work of closing is not done by the valves alone; it is largely shared in by the muscles of the base. I believe that in two cases out of three the valves alone would be quite incompetent to close the openings as they are in diastole, and that in perfectly healthy hearts. The valves of the heart in rigor were competent, because, as you know, rigor is practically a contraction. The base-muscles, as they stiffened, squeezed the ostia into something near their systolic shape and size; the valves were able to do the rest. Imagine now a heart acting within the body; its valves and all being perfectly sound and normal. The base-muscles do their share of the work of closure, the valves promptly complete it. Let now the muscular power of the heart, the base-part of it, at least, become impaired,

or the contraction become less energetic. Instead of diminishing the area of the base by a half, it languidly contracts it—say by a quarter. It will not be wonderful, then, if, from this cause alone, the valves find themselves inadequate. They are asked to close bigger orifices than usual; but not because the orifices have dilated. Those pliable fibrous rings, to which the valves are attached, are pretty tough structures; no ordinary force will stretch them perceptibly, unless their structure become disorganised. The valves are insufficient, not because the base is dilated in this sense, but because it is insufficiently contracted. Restore the power of the enfeebled muscles, and the incompetence disappears, and with it the regurgitation.

When an animal is rapidly bled till it is feeble, a murmur indicating regurgitation from the ventricle is heard with the heart-sounds. You may inject a proper saline solution to make up the normal quantity of circulating fluid; but still the regurgitation occurs. As the animal makes blood again, so that its muscles are again properly nourished, the murmur disappears. In later lectures, and at the bedside, we shall often have cause to remember this factor in the causation of transient regurgitant murmurs. It is because it is a factor not commonly recognised, but yet, as I believe, an essential one, that I have led you gradually, and perhaps lengthily, to see its exact meaning. The hypothesis of dilatation is, we shall see, somewhat strained to account for what we can easily and naturally explain, without assuming any such grave structural change. The picture of the base of the heart in systole, present to many minds, is too much like that given in Quain's *Anatomy* (vol. ii, p. 250), and in other text-books (Fig. 7). The true

Fig. 7.—Base of the ventricles according to Quain: two-thirds natural size. picture is this of Professor Ludwig's (Fig. 3). A glance at these will show the difference better than a long description.

Fig. 8.—Base of the ventricles in systole according to Ludwig: two-thirds natural size.

But, now you will, perhaps, say that I have been proving too much. If the ostia venosa are so gripped and contracted in systole, how do the ostia arteriosa remain patent and uncompressed? Nature has provided for this, and the device she uses is as simple as it is perfect. Consider the left ventricle. We saw that the papillary muscles do not

extend higher than the middle third of the cavity. Even in extreme systole, then, when the projecting muscular masses are all in contact below, there still remains a free space under the valves—the suprapapillary space. You will remember, also, what I show you in this calf's heart (hardened in diastole), that, between the orifice of the aorta and the orifice of the auricle, is placed, as a partition, the great anterior flap of the mitral valve. This flap is, however, not merely a partition, it is continued upwards beyond the fibrous valve-ring, to form the posterior membranous portion of the root of the aorta. The suprapapillary space is thus shut off towards the auricle, and left open towards the aorta by this pendent continuation of the aortic wall, the flap of the mitral. The issuing blood-stream strikes against the ventricular side of the partition, and, by one and the same action, shuts off the auricular opening and swells out the root of the aorta, making it continuous with the suprapapillary space, and leaving the outgoing path unobstructed.

There is a further arrangement for securing that this greater flap shall always be in the right position. The blood must always so strike it that it is thrust the right way, viz., towards the auricle. The two large papillary muscles to which its borders are attached are seated on the outer wall of the ventricle—that is, on the side opposite to the aorta (Fig. 8). During diastole, then, the retreating wall, with its muscles, carry the edges of the flap ever away from the aortic opening. In full diastole, the flap and its cords are stretched aslant across the cavity. Thus, when contraction begins, the flap already lies at such a slope athwart the ostium venosum, that the extruded blood must needs strike against its under or ventricular side.

I say the valve is on the stretch in diastole. This is not what the books usually say. But it is what is always seen when a heart is hardened in diastole; it is plain in this specimen I show you. The flap does not hang loosely down; it is stretched taut from basal ring to muscle tip. If the valve is to be bent upwards into its place, it is plain that, in contraction, these two points of attachment must approach each other. If we think of the papillaries as shortening and shrinking into the muscle-wall during systole, it is hard to see how the valve can act. M. Bassuel might propound his victorious question to us in another form. Now, careful measurements have been made to settle this point; and they show that, while the tips of the papillary muscles do not approach the plane of the ostia at all, they do approach, and that in a very marked degree, to the aortic orifice. What is true of the integral parts of the heart-wall, is also true of the semi-detached papillaries. Their various cross sections move during contraction in planes parallel to the base. The approach of the muscle-tip to the base of attachment of the valve, due to the approach of the muscle-wall alone, is so considerable that the valve might be forced too far through into the ventricle. This is compensated for by a shortening of the papillary muscle itself, which is just enough to bring back its various parts to the levels in which they lay in diastole.

The suprapapillary space may serve yet another purpose. Even at the end of systole, it still remains full of blood. In this residual blood, bathing the under sides of the sigmoid valves, their flaps can have free play. They can unfold themselves, and become sacculated without hindrance, at the instant when systole ceases, and the blood in the aorta seeks to rush back into the ventricle.

To those who read German, I cannot give better advice than that they should study carefully, with the help of these diagrams and models, the original from which this lecture is mainly taken—namely, the paper of Ludwig and Hesse, already cited above. The account they give of the heart's action rests on facts and measurements. It is not simply one more "view", which may be credited or discredited at pleasure.

A TRICYCLE RIDE.—At the Hammersmith Police-court, a Fellow of the Royal College of Surgeons appeared in answer to a summons for furiously riding a tricycle. The constable stated that he saw the defendant riding furiously. The defendant denied that he was riding fast, and said he was fatigued, as he had been a run of sixty miles. The magistrate, however, was of opinion that he was underrating his powers, and fined him 40s. and 2s. costs. The defendant complained of having been taken to the station, followed by a number of roughs. To be riding a tricycle so rapidly after a journey of sixty miles argues well both for the powers of endurance of the rider and the excellence of the machine ridden. On the same day, several other tricycle-riders were fined for furious riding. It is possible that, in the pleasurable muscular excitement and rapid movement, accurate sense of speed is lost. When, however, a mounted constable has to gallop his horse to overtake a tricycle, it is obvious that the tricycle in question is going at a rate exceeding six miles an hour. This is not safe in the streets of a large town.