predisposing him to the disease, or possibly some peculiarity of season favouring its development." We smile with the lofty tolerance of more exact information that such inferences should pass muster in this Society only twenty years ago. Cases, however, I venture to think, come within the experience of physicians and surgeons that would justify Dr. Greenhow's shrewd remark, cases in which incidents of a septic kind—foreexample, phlebitis and secondary embolism of organs—occur after operations for appendicitis, varicocele, varicose veins, thyroid resections, not to mention parturition and operations about the nose and ear passages. In some of these cases which have come under my observation the accidents could be accounted for by no imperfection in the operation nor in the sanitary surroundings of the patient. The wounds have soundly healed, yet the patient has perhaps an embolic pneumonia or a cardiac embolism. These cases are not very frequent, but one meets with them, and they are more common, if I may judge from my own experience and inquiry, in private than in hospital practice. They are, too, the more striking when met with, since they are relatively more frequent in slight than in very severe operations. My impression is that they are accounted for by an unpreparedness of the patient for surgical injury. In some acute and urgent cases, for example, of strangulated hernia, suppurative appendicitis, and the like, the surgeon has no option but to take the risks and operate immediately, but in chronic cases a few days' preparation, or, if necessary, a few weeks' regulation of the patient's habits and secretions would serve to purify him of those effete products of impaired, imperfect, or exuberant tissue change, and inadequate excretions which cause a morbid reaction to local injury. The methods of procedure may be aseptic, but the patient in many instances can scarcely be said to be so.

ELECTRICITY, MYXŒDEMA, AND ORGANOTHERAPY.

In the lifetime of our Society the use of electricity for diagnostic purposes has made great strides, the employment of the electro-magnet for detecting and removing foreign bodies, especially from the eye, of electric light for exploring hollow cavities, and lastly of radiography have been closely watched and studied here with excellent results for diagnosis, but little, so far, of a direct kind for treatment. One of the most valuable contributions to the science of medicine to which this Society has largely contributed, is the description and thorough elucidation of the disease now known as myxœdema, having been first so named by our esteemed member and late treasurer, Dr. Ord. Sir William Gull was the first to describe the disease under another name, and it was afterwards thoroughly investigated by Dr. Ord, and reported upon in a special volume of our *Transactions* by a Committee presided over by him. Apart from the intrinsic value of investigations which have thoroughly established the nature and treatment of this important disease, the study of myxœdema and its treatment by thyroid extract gave an impetus to, if it did not actually originate, the pathological and physiological lines of thought and experimental inquiry, which have shown the influence of secretion products absorbed from ductless glands, and from some glands which are not ductless (ovary, testicle) upon mental and physical health and upon formative symmetry. The sexual glands, the adrenals, bone marrow, spleen, and pituitary body have in this comparatively new light been reinvestigated, and the practical physician is already reaping the fruits therefrom in a greater insight into many previously inexplicable groups of symptoms. The most recent and suggestive outcome in this category is that of the relationship between disease of the pituitary body and that want of growth-control which characterises acromegalous disease, the first case of which recognised in this country was brought before this Society by Mr. Godlee.

Another idea that has arisen from the closer association of laboratory and clinical research is the analogy between the affinity of stains for certain tissues and the selective tissue-affinity of certain poisons. The affinity, for instance, of the toxins of the tetanus and diphtheria bacillus for nerve tissues, and the very analogous selection of the nervous system by the influenza poison are rendered tangibly intelligible to us from our laboratory experi-

ence of the affinity of different stains for muscle, nerve, fat, cell nuclei respectively.

I must not further trench upon your patience by reflections founded upon the work done by this Society and a glance at some of the questions which it has helped to solve. The retrospect is laden with encouragement and suggestions for work yet to do.

PRESIDENTIAL ADDRESS

ON A

CLINICAL VIEW OF SOME OF THE MECHANISMS OF THE HEART AND ITS VALVES.

Delivered before the Chelsea Clinical Society on October 16th,

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Good work achieved is a pleasant retrospect and the best incentive to further efforts; and in that direction this Society may be congratulated upon having earned satisfaction and encouragement. But, in view of the work which lies before us, I must not dwell at any length upon the past, and I shall not attempt more than to thank you for the honour conferred upon me, and to express a regret, shared by all of us, that our rules should have imposed upon us a change in the occupancy of this chair. The early fulfilment of the aims which originally brought our members together has been largely due to our retiring President, Mr. Foster Palmer, ably seconded by Dr. Austin Cooper, our senior Honorary Secretary. His successful management of our affairs has done more for us even than our good fortune, and, though we lose him as a President, the prospect of his continued service in another capacity is for the Society a pledge of continued prosperity.

THE CLINICAL METHOD.

Some of our difficulties in interpreting and discussing the physical signs and the mechanism of valvular affections might perhaps be lessened if we could take a clearer view of the normal mechanisms of the heart. We need more anatomy and physiology. But, in spite of our anxiety to be guided by physiology, we cannot quicken its measured step to suit our clinical urgencies. Upon some points which are essential to us physiologists differ, and physiology reserves its verdict. Perhaps we might endeavour to help ourselves; and something might perhaps be achieved by applying to the study of the normal heart our own clinical method. Without invidiousness the latter might be described as one less exact and less exacting than the methods of pure science, as one confessedly opportunist; weak it may be in the lack of strict experimentation, yet strong in an endless supply of the unerring experiments of disease. Conceived upon these lines, the following fragmentary sketch of the heart's action has been based upon anatomical observations illustrated by specimens, upon postulates which may obtain your assent, and, lastly, upon inferences which are clinical rather than physiological, and which claim the privilege which belongs to the clinical method—that of being put forward provisionally and without prejudice. A few instances drawn from the clinical field may in conclusion serve to illustrate the purpose of this address.

I.—ANATOMICAL INTRODUCTION.

This must be limited to a few essential points. As regards the intricate directions of the muscular fibres, we need only note that, whilst the predominating circular and spiral fibres of the ventricular wall are mainly constrictive, the septum is chiefly made up of fibres which in contracting increase its thickness without lessening its vertical length or that of the ventricle. The top of the septum affords a partial attachment to the foot of the pulmonary artery and to that of the aorta, but it presents underneath the latter a strong declivity, and the interval between it and the vessel—the so-called "undefended space"—is filled by a thin tough membrane which is continuous with the membranous extension of the anterior mitral flap. The papillary muscles are mainly vertical, as well as most of the columns carnea.

When freed by boiling from their fibrous superstructures, the ventricles appear as cones, the right with two basic orifices, the left completely open without any muscular roof whatever, or any trace of mitral and aortic orifices. The mouth of the left ventricle is clearly not an ordinary sphincter; for, were it a simple sphincter, it would periodically constrict the aortic orifice, which should always be free, together with the mitral orifice which alternately contracts and relaxes. We therefore find two sets

of fibres: the constrictive fibres from the ventricular wall which work the mitral orifice, and those passing up to the aorta from the septum, which are not constrictive.

Looking through the cavity of a suitably prepared left ventricle, a thin partition is seen between the small aortic and the large mitral orifice. This membrane, which is continuous with the aortic wall, is the great anterior mitral flap stretched antero-posteriorly between the front and the back of the ventricle by means of two powerful insertions, to which I shall refer again. Thus the mitral and aortic orifices are not circular, but D-shaped; and in each of them it is only the loop of the D which is formed by muscle.

Two of the aortic sinuses with their semilunar membranes are continued below into this membrane and its lateral prolongations, and they face respectively into the left auricle and into the right auricle. The third or "septal" sinus, which is firmly implanted at the top of the septum (Savory), bulges downwards into the right ventricle where it is covered by the muscle of the "fleshy pons" which separates the tricuspid from the pulmonary orifice. This circumstance bears upon the question of the wide conduction of the aortic second sound and of the singularly restricted conduction of the pulmonary second sound—points to which attention has been drawn elsewhere.¹ The pulmonary artery rises above and in front of the septum at its highest point, the posterior pulmonary sinus resting upon it just in front of the aortic "septal" sinus. The two anterior sinuses are also controlled by muscular fibres, the vertical fibres of the conus arteriosus.

THE CURTAIN VALVES AND THEIR "FIBROUS RING."

The auricles which rise in a dome above the two ventricles are wedged apart by the aorta in front, and are separated behind by a thin septum. Whilst the ventricles have no muscular vault, the auricles have no muscular apex and practically no floor, except when the valve flaps rise as a diaphragm at each systole. The valves themselves and particularly the mitral may be regarded as wide inverted cones conducting the blood into the depth of the ventricles. A fusion or adhesion of their fringes would provide the auricle with a floor; this is the tendency of mitral stenosis. The typical smoothness of their internal or auricular surface which is made rough by valvulitis, and the exclusive attachment of the chordæ to their parietal or ventricular surface are points worth remembering. Whilst the fine marginal and sub-marginal chordæ control the delicate fringes, a different but essential purpose is served by the two powerful chordæ which ascend to be inserted into the upper fibrous part of the great mitral flap.

Fig. 1.—Dissection from the bullock's heart showing the relations of the bones of the heart to the aorta, to the mitral and tricuspid valves, and to their fibrous loops. P.O. and A.O., the posterior and the anterior bone; M.M., the insertions of the anterior mitral flap jointly with those of the fibrous loop; T, the tricuspid valve and fibrous loop attached to the posterior bone only; P, the pulmonary artery; S.S., the "septal" sigmoid flap of the aorta and of the pulmonary artery; M, the "mitral" or "left auricular" sigmoid flap; A.A., the "right auricular" sigmoid flap of the aorta.

The so-called fibrous ring at the junction between the left auricle and ventricle, from which the posterior flap is suspended, is thin and pliable even in the ox. It is not a ring but only a horse-shoe-shaped loop, the ends of which, together with some of the muscular fibres of the orifice, have the same attachments front and back as the two insertions of the anterior mitral flap, the latter completing the circle.

The Fibrocartilages of the Heart.—These relations are well seen in this preparation of the valves and of the basal fibrous apparatus removed from a bullock's heart. Dissection has brought into view two bony plates, a large posterior plate, $\frac{1}{2}$ inch long, corresponding to the central fibrocartilage of Sibson, and a much smaller triangular one, $\frac{1}{4}$ inch long, no less important. They each respectively receive one of the mitral insertions, and they stretch horizontally backwards on either side of the aorta along the base of those two sinuses which are not attached to the septum. In man they are rarely ossified, seldom cartilaginous, usually densely fibrous. In this specimen of a female heart they feel like fibrocartilage. The large posterior bony plate also affords attachment to both

ends of the tricuspid fibrous loop. This demonstrates the intimate relation (Sibson) which exists between the fibrous structures of the aorta and those of the mitral and of the tricuspid valves. The connection of the aorta with the adjacent pulmonary valve is established by the septum to which they are both attached.

THE ATTACHMENTS OF THE AORTA AND ITS MOBILITY.

At first sight the most striking peculiarity of the aortic attachment is its looseness. Except over the septum the aortic wall passes into a membrane much thinner than itself, the mitral portion of which ends free in the ventricle. Mobility is the most obvious purpose; another object may be the suppression of any damaging recoil. Closer investigation shows, however, that special provision is made for strength in addition to pliability. The stability of the vessel is insured by the muscular fibres directly inserted into it, and also by powerful muscles acting indirectly; on the one hand the papillary muscles through the two great chordæ steady the anterior portion, and at the sides extensive muscular masses from the two horns of the septum and from the posterior wall of the ventricle respectively are inserted at the edge and upper surfaces of the fibrocartilages.

The traction communicated by these fibres to the fibrous connections of the fibrocartilages, namely to the mitral fibrous loop, to the mitral membrane, and to the foot of the aorta, occurs in a direction downwards, backwards, and to the right, whilst they exert no lateral pressure upon the foot of the aorta. The combined effect of these fibrous and direct as well as indirect muscular attachments seems to be: (1) To protect the ventricular insertion of the aorta against any systolic overstrain; (2) to keep the aortic orifice widely expanded; (3) to shift the latter slightly backwards, downwards, and to the right, thus probably increasing the prominence of the fleshy pons, and helping to protect the pulmonary valve and the conus arteriosus from pressure; and (4) to change the obliquity of the aorta to a more vertical axis.

You may ask what evidence I can offer for the occurrence of these changes. In the many human hearts examined for them I have invariably found as part of the systolic attitude (a) an erect aorta tending to recede out of sight into a deepened aortic corner; (b) a puckering and a grooving of the infra-aortic surface of the septum; and (c) a disappearance, partial or complete, of the space and membrane which rather unfairly go by the name of "undefended," the systole itself tending to close the space, whilst their special function seems to be to facilitate the slight lateral movement of the aorta across the septum and along with it. According to this view the office of the muscles in question is not merely passive; they do more than steady—they adjust the vessel.

The Internal Fulcrum or Axis of Cardiac Contraction.—The external attachments of the heart by means of the great vessels are all basic, forming a system of hinges the complicated working of which is easier to understand than to describe, and sufficiently loose to permit considerable mobility of the heart as a whole, and particularly of the ventricles. But clear notions are also needed as to the internal fulcrum. When a long looped cardiac fibre contracts, which of its two ends is the travelling point and which the stationary, or do they both move? I believe that in spite of the slight oscillations described, the foot of the aorta, steadied by its septal and fibrous attachments, and the septum, are at the time of the systole the central axis towards which everything else moves. The central fibrocartilages being firmly connected with the aorta are an integral part of the same fulcrum, and the numerous ventricular and auricular fibres which are attached to them find in them their relatively fixed point, although I have said enough to indicate that the fibrocartilages are themselves perceptibly acted upon by the muscular contraction, and with them the orifices and the curtain valves, with which they are closely connected. In strictness, therefore, the centre of the septum might be regarded as the most stable point in the whole heart.

II.—THE MODE OF CONTRACTION AND THE SYSTOLIC CHANGES IN THE SHAPE AND SIZE OF THE HEART.

Passing over the familiar outward changes, three significant facts have been determined by Ludwig and Hesse² by means of the so-called Leipzig method, a lucid description of which you may read in Dr. Donald MacAlister's admirable lecture on the Form and Mechanism of the Heart.³ By careful measurements of the heart in full diastole and in complete systole, it was found that in systole (1) the length of the left ventricle is not decreased; (2) the circumference at the base is reduced almost one-half; (3) the mitral and tricuspid orifices are almost completely closed by the ventricular contraction.

Thus the systolic action is mainly constrictive; it approximates to the septum almost every point in the heart wall. It is not unlike the action of the hand-bellows, the moving flapper of which alternately approaches and recedes from the stationary flapper, whilst the excursion of any point is much less at the narrow end than at the broad one. Locally slight shortening of the longitudinal fibres must take place to adapt at every point the considerable convexity of the heart wall to the length of the septum; but there are three situations at which a perceptible shortening occurs, namely, the anterior wall of the conus arteriosus, the muscoli papillares, and lastly the auriculo-ventricular orifices, which are carried downwards and backwards, thereby aiding auricular aspiration and ventricular compression.

The Infra-aortic Space and the Suprapapillary Space.

Efficient systolic emptying of the ventricle is dependent upon a rapid increase in the intraventricular pressure and upon a free way into the aorta—in other words, upon early

and tight closure of the auricular valves and upon freedom from untimely contractions checking the outflow *a fronte*. The so-called "aortic channel" which lies along the septum must have afforded passage to the entire ventricular charge before it finally contracts, and we may fairly assume that the progressive obliteration of ventricular space must begin at the extreme left and end at the aortic region. The entrance into the aorta is kept uncontracted by means of the "infra-aortic space," which remains permanently patent and charged with blood; and a similar arrangement probably prevails in the pulmonary infundibulum. Into this space the sigmoid flaps play without any solid contact or friction, bathed in blood above and below, and to this they owe their life-long preservation as delicate filmy membranes.

Of greater practical interest to us is the "supra-papillary space," a varying space complementary to the aortic space. Within it we notice the important "retro-mitral space," which is included between the posterior mitral flap and the heart wall, although it would be warrantable to apply this term to the entire ventricular district beyond the plane of the anterior mitral flap. During systole, when the lower ventricular space is a solid mass of contracted, tightly-packed muscles, the suprapapillary region is imperfectly filled by the valve membrane and its chordæ. According as we assume that it retains much or little blood, our ideas as to the mechanism of mitral closure and of its pathological failures must vary. There can be little doubt that the great mitral flap is driven to the left by the systolic blood pressure, and that the infra-aortic space absorbs a great part of the suprapapillary space. If blood could be entirely excluded from the suprapapillary district, the chance of any leakage into the auricle would be reduced to *nil*. This would mean complete adaptation of the membranes to the left ventricular wall itself, such as is observed in the case of the internal tricuspid flap. There are, however, reasons for supposing that the retro-mitral space may not be completely drained, and that a retro-mitral cushion of blood may remain as an additional help to secure the obliteration of the orifice. We shall see that whatever the precise mechanism of the mitral closure may be, it must be credited with considerable adaptability to varying conditions.

The Progress of the Blood through the Heart.

This is too familiar a subject to call for many remarks. The muscular contraction and the elastic recoil of the myocardium, which are the driving forces, are also the indirect means of interrupting the flow by shutting off the cavities from each other. Ample provision is made for a continuous supply to the cardiac pump, as, for instance, from the pulmonary veins; but the heart's action renders it intermittent.

On the left side the first delay is produced at the auricular orifice of the pulmonary veins, which have been credited with a systole of their own. Whether it be their independent contraction or the systole of the auricle that bars the way, the blood accumulates within them for a brief period, but during the much longer remaining time it is pouring steadily into the auricle after the initial sudden aspiration of their delayed contents by the auricular relaxation.

The second stoppage and accumulation begin within the auricle with the closing of the mitral and continue during the ventricular systole. The third stoppage is brought about by the closure of the aortic valves, and blood then begins to fill the ventricle. The powerful ventricular aspiration rapidly unloads the auricle through its now widely patent orifice. This is the great suctional ventricular inrush. The volume of the heart as a whole is not altered, but merely that of its component parts. For a while blood continues to flow in unobstructedly from the auricle and the pulmonary veins, but with diminishing speed. When the negative ventricular aspiration is exhausted active contraction follows. The veins and the auricle contract and rapidly complete the ventricular repletion. This is the second and final ventricular inrush soon to be followed by the ventricular systole. But the delay is not at an end. During a brief and variable interval the blood is held by the ventricle, and its charge neither grows nor lessens. But its pressure is being raised; a struggle between the incipient ventricular contraction and the resistance of the aortic blood pressure is proceeding within the pressure chamber which the mitral valve maintains closed.

Potain,⁴ however, contends that the closure is not yet completed, and that meanwhile, as described by Samways, the persisting auricular contraction is the means of preventing regurgitation. He terms this short delay the "ventricular presystole." Chauveau,⁵ who refuses to admit any overlapping of the two systoles, and who believes in an immediate relaxation of the auricle, defines the interval in question, under the name of "intersystole," as a period for internal adjustments, and in particular for the closure of the mitral by the papillary muscles. Almost alone among physiologists he assumes that the contraction of the latter precedes that of the ventricle.

I would suggest provisionally that the mode of closure to be presently described as membranous or diaphragmatic may belong normally to this presystolic period, and that it may be made more secure by a continued contraction of the lower part of the auricle and of its appendix, which may last up to, but probably not outlast, the first sound. From that moment the ventricle works alone and at high pressure. Every fibre within it shares in the effort to set in motion in an instant the vast column of blood which lies beyond the semilunars, and not least is the share belonging to the papillary muscles. The rest of the systole needs no further comment. The outflow continues, but the blood pressure steadily falls until the sigmoids, closing once more, mark the end of the cardiac cycle.

THE MITRAL VALVE AND THE MECHANISM OF NORMAL MITRAL CLOSURE.

The normal mitral closure is due to the contraction of the official fibres and to the close adaptation under blood pressure of the valvular membranes. The older view, which was based upon misleading schemata, and which failed to take into account the systolic contraction of the muscular orifice, need not, however, be entirely discarded. Clinical observers familiar with the singular adaptability of the heart to the varying conditions of disease are prepared to consider that the normal cardiac mechanisms are not of necessity uniform in all subjects. Two identical hearts are probably as impossible to find as two identical faces, and differences in function must follow the differences in structure. Moreover the same heart may vary in its contraction during periods of vigour or of exhaustion. This reservation should never be omitted in our discussions.

The mitral membrane is not made up of separate flaps, but is a single membrane extending continuously all round the orifice (see specimens). It forms a shallow wide-mouthed funnel, of which the narrowing funnel of stenosis is merely an exaggeration. Nevertheless the anterior and posterior flaps are well individualised both in their anatomy and in their function. The great anterior flap fulfils various purposes, but it never ceases to be a ventricular partition, and during systole it is kept spread out and smooth. The posterior flap, which forms the greater part of the circular membrane, but is much shorter in the vertical direction than its fellow, is essentially mobile, being attached to the entire length of the fibrous loop and of the contractile border of the ventricular wall. During systole it is folded into numerous pleats like a purse; but the pleating is done not by the pull of a string, but by the contraction of the official fibres, and the pleats are arranged by the chordæ from below. The stretching of the great flap is effected by its two strong diverging chordæ and higher up by the two mitral insertions which connect it with powerful muscles and with the aortic roots and fibro-cartilages; its lower part and its fringe alone are capable of flapping to the extent which official space and chordæ will allow, but during complete oblitative systole there is neither much space nor much rope, and this flap as well as the posterior flap is drawn almost vertically downwards.

The High Closure and the Low Closure of the Mitral Valve.

These are the two extreme types associated respectively with an uncontracted and with a contracted orifice. The first, seen in dilated, in partly paralysed, and perhaps in some peculiar normal hearts, is relatively precarious, the second, which is distinctive of thoroughly normal and strong hearts, is made doubly secure.

As often as I have opened hearts in fairly good systole—for absolute systole must be quite exceptional in dying hearts—I have found the flaps drawn down by the papillary muscles. I

do not regard this attitude as due to rigor mortis or as exaggerated by it, but on the contrary as falling short of the yet greater contraction which must occur at the acme of systole. More or less depression of the flaps is the inevitable result of systole, and its extent may be taken as a measure of the quality of the mitral closure, which may be accordingly perfect, barely efficient, or defective.

Should the systole fail to empty the retro-mitral pool or to obliterate the muscular orifice, the closure will be almost exclusively membranous, the anterior flap much more oblique and at its lower part almost horizontal, the posterior flap less pleated and likewise horizontal, the opposed fringes of the flaps alone coming into apposition in a vertical direction. This is the high closure, or purely membranous closure by horizontal adaptation. In the normal type of low closure the orifice is contracted, the membranes are vertical, and they are in apposition not only at their fringes but over a considerable part of their surfaces, which are pressed back to back and applied to the outer ventricular wall by the intra-ventricular blood pressure, whilst their attachments may either meet if the muscular orifice should be tightly closed or be separated by a small wedge of auricular blood, and at the same time bulged slightly upwards by the ventricular blood. This is the perfect and secure musculo-membranous closure of a completely contracting and emptying heart; and it is obvious that the farther down the membranous funnel is drawn, so much the more extensive is the surface of mutual contact, and so much greater will be the tendency of the long anterior flap to apply itself to the ventricular wall below the level of the posterior flap, as a first defence against regurgitation, and thus to shut off entirely the mitral corner from the main pressure chamber of the ventricle. Compared to this there is little security in the mere diaphragmatic closure by membrane, which is suited only to meet the diminished pressure associated with an imperfect contraction of the orifices, of the heart walls, and of the papillary muscles.

THE FUNCTIONS OF THE PAPILLARY MUSCLES, VALVES, AND CHORDÆ; AND THE FIRST SOUND OF THE HEART.

How is the normal systolic position of the valves brought about after their diastolic elevation? As soon as the auricular systole and the early presystolic contraction of the ventricles have overstretched everything, the powerful and rapid papillary contraction probably takes place simultaneously with the first sound. By this the chordæ and their membranes would be stretched downwards and made taut, helping to depress the ventricular orifices. The anterior mitral flap, whilst transmitting additional tension to the aorta and to its extensive membranous and fibrous connections, would probably remain moulded by blood pressure into a concave shape as part of the infra-aortic space. Its rapid tension between its upper and lower attachments imitates in action, in driving force, and in sound that of a sheet skillfully shaken from both ends. It also acts as a tendon with an extensive insertion, and its function as a pressure-pad has been fully explained. These varied results of the contraction of the papillary muscles almost suggest that the latter supply from a distance the place of the absent basic muscles for the open side of the ventricular cone.

Another important function of the papillary muscles is their share in the production of the first sound. The fact that this sound is still obtained in a heart contracting without any fluid contents, in which the valves cannot be made tense by blood pressure, has been used as a proof that the sound is largely one of muscular vibration. It is, I think, equally compatible with a view that the sonorous tension of the valve is due to the papillary contraction, whilst counter-tension is kept up by the systolic retraction of the two mitral insertions and of the aorta. The characteristic booming sound is exactly that which we might expect from the tension not of one membrane, but of a long range of membranes varying in size, in shape, and in quality.

I need hardly repeat that not the least of the papillary functions seems to be that of steadying and of adjusting the aorta from below and of providing for its patency. The papillary muscles are tensors of the aorta, a duty which is shared by numerous muscular fibres working upon other points of its circumference. The relation of the papillary muscles to the movements of the mitral flaps can hardly be questioned, but it is strikingly demonstrated in some strange

hearts such as those exhibited, in which one of the muscles is continued upwards and is directly inserted into the surface of the flap without any chordæ. The powerful depression of the orifice which so long a muscle must be capable of effecting also suggests that the ventricular systole by depressing the orifice must exercise a pumping action upon the auricular contents.

THE AUTOMATIC REGULATION OF THE AURICULO-VENTRICULAR PERISTALSIS AND THE DIASTOLIC FUNCTION OF THE VALVES AND CHORDÆ.

Continuity of muscular fibres across the auriculo-ventricular groove does not seem to obtain in man; of this you may judge from the specimens. A. F. Stanley Kent⁶ has however described in animals bridging fibres which he believes may transmit the systolic stimulus. He has also traced some continuous fibres passing from the auricular into the ventricular septum. W. His, jun.,⁷ limits this continuity to a single bundle of septal fibres, but he has demonstrated it in man as well as in animals. In the vertical sections which I have made through the left auriculo-ventricular region, you will notice that the descending bundle of auricular fibres and

Fig. 2.—Vertical section through the auriculo-ventricular junction, from the human heart, showing the complete separation of the auricular wall from the ventricular myocardium and the connection of the two cavities by endocardium only; E, endocardium; P, epicardium; U, the "fibrous loop" at the bottom of the auriculo-ventricular groove; V, the posterior mitral flap with chordæ and papillary muscle; R, the "retro-mitral" space; M, the delicate external sheath enclosing the myocardium and uniting with the endocardium and fibrous loop at U.

the thick rising shoulder of ventricular orificial fibres are both isolated between their endocardium and a delicate outer sheath, these two membranes uniting before they are inserted into the fibrous loop. The loop also gives attachment on its internal aspect to the duplicature of endocardium which constitutes the posterior mitral flap. Between the two sets of muscles there is no visible, I might say no probable, continuity, and the connection between auricle and ventricle is absolutely reduced to the slender endocardial membrane; this, however, is amply protected from outside by the soft auriculo-ventricular fat embedding the vessels, and it is rendered more elastic by the epicardium which passes over it and consolidates the groove. By removing the fat the fibrous loop is easily exposed. This is an admirable arrangement from our clinical standpoint, ensuring complete independence between the two myocardia, and allowing them and the valvular attachment also to play up and down, auricle into ventricle, in obedience to the papillary function; but it does not explain the automatic punctuality of the systolic events.

Possibly—but this suggestion is made under all reservation—the regulation of the rhythm may be in part mechanical, and analogous to that of most of our muscular adjustments, namely, essentially myotatic. It is not unreasonable to look

for a double function in the valve flaps and chordæ, under the opposites of systole and diastole, analogous to the two opposite functions of the myocardium. The "open parachute" diastolic attitude of the flaps is only a partial closure incapable of stopping the continued inflow of blood, much less the final inrush due to auricular systole. After the latter, and during the brief presystole of the ventricle, it may provide a complete membranous closure for the still imperfectly contracted orifice, but papillary contraction would soon bring down the flaps to their vertical or systolic attitude. The chordæ likewise would have two positions of tension, a momentary diastolic tension upwards when the flaps are fully floated up, and a more sustained systolic tension downwards; and the latter might conceivably be called forth by the former.

More broadly we might regard the auricular systole, which presumably increases the diastolic elevation of the valves and the upward tension of the chordæ, as an inducement for the ventricle to contract. Each of the chordæ would be likely to exert a pull upon its own set of muscles in some definite succession, according to its individual length. This might perhaps account for the partial presystolic contractions which are so difficult to explain, and for the equally mysterious lateness of the papillary jerk. It so happens that the retro-mitral groove is the seat of very short tendons hidden beneath the posterior mitral flap; their early tension might be the means of exciting the extreme left of the ventricle to early contraction. MacWilliam's experimental sections of the chordæ seem to have been carried out entirely below the level of these tendons. Once started the mechanical excitation would travel from segment to segment of the fibres till it reached their most distant end; and I do not lose sight of this alternative explanation for the lateness of the papillary contraction. Another myotatic agency might exist in the pull exerted by the auricular systole upon the fibrous loop itself.

A different explanation is that by a direct overtension of the heart wall, its various fibres being unevenly stretched by the growing ventricular charge, and therefore successively excited according to the different length of their course in the ventricle. In both cases the regulation as to time and as to energy of contraction of various parts of the heart would be under the influence of the highly variable scale of internal pressures, one of great delicacy and capable of affording some explanation for the great variations of the cardiac rhythm in health, and for some of its remarkable pathological abnormalities.

III.—CLINICAL ILLUSTRATIONS.

Functional and Organic Mitral Murmurs.

1. The common systolic functional murmurs are still imperfectly explained. Their eventual disappearance suggests that the valvular structures are sound, and that there is merely some muscular or neuro-muscular derangement, with temporary failure of official closure as a result. 2. Orificial defects may also arise from structural causes. The murmurs are then organic, but more commonly these murmurs are associated with valvular deformities. Leaving aside vegetative valvulitis and ruptured valves, the structural changes at the onset may be so slight as to elude a superficial inspection, and they may produce little more than transient limitations of movement. But unlike the rheumatic joint, the heart can never be absolutely rested, and if the infiltration is not merely myocardial, but should also affect the valve, it is likely to progress to the verrucose stage, and to those roughenings, thickenings, puckerings, and obstructions which we all know tend to leakage by faulty apposition.

Myocardial Stiffness as a Cause of Valvular Defect.

If, as originally contended by Sturges, acute rheumatism is capable of affecting the whole heart, the chief stress of minor attacks might variously fall upon the fibrous and membranous, or the muscular, or the tendinous structures. And bearing in mind the special liability of the orificial and of the papillary muscles, it is conceivable that, short of any serious damage to other structures, these muscles might suffer in their finer adjustments. Systolic orificial closure might be impeded or delayed, with regurgitation as a result; or muscular stiffness might have the opposite effect of delaying the diastolic expansion of the ventricle. And the rapid release of the valves and chordæ, a circumstance which, if coupled with any passing oedema of the flaps themselves, might well account

for the premonitory sounds of mitral stenosis. A majority of the mitral functional murmurs not due to atony may be of this kind, and the recovery from the myocardial engorgement and stiffness may be permanent. Too often, however, it is only temporary, and the seeds are laid for structural changes.

Epicardial Stiffness as another Cause.

We have studied in vertical sections the normal auriculo-ventricular groove. See how severely in this instance (Fig. 3) its contents have suffered. Replacing its delicate fat

Fig. 3.—Vertical section from a case of indurative pericarditis showing fibrous thickening in the auriculo-ventricular groove, in contact with the "fibrous loop" and endocardium. F, dense fibrous false membrane; P, epicardium; M, delicate external sheath enclosing the myocardium; E, endocardium; U, the "fibrous loop" in section; V, the posterior mitral flap; R, the "retro-mitral" space.

a tough flesh-like fibrous deposit has crept into its recesses, welding together the auricular and the ventricular muscles, destroying their finer mutual play, and crippling the orificial contraction. But it has also attacked the fibrous loop and the outer surface of the endocardium, that slender and only barrier between the groove and the valve. We now realise how pericarditis may determine a valvulitis by extension.

EARLY MITRAL DISEASE AND ITS EVOLUTION INTO OPPOSITE TYPES.

In the earliest stage of mitral disease we discover none of that differentiation which afterwards is so marked. The mitral lesion is almost invariably localised near the edge of the flaps where their auricular surfaces meet, and have to bear friction and pressure during the "soft" stage of their rheumatic swelling. Why should the late result be in one case pure mitral incompetence, in the other pure mitral stenosis? When and where does the difference originate? I am impressed with the probability that there is from the first an essential difference; but I doubt whether it can be mainly traced to any peculiarities, observed or assumed, in the early valvular lesions.

Anatomically there is room for differences which may have been overlooked, particularly in connection with the chordæ tendineæ. These may be in the one set of cases much more extensively involved than in the other. Again, the anterior and the posterior incisures, which are peculiarly rich in chordæ, are perhaps more liable than other parts to the change. Attention must also be called to the fact that shortening of the chordæ may be secondary. Where there is puckering of the flaps the retreating edge of the fibrosed valve must drag upon the fine chordæ attached to it—nay, probably they are involved in the fibrosis. These changes often pass unnoticed at necropsies, but they may be sufficient to bridle the flaps, to cripple their free swinging, and to transform them from flaccid into stretched membranes. Lesions of this sort are probably not limited to any special set of cases, and do not, therefore, supply the desired explanation.

Clinically definite distinctions exist. The murmur of structural mitral incompetence is audible, practically speaking, from the first, and once heard it is always heard again. But, whilst mitral incompetence develops early and abruptly, mitral stenosis can only be slowly produced. The stethoscope may catch its early forebodings, but these are often inconstant, and it is significant that they are in most cases not complicated with any audible regurgitation.

Perhaps the determining factor resides less in the peculiarities of the lesions than in those of the action of the valve. In early valvulitis I am led more and more to ascribe mitral incompetence to defective orificial closure and the early disposition to stenosis to the absence of this defect. A fairly safe guide in this study is the nature of the ultimate deformity of the valves. In mitral stenosis the flaps are drawn down and often elongated to an extraordinary extent; but in pure mitral incompetence there is no shortening of the chordæ and no elongation of the flaps, which, on the contrary, are much retracted towards their attachment. We are reminded of the contrast between the high closure and the low closure, and this contrast seems to provide us with a rational theory.

Assuming as a common factor the inevitable fibrosis and retraction incidental to all warty valvulitis, the direction of the progressive change will vary with the habitual attitude of the valve. If the valve be constantly kept well down in the ventricle, fibrosis and shortening are likely to overtake the chordæ. But if from abnormal patency of the orifice the flaps be raised high at each systole, the flaps and not the chordæ will be the chief seat of shrinkage. The chordæ will be submitted to a systematic elongation at the time when, owing to rheumatic or inflammatory softening, they are most easily stretched, their weakness thus becoming a protection against all danger of stenotic shortening. Stenosis, according to this slightly paradoxical view, would be the disease overtaking strong, normally-acting hearts; incompetence the disease of yielding, atonic myocardia. And this, I believe, will agree with your experience of the great prevalence of the stenotic affection, and of its predilection for young and healthy hearts with good cardiac sounds.

THE LATE OR FUNNEL STAGE OF MITRAL STENOSIS.

(*Auricle in Ventricle.*)

I now turn to the totally distinct conditions of advanced mitral stenosis by which I do not understand the terminal condition of broken compensation. There may exist in exceptional cases as a "horizontally" stenosed mitral valve, one without any cupping or depression. My remarks will not apply to that condition. The usual anatomical features are the enlargement of the auricle in every direction, but particularly downwards, either in the shape of a funnel or of a cup, and the considerable stretching of the mitral membrane by the retarded auricular contents. I regret to have to add to the complexity of the problem of mitral stenosis and of its physical signs by introducing an additional unknown quantity. The funnel seems to me to take a large share in all the aspects of this stage. Its presence entails a radical alteration in the cardiac mechanism. Whilst normally the auricle is intended to contract independently of the ventricle, in this affection it permanently invades the upper ventricular space—an auricle inside the ventricle, and it is thenceforth subject not only to its own contraction, but to that of the ventricle also. I can only touch upon a few of the features of this strange complication, as it respectively affects the ventricular, the mitral, and the aortic and arterial functions.

(a) *The Ventricle.*—The most obvious fact is the encumbrance of its space. During systole the mitral membrane of the thoroughly healthy heart is stowed away like a thin empty bag, but in stenosis the bag is at its maximum distension. We must remember that whilst the auricle proper is straining with all its muscle to deliver its blood into the funnel there is no contractile element in the latter; it is a passive reservoir, a dilated channel. In the first place, then, it plays the part of a foreign body wedged into the ventricular orifice, and distending it at the moment when it should be contracting. Great delay must follow, and there is little wonder that an unusually long presystole should be a distinctive, and in the matter of impulse a tangible, feature of stenosis. This pre-

systolic delay and impulse have of late attracted much notice, and we owe to Potain, Chauveau, Bard, Sansom, Samways, Brookbank, Gibson, Gibbes, and others some interesting literature which it is impossible to discuss otherwise than by suggesting this additional explanation.

(b) *The Systolic and the Mitral Events.*—Of the normal behaviour of the mitral valve little is left but the tension of the stiffened membrane by papillary effort, which to my mind is a sufficient cause for the loud first sound. Everything else is changed. The long presystole has been spent in a struggle with the contents of the funnel which delay the rise of ventricular pressure, for they are steadily leaking away, possibly, I would suggest, in two directions, though I speak with some hesitation, believing that things must vary with cases, and the cases vary greatly. For instance, has the ventricle already completed its charge or is it still further dilatable by the auricular pressure? Is the auricle empty or still full and stretched by residual blood? In a word, on which side of the valve is the maximum pressure in the individual instance? Without due answers no general statement can be made. One thing, however, seems to be certain; however long the auricular systole may be assumed to last, it relaxes earlier than the ventricular systole, and therefore if any blood still remains in the funnel, this would find at that moment an easier way into the auricle than into the high-pressure chamber of the ventricle. I regard it as possible that in the average cases—those cases which adapt themselves most successfully to their mechanical difficulties—the ventricular orificial fibres close up early upon the bag and drive the blood from it with rising speed and pressure into the ventricle, whilst a great part of it also is forced back into the auricle, this reflux continuing if necessary after the onset of the true ventricular systole, which probably checks the intraventricular flow.

THE DIASTOLIC AND PRESYSTOLIC MURMURS.

I may now briefly refer to the influence exercised by the funnel upon the sounds and murmurs. In those cases where the ventricle is still struggling successfully, and manages to empty the funnel, Sansom's rational explanation is that which I adopt, with a slight modification, for the first and earliest diastolic *bruit*—namely, that it is due to the sudden descent of the blood dammed up in the auricle into the capacious funnel. As to the "rumble" kept up in some hearts throughout the diastole, I am not prepared to express any positive opinion. I incline to regard it as due to the injection of a strong stream of blood from the often irregular and rough orifice across the stiffened papillary tackle.

The presystolic murmur is, I believe, that of a fluid vein likewise making for the apex. It might be compared to the stream from a syringe forcibly emptied under water, for the ventricle is already nearly full, and the chordæ less apt to vibrate. Its crescendo character may be explained by the growing pressure of the ventricular orificial presystole upon the funnel.

The Systolic Murmur.

The occurrence in many cases of a murmur absolutely continuous with the first sound has been the strongest argument of the supporters of Barclay's theory which regards the presystolic murmur as essentially systolic and regurgitant. I have just stated my belief that it is produced by that portion of the ventricular systole which we are agreed to define as the presystole; but that it is not a regurgitant but an onward murmur. Its continuity with any subsequent regurgitant murmur is compatible with that view, and I know of no other means by which it could be accounted for. My provisional explanation is that, during the presystole, there are two streams from the funnel, the auricular systolic pressure preventing, however, much regurgitation, but that as soon as the first sound marks the cessation of the intra-ventricular flow the reflux is so much increased and accelerated as to cause an audible fluid vein. According to this view there is no absolute reversal of current; some blood was previously being driven back into the auricle, and therefore there need be no interval between the first sound and the murmur.

The Disappearance of the Second Sound.

In all this I have assumed that lateral pressure is brought to bear upon the funnel, and finally succeeds in emptying it.

But if the contractile fibres of the ventricular orifice had been worn out by the invincible resistance of a permanently distended auricle and funnel some of the murmurs would be obscured. This is precisely the condition observed at the later stages. Not only the presystolic murmur disappears, but with it the second sound at the apex. I am not sure that any explanation has been given for this mysterious disappearance, but I believe that it is to be found in the presence of the funnel. You are aware that the aortic second sound is not really extinguished in these cases, and that it is still audible at the base, though often much enfeebled. The fault is clearly one of conduction, and until lately I had failed to account for the causation of the defect. My present opinion is that the conduction of the sound is "blocked" by the funnel, which at the time of maximum systolic contraction of the heart usurps the infra-aortic space. Doubtless its pressure may sometimes be felt by the semilunar flaps themselves, and may damp their vibrations with corresponding enfeeblement at the base. But apart from this the pressure upon the septum may so much interfere with their conduction as to render them inaudible at the apex.

(c) The influence exercised upon the arterial system is not limited to the aorta and to its valvular sound, but may be traced in the pulse. The pulse of mitral stenosis is of two opposite kinds, according as compensation is perfect or failing. In the former case it closely resembles that of moderate aortic stenosis, and I believe that it is due to a slight obstruction in the aortic current. Formerly I supposed that the aorta suffered pressure in its first portion just beyond the ventricle, owing to the distension of both auricles and of the pulmonary artery; for further details I must refer you to a paper which I published on that subject.⁹ That view may have been partly correct, but I am now disposed to regard the obstacle as intraventricular, and situated in the "aortic channel" rather than in the aorta. In the compensated stage the funnel merely encroaches upon and narrows the infra-aortic space, but when compensation fails its increasing size sets up a dangerous obstruction.

My present task is at an end. A few illustrations were needed to justify the mode of study which I have ventured to recommend, and to show you that its opportunities are not exhausted but almost inexhaustible; but these were merely incidental. The main purpose was to indicate the importance of an adequate groundwork for our intricate clinical studies and the greater use which might be made by clinicians of our yet imperfect insight into the anatomy and physiology of the heart.

The crude sketch which I have ventured to present to you invites criticism and claims future revision, but it may perhaps be of temporary use in reminding us of the complexity and of the variability of cardiac conditions even in health, of the need of a closer inspection of the footprints of disease, and of the futility of discussions which do not discriminate between its variations, its extremes, and its essentially dissimilar stages.

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THE MOLOKAI COLONISTS.—From the *Medical Age* we learn that there are at present 1,100 lepers residing in Molokai, 625 being males and 475 females. Of these 3 are German, 4 are British, 5 Americans, 6 Portuguese, 37 Chinese, 62 half caste, and 984 are Hawaiians.

NEW DOG-MUZZLING ORDERS.—In consequence of the recurrence of rabies in South Wales, the Board of Agriculture has published orders reimposing the regulations as to the muzzling of dogs in parts of the counties of Brecon, Carmarthen, and Glamorgan. The orders came into operation in each case on the 15th inst.

A CLINICAL LECTURE

ON THE

PREVENTION OF VALVULAR DISEASE OF THE HEART IN CASES OF ACUTE RHEUMATISM.

Delivered at the Liverpool Royal Infirmary.

By RICHARD CATON, M.D., F.R.C.P.

Physician to the Infirmary.

GENTLEMEN,—It is your constant experience and mine day after day on the medical side of this hospital to meet with cases of organic heart disease. These poor people come to us suffering from dyspnoea, dropsy, and weakness, after too often a severe struggle to accomplish the daily task needful for the earning of a livelihood. They have striven hard against increasing difficulties, until at length Nature can do no more, and they come to us almost in despair. We treat them as out-patients, or more frequently take them into our wards, and give what temporary help we can. Nearly always we get the same sad story from them—a bad attack of rheumatism, after which they were never the same as before; increasing breathlessness, then swelling of legs and feet, and inability for hard work. It is a painful experience for the doctor, especially when, as so often happens, the patient is young, and his whole life is blasted by this terrible breakdown of circulation, a breakdown the urgent symptoms of which we can often alleviate, but the underlying essential defect of which we can never repair. Under these circumstances it is not wonderful that some of us have felt the urgent need for efforts to prevent this injury of the cardiac valves in rheumatism from which so much suffering and disablement result. No one can deny that the inquiry is important and is worthy of our best efforts. Nevertheless it is the fact that the great majority of our profession at present, while treating the rheumatic affection to the best of their ability leave the cardiac risk to fate; they are deeply concerned if endocarditis occurs, but believe themselves powerless to prevent or influence it in any way.

Many of you have observed the care with which in my wards the heart is examined daily in every case of acute rheumatism, and also that certain rather unusual measures of treatment are applied. They are employed with the object of preventing organic valvular disease. I have told you why I employ these measures; let me explain to you the theory on which the measures are founded, and give you a brief summary of the results which have hitherto been attained, and finally I wish to lay down one or two principles which it seems to me we should always keep in mind in the treatment of these cases:

1. On what theory can we reasonably hope to exert any influence on the endocardium, to prevent or assuage rheumatic inflammation, to remove the products of such inflammation, and prevent the crippling of the valve?

In the first place we must stop the rheumatism as rapidly as possible, and prevent all aggravation of it by chills. We therefore keep the patient absolutely at rest in bed; profuse sweats usually occur, in which there is great danger of chill, I therefore clothe the patient from head to foot in a warm flannel garment; a large stock of these vestments is kept in my wards expressly for rheumatic cases. Salicylates are given in full dose, often with alkalies, and chologogues in such measure as to cause free evacuations but no diarrhoea. The diet is confined to milk and light farinaceous food; no red meat is given for a long time. The patient is kept in bed long after all pain and fever are gone, for never forget that the salicylate treatment, while removing pain and fever in twenty-four or forty-eight hours, does not remove the rheumatic entity itself (whatever that unknown entity may be) after less than two or three weeks of steady administration. The treatment I have thus far mentioned has nothing particularly new in it, but if you carry this out with strictness you will have a low percentage of cardiac complication. Out of many hundreds of cases, I have only had about 15 per cent. of cardiac troubles, which is a low average.

But even if you do all that, you will have some cases of cardiac trouble, and of course it will often happen that the mis-