An Address ON MITRAL STENOSIS, Delivered at the Opening of the Winter Session, Post Graduate Course, Mount Vernon Hospital for Consumption and Diseases of the Chest

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An Address on MITRAL STENOSIS.
DELIVERED AT THE OPENING OF THE WINTER SESSION, POST GRADUATE COURSE, MOUNT VERNON HOSPITAL, FOR CONSUMPTION AND DISEASES OF THE CHEST.
BY
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[After some introductory remarks, Sir James Barr stated that his address was based on his own previous work on the subject brought up to date. He considered the subject much too large for a single address, so he would confine his attention chiefly to the debatable points in the clinical manifestations.]

The significance of the murmur of mitral stenosis was first clearly recognized by Fauvel, and by him designated presystolic, but it was the late Sir William Gairdner who established the clinical signs of the disease and their pathological importance, so that when the murmur, which he termed auricular-systolic, or briefly the A.S., murmur, is present, the disease becomes one of the most easily recognized affections of the heart. Sir William Gairdner’s first contributions on the subject were published in his Clinical Medicine in 1862, and there has since been a slow but gradual evolution in knowledge of the subject, and with the great amount of literature which has been devoted to its exposition, the real additions to knowledge are comparatively trivial, and the value of some of these additions an unknown quantity. It should be clearly understood that the disease is easily diagnosed apart from any murmur, but such diagnosis is very imperfect unless it takes into account the extent of the lesion and its effects. There is still much to be done in the way of prevention and in retarding the evolution of the disease.

ETIOLOGY AND PATHOLOGY.

Of all the causes or exciting conditions of endocarditis and pericarditis, rheumatism holds the first place. The fibrous tissue seems to be specially vulnerable to the rheumatic poison, but the myocardium may be involved as well as the whole endocardium and pericardium in the inflammatory process. The valvular lesion is determined by the violent collision of the vulnerable valves, and a review of causation has a most important bearing on prevention and treatment, as by relieving the laboured action of the heart in the early stages of endocarditis the mischief can be avoided or mitigated.

When the rheumatic poison, whether it be animate or inanimate, sets up endocarditis, the myocardium is, in the majority of instances, more or less involved. The systole of the ventricle is slow and labored to overcome the high intracardiac pressure which is set up by increased peripheral resistance. As compared with the temperature the pulse in these cases is not very frequent, and may not exceed 90 even with a temperature of 103° F. The pulse is slow, full, and moderately strong, and the tension is well maintained. The blood pressure is much higher than it is in typhoid fever, pneumonia, or diphtheria. Among the earliest signs of endocarditis is a dull first sound and a delayed radial pulse, so that the former can be heard an appreciable interval before the latter is felt. By careful attention to these signs, endocarditis can usually be diagnosed a few days before any murmur.

The much more frequent occurrence of valvular disease in the left heart than in the right is due to the much greater strain and more violent collision to which the valves are subjected. When the mitral valve is involved we get a beaded row of vegetations on the auricular surface of the valve about ½ in. or 3 in. from the margin where the cusps come into most violent apposition; and when the aortic valve is involved the damage occurs on the ventricular surface at the angle of the cusps, which are brought into forcible collision during their closure. The vegetations arise from proliferation of the cellular elements in the sub-endocardial layers of the endocardium. The cells first through the endothelium, and fibrin is deposited on the rough surface. This has a most important bearing on treatment, as by keeping the blood pressure extremely low in rheumatic fever, endocarditis is avoided.

Unfortunately the mischief has often occurred before we see the patient, and when the valve has been once damaged there is a marked tendency for the mischief to be maintained and perpetuated, even under the ordinary actions of the heart. It is much aggravated by violent collision of the cusps during palpitation, or from high intra-cardiac pressure from any cause, perhaps from the medullary interference of the physician with that much-abused drug, digitalis. When free mitral regurgitation takes place, the blood-pressure falls, and the pulse increases in frequency.

Children are very liable to rheumatism, especially those with a hereditary tendency to the disease; rheumatic affection may be so slight as to escape notice, and in many cases endocarditis is the only gross expression of the rheumatic affection. Following this endocarditis, which may even not have been detected, mitral stenosis is not an infrequent sequence. Many writers have asserted that mitral stenosis is essentially a disease of early life, and some believe the disease to be frequently congenital. While not denying the occasional congenital origin of the lesion, I think it must be extremely rare, as the affection is very seldom detected under 5 years of age. It is very common in youth, but may occur at any age.

When it is directly associated with acute rheumatism the edges of the cusps which are principally affected become gradually united so that the funnel form of the lesion is eventually established. Frequently these cases are associated with incompetence, pure and simple, due to the fibrous contraction of the auriculo-ventricular orifice and agglutination of the edges of the valve segments, they finally end in stenosis either with or without regurgitation. Mitral stenosis is frequently associated with chorea, and here the mechanical determination of the disease is very apparent. It may arise in cases of protracted anemia associated with high blood pressure and mental excitement, but it does not arise in cases of exophthalmic goitre in spite of the violent palpitation, because the blood pressure is low and the lime salts, which are another determining factor, are deficient. It is not uncommon in chronic Bright’s disease, and in these cases mechanical strain plays a most important part in its causation. In such cases the orifice is not usually much contracted, but the whole segments of the valve are much thickened, and may be atheromatous or contain calcareous nodules consisting of lime salts or sodium urate. The musculi papillares are much hypertrophied and the chordae tendineae are thickened. The insertions of the chordae are spread out over the greater part of the cusps, so that when there is long-continued high intracardiac pressure the whole valve becomes very much thickened, and there is an increase in the fibrous tissue both to puckering or contraction of the segments and the diaphragmatic or button-hole variety is produced. Syphilis has also been set down as a cause, but if so, it must, in my opinion, be a very rare one.

Byrom has shown that in some cases large calcareous nodules in the walls of the ventricle—causing obstruction—are syphilitic gummata which have become calcified.

Mitral stenosis is more common in females than in males, for the simple reason that rheumatism and chorea occur more frequently in girls than in boys, and anaemia and all emotional disturbances have a special predilection for the female sex. However, men are much more liable to this disease than is generally supposed; and, in fact, the disease is much more common among both sexes than is usually recognized. This, from the fact that it is often overlooked when associated with mitral regurgitation. When associated with tricuspid stenosis I have seen as many cases of the double lesion in men as with mitral stenosis is frequently associated with pericardial adhesions, and this arises from the fact that pericarditis is almost as common as endocarditis in children.

The disease is, as a rule, slowly progressive, and although sooner or later it leads to the death of the individual, yet under proper care and treatment it is very slow in its progress, and is quite compatible with a fairly long existence. I have known cases of mitral stenosis of twenty to thirty years’ standing. In some cases of
Mitr al Stenosis.

When we hear at the mitral area a murmur immediately preceding the first sound, or even a murmur during the long pause, it is a very simple matter to recognize the fact that the obstruction to the blood from the auricle to the ventricle, but such an observation cannot be properly called a diagnosis. We want to know the nature of the obstruction, the size of the orifice, and the effect on the circulation. Any observer can recognize a presystolic murmur, but it requires some training to interpret its significance. A great many men base their diagnosis of the lesion on the presence of the characteristic murmur, and as it is absent in a large number of cases, they thus fail to recognize the disease, even in the very extreme and most urgent cases.

In a large number of cases in which the murmur is well pronounced, there is an interference of the physician is uncalled for; but in many cases in which it is very slight, judicious treatment may be of vital importance. In many cases the only murmur heard is a mitral systolic, due to regurgitation. Now, in my opinion, the treatment of these two affection is identical, the only difference being, and the failure to recognize the underlying condition of mitral stenosis may be fraught with great danger to the life of the patient.

The characters of the murmur and of the first sound may help us to estimate the nature of the orifice and the condition of the cusps, but it is only by an examination of the pulse, and by a careful study of the effects of the obstruction, that we can arrive at the far more important calculation of the size of the orifice or the amount of obstruction.

Of all the signs of mitral stenosis, the murmur is the most obtrusively manifest, and, therefore, has perhaps received undue attention, to the neglect of other more constant, and to my mind more important, signs. The characters of the murmur in various forms of the condition, have been described and depicted by numerous writers, that very little new can be said on the subject, but, nevertheless, the intensity of the murmur prevents our passing it over in silence in the consideration of another lesion which, in a number of cases, is the prominent feature. There is, perhaps, no cardiac murmur which such a conflict of opposing opinions has been waged. Even the very rhythm of the murmur has been disputed, but at last we have arrived at an almost unanimous opinion that the murmur occurs prior to the systole of the ventricle. Sir William Gairdner happily designated it as "auricular systolic," which implies a theory as to the causation of the sound, as well as rhythm, which has been objected to in this manner, that the auricle is so thin and dilated as to be incapable of any effective contraction, and the objectors substitute the theory of elastic recoil of the auricle, forgetting that under such circumstances there can be no recollection of the auricle, and under no circumstances can the auricle be compared to the sorta or to an overstretched india-rubber ball. The atrium may be so thin and dilated as to be incapable of any forcible contraction, but it is very rare for this to happen with the auricular appendix, and the current induced by its contraction may be sufficient to give rise to a murmur. When the muscular contraction of the auricle is so weak that it can only produce a faint sound or booming first sound unless the valve be so contracted as to form a septum. The character of the first sound at the apex affords valuable evidence as to the condition of the auricles.

In the evolution of mitral stenosis any excess of calcium ions in the blood plays a very important part. The so-called "good healing flesh," simple means that there is an excess of calcium cement in the blood and tissues. Under certain circumstances this may be very beneficial, but whenever there is any tendency to sclerotic changes in the body, Nature's efforts at repair are apt to be far in excess of the requirements. In the case of mitral stenosis, these excessive calcium ions may be the determining factors in the production of the disease. They raise the blood pressure, increase the force of contraction, give rise to hypertrophy of the muscular papillae, cause violent collision of the mitral cusps, increase the formation of the fibroid tissue, and gradually cement and unite the cusps together. I shall have more to say on this subject when we come to speak of prevention and treatment.

The suction of the ventricle is much aided by the high intraventricular blood pressure. Sir William Gairdner recognized and described the murmur as occurring at different periods, and even during the whole period between the systolic and diastolic events. The different varieties of murmur were named by the late Dr. Bronze, early diastolic, mid-diastolic, late diastolic, and entire diastolic. The late diastolic corresponds to the presystolic type. It is a deep, rumbling sound, which may be felt, and heard, in the aortic region. The early diastolic may be purely mitral in origin; but I am convinced that it is often confounded with a short, soft, blowing, diastolic murmur, which not infrequently occurs
in this disease at the moment of closure of the pulmonic valve, and which arises from regurgitation through the pulmonic orifice into the right ventricle owing to high pulmonic tension and dilatation of the pulmonary artery. This regurgitation is filling and blowing, and is known as the second sound. It is prolonged well into the long pause and nearly up to the first sound. The occurrence of the early diastolic and mid-diastolic mitral murmurs depends on high intrapulmonic blood pressure and the aspirating force of the left ventricle. The size of the orifice and the nature of the valve segments will largely influence the character of the murmur, as it occurs at a later stage and is not associated with the regurgitation of the left heart by being reached by at least a half, but when you are percussing out the heart the respiration is very quiet, and therefore it is a heart full of blood which is being aspirated during the diastolic period. The deep cardiac dullness by percussion, but, where the costal cartilages are fairly pilant, with a little practice you can palpate out the deep area with your eyes shut. The enlargement of the left auricle can be made out both by percussion and palpation, and is one of the commonest phenomena in obstructive mitral disease, but in some cases of associated mitral and tricuspid stenosis I have noted its absence. Here the comparatively small amount of blood passing through the narrowed tricuspid orifice was carried on without causing great distension of the left auricle.

The pulmonic second sound is accentuated. The intensity of this sound at the pulmonic orifice largely depends on the amount of lung intervening between the artery and the chest walls. Frequently the sound is better heard over the conus arteriosus or right ventricle, and if in this region there is a slight systolic murmur. Again, the murmur may be absent in the very late stage, when much can be done for the comfort of the patient, and when it is most important to diagnose between mitral stenosis and mitral incompetence. I think, therefore, we should endeavour to recognize the disease independently of any murmur.

Enlargement of the Left Auricle. One sign to which I attach considerable importance is an extension of the cardiac dullness in the direction of the left auricle. Under normal conditions a line drawn from the middle of the suprasternal notch to the nipple point on the left fourth costal cartilage where the nipple should be, and which for convenience I call the supra-sterno-mammary line, should mark the left outer and upper limit of the normal area of cardiac dulness. Any extension of the cardiac dulness beyond this line in the direction of the outer part of the infraclavicular region should raise a strong suspicion of mitral stenosis. This portion of the heart is usually well overlapped by lung, and so it requires very delicate percussion to elicit the extreme margin. I may here premise that the whole deep cardiac area should be carefully mapped out; we want to know the line and position of the whole heart and of its individual parts. The anterior surface is to a greater or lesser extent overlapped by lung, and so in every case the percussion note of the deep cardiac area is only relatively dull; and in order to get the first shade of dulness, we should, in my opinion, percuss very gently from without inwards. In this delicate percussion I think it is a mistake to interfere with the slight vibrations of the chest walls, and therefore I prefer an ivory pleximeter to the finger. When you wish to bring out the slight dullness or impaired note of the lung in the very early or hyperaemic stage of pneumonia, then the vibrations of the chest walls should be stopped by the whole left hand laid firmly on, and the middle finger percussion. In this way you avoid the confusion arising from the resonance of the vibrating chest walls, and you obtain the full amplitude of the resistance to the stroke. In the case of the heart, however, it is not a question of resistance or even dullness, but of variation in the percussion note. It is a very good exercise to percuss out the cardiac dulness in the cadaver and compare that with the actual area occupied by the heart. The superficial cardiac dullness gives you some information about the position of the anterior margin of the left lung, but, so far as the heart is concerned, it is of no value to yourself. The cardiac dulness is usually given much too small in standard textbooks; if you compare these pictures with the areas occupied by the heart in the post-mortem and under the zapper, you will be struck with the difference. Turn the lungs aside and you will find the anterior surface of a normal heart is as large as many illustrations of extreme hypertrophy. In none of Sansom’s cases of mitral or aortic incompetence is there any extension of the cardiac dulness beyond what I have termed supra-sterno- (or simply sterno-) mammary line. This, I believe, is not because it was not there, but because it was not mapped out. This careful demonstration of the cardiac dullness has yielded valuable results in my hands, and I hope that others will find it worthy of accurate observation. One authority on the heart, who does not believe in tripping, free tripping and acute mapping of the deep cardiac dulness, says that when he sees anyone attempting to do so he inquires if the percusser is trying to map out the heart in systole or in diastole. No doubt...
Its absence, therefore, may be of more grave significance than its presence. We should therefore not view it as a certain sign of mitral stenosis, nor fail to recognize that lesion when that sign is wanting.

The double second sound is frequently more marked over the right ventricle than at either the pulmonic or aortic cartilages has been urged against the theory of its aysynchronous origin. But the site of the greatest intensity of the normal second sound is a variable; in at least 50 per cent. it is also loudest over the right ventricle. If the pulmonary artery is uncovered by lung, even the normal second sound appears very accentuated. The pressure of the right ventricle may be felt through the base line, but a good padding of elastic lung tissue interferes very materially with the conduction of the cardiac sounds. The mitral first sound may be well conducted to the parties through the impulse, even when the point of impact has been entirely formed by the right ventricle; but then the second sound occurring at the beginning of diastole is apt to be absent at this point owing to the impact of the dilated right ventricle not being well sustained against the chest wall. The facts of each individual case should be examined in the light of the conditions present, and not by the theoretical considerations of the examiner. The sound, whether single or double, is best heard at the point of the chest through which it is best conducted by some solid media. Frequently, when the chest is a differential stethoscope at the aortic and the other at the second sound, a double second sound can be heard when only a single sound is heard at either point.

The double first sound is not an infrequent, nay, it is a common, event in mitral stenosis. It is not put down as a sign in our textbooks, nevertheless it is a fact. It is very difficult to account for, as it must have been heard hundreds of times by every experienced auscultator. The only explanation which I can offer of this want of recognition of a matter of daily occurrence is the fact that this doubling of the first sound in mitral stenosis is often accompanied by an or tumbling action of the heart, which renders the sounds very complex and difficult to analyse. When the heart is working steadily there may be slight dissociation of the tricuspid and mitral first sounds, which is appreciated by the ear, but there may be not complete separation of the two elements. In these cases the right ventricle invariably initiates the contraction, and the tricuspid element comes first, but before it is completed the mitral element is heard. This quasi-doubling, or in many cases true doubling, may be followed by a single or a double second sound. Frequently the right ventricle, which is better supplied with blood, gets so far ahead that it starts contracting before the left is half filled, and so you get a tumultuous or rolling action of the heart. There is a strong pulsation felt over the right ventricle, and this is accompanied by a loud, clear, tricuspid sound. In this way you are propagated to the left ventricle, and this is accompanied by a short, sharp, mitral first sound. If this occur very soon after the preceding effective left ventricular systole, when there is very little blood in the left ventricle, the aortic semilunar valves may not be opened, and in this way you may have twice as many cardiac contractions as pulsations at the wrist. When there is some blood in the left ventricle the semilunar valves may be opened, and you may even have five or six of these small systoles shown in a sphygmogram before the lever reaches the base line. What, then, is the cause of this quasi-doubling of the first sound? The heart (or rather of its side) may be so powerful as to shake the precordium? It is, in my opinion, undoubtedly due to the overloaded right heart trying to get rid of its congestion. The left ventricle, while the left ventricle is itself.
abortive pulsations in the down stroke. This goes on till the right ventricle has disposed of its superfluous charge of blood, and then the rhythm may become quite regular until the second sound is heard. This over-distension of the right side, with initiation of contraction in advance of the left, in some cases takes place in a regular rhythm, so that we get the so-called pulsa bigeminus, trigeminus.

On auscultation of the heart this asynchronism at the commencement of ventricular systole may be so slight that the mitral first sound becomes merged in that of the preceding aortic sound, and thus we only get as indication the first sound; or the asynchronism may be so great that we get a regular tumbling action of the heart, and there may be two first sounds with one or two second sounds for a considerable interval as I have been explained. This peculiar action is most easily observed when the cycles are infrequent.

The sequence of events may be briefly described as follows: Starting with a forcible contraction of the left ventricle, which is evidenced by a strong apex impulse, loud mitral first sound, strong pulse, and well-marked aortic second sound, this full discharge will fill the contracted arterial tree, keeps the arteries full between the beats, and produces a very gradual down stroke in the sphygmmogram. The contraction of the right ventricle which accompanies this complete systole of the left may only partially empty its distended cavity; its first and second sounds are synchronous with those of the left side, but less pronounced. The partially emptied right ventricle is then recharged, initiates contraction which is propagated to the left ventricle, while the latter is only partially filled. In this case the systole of the right ventricle is more powerful, its impulse stronger, and sounds louder than those of the left. This may only occur as an occasional irregularity, or, on the other hand, the right systole may keep in advance of the left for several cardiac cycles, and you may have an equal number of so-called extra-systoles, recorded in the sphygmmogram before the lever reaches the base line. What is felt in the pulse as an intermission, or an irregularity in force and rhythm, or is recorded in the sphygmmogram as one or more abortive pulsations, is represented on auscultation by an irregular tumbbling action of the heart, or a loud thump followed by one or more irregular rolling motions. When the mitral orifice is very much contracted, you may have a tricuspid first sound, then a mitral first sound, followed by a single second sound; or this may be resolved into aortic and pulmonic sounds, for one carotid pulsation. This peculiar rhythm of the heart is always associated with irregularity of the pulse, as increased by the lime salts, digitalis and caffeine, and is lessened by decalciyfying agents, by nitroglycerin, and atropine. It is also relieved by such agents as lessen the venous congestion.

Although this peculiar action is very pronounced in mitral stenosis, it is by no means limited to that disease. It may arise when the innervation and balance of blood between the two sides is deranged from any cause. It is frequently associated with dilatation of the right side of the heart, as occurs in emphysema, with fatty degeneration of the heart, and atheroma of the coronary arteries. It is not uncommon in the infiltrated or degenerated fatty hearts of elderly people affected with an acute blood poison, such as erysipelas, septicaemia, or diphtheria. It may occur in goit, either acute or latent, but, as a rule, in these cases there is usually a true intermission; the whole heart comes to a standstill for one cycle. In a case of Stokes-Adams disease, which I published in the British Medical Journal in 1876 when the patient took a very deep breath and held his chest expanded, the frequency of the contractions of the right side of the heart was increased, but after a couple of beats the left ventricle came to a standstill and an apneic or aseptic seizure was induced in the large intrapulmonary vascular cavity was repelled. In many cases the peculiar rhythm arises from the fact that the right side of the heart is usually more susceptible to alteration than the left.

It is not sufficiently recognized that the heart is not a single organ, but two organs—each of which has got its own definite work to perform—linked together for mutual assistance in cases of stress or difficulty.

All parts of the heart can be excited to contraction by various stimuli, except during the refractory period; but the right auricle is the most sensitive part of the heart, and responds to the slightest irritation. This over-distension of the right auricle has become quiescent in death. The two auricles are intimately linked together by a community of muscular fibre, and, although the wave of contraction may, and I presume usually does, commence in the right auricle, it is quickly propagated to the left, and consequently their contractions are simultaneous. When the auricles have driven a sufficient amount of blood into the ventricles to raise the elastic pressure to contraction, the left auricle is driven to systole begins, and it is rapidly propagated over both ventricles. The right ventricle is usually more sensitive than the left, or is better supplied with blood, as when you get doubling of the first sound as I have been explained. This peculiar action is most easily observed when the cycles are infrequent.

It is often slower in completing its work than the more powerful left ventricle, because when you get doubling of the second sound the final element is frequently pulmonic. In Miller's experiment and in cases of pulmon paroxysm the systole of the left ventricle may be delayed or abolished for one or more beats, owing to defect in the diastolic tension. All parts of the heart act rhythmically; but even in perfect health the rhythm is constantly liable to considerable variation which are readily detected if you will take the trouble to measure the various periods of activity and rest, not in fifths of a second, but in fifths or hundredths of a second.

The length of diastole depends, not on the amount of blood to be driven, the power of the ventricle to drive it, and the resistance in front. If, then, the commencement of systole depends on distension, and its length on varying factors, it would be very strange if the two sides of the heart were incapable of acting to some extent independently. When the balance of blood pressure in the two sides of the heart is disturbed, it would be a very extraordinary provision in nature if one ventricle had to wait on the other, or prolong its contraction, not in accordance with the work it had got to do, but in accordance with the necessity of its fellow. Although in mitral stenosis the right ventricle invariably initiates the contraction, yet in many other conditions the left ventricle acts in advance.

[Sir James Barr exhibited numerous tracings showing extra-systoles in mitral stenosis and other cardiac affections, and also the effects of respiration on the circulation.]

These views on the myogenic function of the heart, and on the production resulting of the systole in the two sides, are generally accepted in the present day. If you wish to get up-to-date information on the subject, I can strongly recommend you to Dr. James Mackenzie and Professor Wenckebach. These are very able and very useful observers, whose writings will repay perusal whether you agree with them or not. Their facts are, as facts always are, extremely valuable, but I do not wish you to accept the superstructure which they have reared. The heart is a subject that has been subjected to some rather than without due inquiry. They have already too many disciples who have swallowed everything, and often belch forth the work as if it were their own, without, in my opinion, due acknowledgement. One recent writer even produces Wenckebach's trotting horse to illustrate the myogenic function of the heart, and the rider to represent the control of the nervous system. It is really too bad of Wenckebach to go us such crude skeletons when his disciples trot them out it becomes rather more than a joke. From the physiological side there have been numerous writers, but personally I prefer the work of Gaskell, because he makes it his business to draw conclusions which are not warranted by his facts. The myogenic function as given by Wenckebach and Mackenzie may be briefly stated as follows:

The muscle cells of the cardiac muscle possess not only the properties of excitability and contractility which they have in common with voluntary muscles, but also those of stimulus production and stimulus conduction.

All the muscle cells of the heart are supposed to possess the property or function of manufacturing a stimulus to contraction, but this function exists in a special degree at the junction of the great veins with the auricle, or at a point which has been described and designated by Keith
as the sino-auricular node. As this stimulus, whatever it may be, is most readily produced and in greatest quantity at the sinus or sino-auricular node, the time of cardiac contraction is set there. From this point the stimulus is conducted forward in both its wake there and through the muscular contraction. From the auricle it is conducted along the bundle of His to the ventricles, and here the propagation is slower. According to Mackenzie the e-c interval, which includes the contraction of the auricle, the propagation of the stimulus along the auriculo-ventricular bundle, and the pre-systolic period of the ventricular contraction, occupies in normal hearts about one-fifth of a second. Other tissue possessed of a pseudo-myogenic” 

"The necessity for this theory seems to have arisen in the brains of certain individuals from the fact that the hearts of the tortoise, frog, etc., go on beating in a rhythmical manner when removed from the body, and quite free from all control by what has been termed the more highly specialized function of the mammalian heart. The heart of the tortoise possesses the function of rhythmic contraction in a very high degree, and all muscles which are employed in some extent to some extent be, and can be made to contract by any stimulus, mechanical, chemical, or electrical, but the mammalian heart will only continue to beat rhythmically under the stimulus of a certain diastolic tension in its coronary vessels. The right side of the heart is more excitable than the left, and responds to a lower pressure.

The popular myogenic theory also presupposes a regular rhythmic discharge of stimulus material, hence in a normal heart there should be perfect synchronism between the two sides, and the pulse periods should be equal, whereas we know that in a healthy heart there is a doubling of both sounds and great variations in the systolic and diastolic periods with every deep respiration. This theory tends nowhere but to the grave, whereas if you believe that the heart is merely the mechanism by which the diastolic tension, you can often save lives by putting your theory into practice.

Many deaths from syncope could be averted by simply hanging the individual by the feet. The large, largely known in chloroform syncope, but it should be firmly inculcated in every ambulance course. Some of my earliest experiences in this direction were conducted before there was any Vivisection Act. After the apparent death of a dog from chloroform, he was resuscitated by hanging him up by the hind legs. In this way you can quickly raise the tension in the right side of the heart, and when it starts to work the left is very likely to follow suit.

The Character of the First Sound in Mitral Stenosis.

The first sound is unusually loud, clear, sharp, abrupt, and banging or thumping. It may be a thud, resembling the accentuated pulmonic second sound, only nearly twice its length. Certain writers, thinking that the mitral valve is not in a condition to generate sound, have added some difference in excitation potential, we might form some conception of its nature and action.

Regarding the propagation of the stimulus there is no evidence, but it is assumed that it does travel and that the rate of propagation of the contraction wave in the heart is so fast, reaching 5 metres a second or more, that, for all practical purposes, we may say that the contraction of the whole ventricle is synchronous.

Dr. Mackenzie holds that when the ventricle takes on its character of rhythm, extra-systoles are fewer than those arising from a propagation of the sinus or auricular rhythm, hence on some occasions the auricles and ventricles may be contracting simultaneously. It is also held that when an e-c stimulus enters the auricle, it renders the muscle refractory and incapable of responding to the ordinary physiological stimulus when it arrives from the sino-auricular node. Consequently we go on, frequently, to pass the a-systole, or extra-systole, and after this long rest the ventricle responds to the second physiological stimulus, and we then get a large compensatory beat. As this physiological stimulus is supported, and the extra-systole up to the compensatory systole should be equivalent to two pulse periods, but all the observers hitherto have never been able to stretch the time out so long, hence it is assumed by way of explanation that during the long rest the muscle cells become charged with stimulus material, and their excitability is increased, and so the muscular contraction occurs a little too soon. In many cases you may have a good many extra-systoles between the physiological beats, and then the muscle is quite ready for another. The whole of the muscle is in a state of rest, and there is no excitation. This theory is very nice and plausible, and deserves to be true, but like a good many other theories, it has not been considered necessary to establish the truth of the premises; not one scintilla of evidence has ever been adduced to show the muscle cells, or any node of the bundle, generate any stimulus which excites contraction, and there is no information as to the nature of the stimulus. I said in 1906, and I say still, that the so-called myogenic stimulus, or the production of a stimulus of contraction as a function of the cardiac muscle is, so far as we can infer, purely an inference, perhaps, an influence which exists in the brains of certain writers rather than in the hearts themselves. Of course there may be a stimulus, but before believing in it I should like to know what it is. If it were a salt of potassium, or sodium, or calcium, or any other substance, or some difference in excitation potential, we might form some conception of its nature and action.

Regarding the propagation of the stimulus there is no evidence, but it is assumed that it does travel and that the rate of propagation of the contraction wave in the heart is so fast, reaching 5 metres a second or more, that, for all practical purposes, we may say that the contraction of the whole ventricle is synchronous.
clear, short, and sharp element of tricuspid origin is very different from the loud booming first sound heard during the quiet action of the same heart. The character of the murmur is also altered to evidence as to the nature of the segments and the orifice. When the orifice is rough and thick, and the auricular contraction vigorous, you may get a rough purring presystolic thrill felt at the apex.

The Pulse.

The pulse is very variable. When the compensation of the right ventricle is well maintained, then the pulse is increased in volume and is full and firm, and fairly regular. When the mitral orifice is very much contracted it becomes very small, infrequent, weak, and irregular, both in force and rhythm, with numerous interpolated systoles. When the right ventricle is much dilated, falling in power and force, regurgitation through the tricuspid orifice, then the pulse becomes frequent, quick, small, weak and very irregular in force and rhythm. Interpolated pulsations in the downstroke of the sphygmogram are present in all cases of marked stenosis. These pulsations represent systoles and never occur before the aortic notch in the primary or subsequent pulsations. The left ventricle contracts on a small quantity of blood which may be barely sufficient to raise the aortic valve, thus producing a very abortive pulsation in the down stroke, or it may be enough to raise the lesser part of the sphygmogram on the normal height to maintain the ordinary cerebrum in the arteries. You may have even five or six of these pulsations recorded in the sphygmogram before the lever reaches the base line. At other times the beats may be perfectly uniform and regular for a long period.

Condition of the Lungs.

A result of the chronic overloading, with heightened pressure in the pulmonary circuit, we get increased cell proliferation and diapedesis of the blood corpuscles, giving rise to the condition termed brown induration. In some severe cases the lung tissue becomes quite tough and leathery.

There is also bronchial catarrh, and not infrequently haemoptysis. These lung changes give rise eventually to complicated emphysema. The lung symptoms frequently become so prominent as to obtrude themselves on the attention, and obscure the underlying and primary mitral affection.

Congestion and Collapse of Pulmonic Lobules.

In the majority of cases of mitral stenosis, if you carefully percuss the back of the chest, especially after the patient has been in the dorsal decubitus, you will find small, circular, and oval areas from about the size of a shilling to that of a crown-piece, of comparative dullness. Over these areas the vesicular murmur is doubled, and the sounds are rather sharp and tubular, and frequently inspiration is accompanied by a few crepitant rales, and the heart sounds are often clearly conveyed. These little areas of dullness arise from congestion of the alveolar tracts of the pulmonary vessels, and partial collapse of the air cells; the blood is retained a long time in the pulmonic vessels, some of the airspaces are not required, and the air cells are encroached on by the overloaded intrapulmonary vascular cavity. The dullness is never absolute, and the areas are apt to vary from day to day, some clearing up, and others becoming established; they clear up when the patient is moving about, taking deep inspirations, and when friction has been applied to the back. This sign I discovered over thirty years ago, and it is one to which I attach considerable importance.

The percussion note is only relatively dull, and it is best elicited by deep percussion near the pleximeter; there should be no interference with the vibration of the chest walls.

The Liver and Spleen.

The liver is passively congested, especially when there is failure of the right ventricle, and the whole venous system is engorged. The hepatic dullness may extend below the level of the umbilicus, but the spleen is not usually much enlarged.

The Veins of the Neck.

There is frequently visible pulsation in the veins of the neck, especially when the patient is recumbent, and this may be either auricular or ventricular in rhythm, or both. In tricuspid stenosis the tension in the veins of the neck is much higher than in mitral stenosis alone. In tricuspid stenosis the veins of the heart are much dilated, and the tension in the substance of the ventricles become like large sinuuses.

Droops.

Droops does not occur until there is failure of the right ventricle, and, unlike that in mitral regurgitation, it frequently begins as an ascites. When it starts in the lower extremities it is due to failure in the left ventricle with fall in the arterial blood pressure and proximation to any venous engorgement. Patients suffering from tricuspid stenosis are particularly liable to hydrothorax when droops set in, owing to the obstruction to the vena azygos.

The Size of the Orifice.

I am in the habit of weighing all the factors in each case, and then mentally estimating the size of the orifice. The evolution of mitral stenosis; they raise the level near the mark. The estimated measurement which I generally employ are the size of a lead pencil, the tip of my little finger, my little, ring, middle or fore finger, my thumb, or two fingers.

Thrombosis and Embolism.

Thrombi are not infrequently formed in the left atrium, and are readily carried to the brain, spleen, or kidneys. The left middle cerebral is a site of a plug, and then we get aphasia and right hemiplegia. The usually soft and unorganized nature of the embolus renders treatment very successful. All lime, salts should be removed from the diet, decalcifying agents and strong ammonia freely used.

The Lime Salts.

The calcium salts are usually in excess in the blood and in the muscular tissue, largely owing to milk forming a staple article of diet. It is unfortunate that the ideas of a large number of medical men as to suitable diets for invalids should be restricted to milk and the numerous abominable milk foods found on the market. In fact, some men's ideas are so fixed that you might almost think they were set in plaster of Paris. For the brains of such I would like to prescribe a long course of decalcification. The calcium ions, in my opinion, largely contribute to the evolution of mitral stenosis; they raise the level near the mark by increasing the tone and contraction of the smaller arteries, and by augmenting the viscosity of the blood. At one time I thought the heightened arterial blood pressure was due to arteriolar contraction, but I am now quite willing to give a fair share of the increase to the viscosity of the blood, as I find that the arterial blood pressure can be rapidly and permanently lowered by the use of decalcifying agents. As the left ventricle has to do more work and has more muscular tissue than the right, it gets a lesser supply of lime; its muscular tissue and the musculi papillares get hypertrophied. The violent tension of the mitral cusps leads to calcification, and the great supply of calcium ions contribute in no small measure to the formation of fibrous tissue, and the cementing together of the edges of the valve. When patches of atheroma have taken place anywhere, the abundance of lime salts are readily deposited in the form of calcium soaps. Dr. Blair Bell has shown "that ordinary fibrous, especially scar tissue, is relatively exceedingly rich in calcium salts."

The calcium ions increase the force of the muscular contraction but diminish muscular irritability; hence the left ventricle often becomes much less responsive than the right, and in this way the calcium ions contribute to the production of the peculiar cardiac rhythm which I have described. The conclusions of Loeb as to the effect of various ions in muscular contraction are given by Wageneckbach as follows: 1. There are certain ions—for example, Na, Cl, Li, Br, I, and others—which (in solution under a pressure of 4.9 atmospheres) are capable of producing rhythmic contractions in muscles. These ions do not produce such contractions by increasing excitability, because (1) the contractions continue when the irritability is diminished; and (2) they do not occur in solutions of non-conductors (for example, glycine, sugar) at the same osmotic pressure, even when the excitability is normal. It is more natural to think that these contractions are due to definite chemical combinations which these ions form in the muscle.
2. There are ions which inhibit the rhythmic contraction of normal muscle fibres—for example, Ca, K, Mg, Bi, Ba, Co, Mn. The results of those between the large pulegeones of the various ions mentioned here, does not depend on a reduction of the excita-

bility. For on adding a little CaCl2 to some physiological salt solution, its rhythmic contractions are depressed by the excitability of the muscle is less impaired, and lasts longer than it does in physiological solution, when their rhythmic contractions do not occur. It is more natural to think that the chemical combina-

tion which the ions Ca and K form in the muscle renders its rhythmic contractions more difficult to produce.

3. Hydroxyl or hydrogen ions accelerate the production of rhythmic contractions when subjected in sufficient dilution to solutions of the ions that excite contractions, as enumerated in 1. But when they are put into solutions of non-

contractile ions, they have the same effect. They have, therefore, a catalytic action in the production of rhythmic contractions, but are unable of themselves to produce rhythmic contractions.

4. From the very scanty material that is yet available it seems that only ions, and not non-conductors, have the power of setting up rhythmical contractions in voluntary muscle.

5. The laws regulating the periodic action of the ventricle appear to be similar to those that have been given here for striated muscle.

In all experiments that have been described here it is the muscle substance itself that sets up this rhythmic action, since periodic contractions cannot be started from either the nervous or spinal cord through the solutions mentioned in 1.

The human heart requires very much less pressure than that stated by Loeb for involuntary muscle to maintain its rhythmic contraction. The ordinary atmospheric pressure or less is sufficient for the right auricle, a little more for the left auricle, a little more for the ventricle, a little more still for the left auricle, and often considerably more for the left ventricle. Large hypertrophied and degenerate hearts require high diastolic pressure to maintain their excitation and motion in such cases it is not wise to attempt to reduce the arterial blood pressure very low, because syncope does occur it is very difficult to get sufficient pressure up to the heart anyway.

The amount of calcium and of potassium, stated by Loeb to be inhibitory agents, are scarcely comparable, as the former increases the force of the muscular contraction and delays the intermediate; while the latter has a paralyzing effect on contraction, but hastens dilatation. Sydney Ringer long ago showed that the salts of calcium and potassium were antagonistic both in physiological and toxic doses, but in this antagonism their actions were complementary in maintaining the rhythmic contractions of the heart. I reproduce some of Blair Bell’s tracings of the effects of calcium chloride on the circulation of the blood. The first tracing (Fig. 1) shows the rapid effects of a lethal dose, the heart remaining firmly contracted in systole.

The following is a very interesting case of dissociation of the two sides of the heart, or what is called hemisystole. This woman had fallen into the hands of the Filipinos, I mean the surgeons, and had the following operations—not all at once, but at least on had an appendectomy by four different surgeons in the last fifteen years: Curéted two or three times, both ovaries and tubes removed, several uterine fibroids, the appendix, hysterectomy, and adhesions from previous operations too numerous to count. It would seem that the only thing of importance which was not removed was the pain from which she suffered, and for which in vain she sought relief. She might have appropriately said in the language of Byron:

Count o’er thy days from anguish free,
And know, whatever thou hast been,
Came better not to be.

M. L., aged 41, admitted to the Royal Infirmary under my care August 6th, 1908, complained of right abdominal pain, pain in right shoulder, lower abdomen. Her previous history has been briefly recorded above. These symptoms have been present for seven years. At first she had a very bad pain near the right side of her abdomen, which was relieved by the administration of soothing bitters, and which lasted for about three days, and was not relieved by anything. The pain became worse, and was now accompanied by nausea, vomiting, and diarrhoea.

The condition of the patient was very bad. She had great tenderness over the abdomen, and the peritoneum was much increased. The heart’s action was feeble, and the pulse was 120. The blood pressure was 210/110. The patient was given narcotics, and the symptoms gradually improved.

The pulse was 100, and the respirations 20. The patient was given digitalis, and the condition improved. The patient was discharged on the 12th of October, 1908.

The post-mortem examination was made by Dr. E. E. Glynn twenty-four hours later. The following is abstracted from his notes:

Lungs: A few old scars and adhesions at the apices, but no active tuberculosis. Heart: 14 oz., aortical normal. Right ventricle slightly hypertrophied and tended to dilate. Left ventricle hypertrophied and cavity smaller than right; musculi papillares thick, mitral orifice admits two fingers. Calcification of aorta and coronary arteries. The veins were congested, the size of a pea in the posterior cusp. Aortic valves normal. Coronary arteries normal. The pericardium was thickened, over with small white nodules, apparently lymphoid. Kidneys cyanosed, but otherwise normal. The section of the left ventricle shows intima thickened, with a few patches of intima and several small spots of intima and several small spots of intima. A section of the heart muscle stained with acid fuchsin showed no evidence of fatty degeneration.

Dr. Pauliland Hick to-day found no changes in the mouths of the right and left ventricles respectively, and the heart weighed 124 oz. of deci-normal hydrochloric acid. He then examined the stomach according to Blair Bell’s method and found that the gas from the right ventricle gave an index of 0.3 and that from the left 0.7. Even after the autopsy it is difficult to explain why.
Fig. 1.—Effect of calcium chloride (1 c.c.m. of a solution of 0.5 gr. in 1 c.c.m.) upon the heart.

Fig. 2.—Effect of CaCl₂ (0.5 gr. in 1 c.c.m.) upon the heart. First injection, of 0.05 c.c.m.

Fig. 3.—Same solution, second injection, of 0.18 c.c.m.

Fig. 4.—Same solution, third injection, of 0.6 c.c.m.

Fig. 5.—Same solution, fourth injection, of 0.9 c.c.m.

Fig. 6.—Fourth injection (continued).

Fig. 7.—Fourth injection (completed).

The above tracings were taken with a cannula in the carotid artery of the rabbit, and the injections were made into the femoral vein.
the heart should have suddenly stopped in diastole, but it is
interesting to note the greater calcium content in the left
ventricle than in the right.
[The treatment of mitral stenosis was also dealt with, but
as this address was very long Sir James Barr has reserved
that portion of it for publication on a future occasion.]

A Lecture
ON THE
OCCURRENCE OF HEART-BLOCK IN MAN.
AND ITS CAUSATION.
DELIVERED AT THE LONDON HOSPITAL MEDICAL SCHOOL
ON NOVEMBER 13TH, 1906.
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In the opening remarks it was assumed that the audience
was aware of the evidence for the electric theory in the
lower and cold-blooded vertebrates, that the heart beats
have their origin near the mouths of the great veins and
that the contraction passes as a conducted wave from sinus
to atricle and auricle to ventricle.

We may proceed at the present time to a consideration of
certain general propositions which have an intimate
bearing upon clinical work, and to a discussion of the
experimental and clinical evidence which upon which the modern
view is based, that in mammals the auriculo-ventricular
bundle is the path of impulse conduction between auricle and
ventricle. It will be obvious that, in demonstrating that the
auriculo-ventricular bundle is the means of stimulus transmission, we shall not only prove that this
bundle, already shown to be morphologically identical with the
structures of the myogenic theory as a whole,

THE EVIDENCE FOR CONDUCTION BY MUSCULAR CONTINUITY
IN MAMMALS.

The fact that dissociation of the usual co-ordinate
sequence of contraction in the chambers of the mammalian
heart can be understood in many centuries, though the cause of it remained for a long while obscure.
Harvey, in his essay on the movements of the heart,
stated that he observed the prolongation of the interval between the auricular and ventricular
beats of the dying heart, and noted an occasional ventricular
response following upon several auricular contractions
under similar circumstances.

Two years after the publication in which His described
the auriculo-ventricular bundle, the same observer
attempted, in conjunction with Graunfield, to destroy the
connection between auricle and ventricle in the heart of a
rabbit, and apparently with success. In a preliminary
communication he records the fact that complete dissociation
of the auricular and ventricular rhythm was produced,
though neither detail nor tracing is given.

In 1894 Humbert made further observations. He
operated on the heart in situ, opening the right auricle,
injuring the region of the bundle, and then reclosing
the wound. The experiments were performed on the dog and
yielded no satisfactory results. The method was discarded
for that of perfusion. Using Locke’s fluid and perfusing the
dog’s heart, he recorded the fact that cuts or cauter
wounds in the neighbourhood of the bundle have no
effect, but that when the bundle itself is interfered with
an arrhythmia is produced. In the single tracing which
is given complete dissociation appears to be demonstrated.

The first important series of experiments were performed
more than ten years later by Hering upon the perfused
dog’s heart. He showed that sections traversing the

bundle produced complete heart-block, and illustrated his
communication by excellent tracings. He further found
that no other incisions would produce the result. Almost simultaneously Erlanger published his first observations. In the first instance, an attempt was made to
pass a ligature through the heart walls so as to include the
bundle. In seven experiments performed, heart-block was
obtained on one occasion only. An auriculo-ventricular
clamp was then tried, a method similar to that employed
by Gaskell for the cold-blooded heart. In seven experi-
ments heart-block was obtained on two occasions. The
third method introduced yielded results of far greater
value, and was finally adopted permanently owing to the
uncertainty and unsatisfactory nature of the earlier
attempts.

Erlanger devised a special clamp, one blade of which
was introduced into the left ventricle and driven under-
neath the bundle as it lay on the septum. The clamp
could be introduced and tightened without interfering with
the remaining heart muscle at any extent.

The results obtained in this way have been very striking,
and are in the main a complete confirmation of the similar
clamps experiments of Gaskell. The following is an abstract
of Erlanger’s conclusions:

By compression of this bundle all stages of heart block may be
obtained. These include:
(1) An increase of the inter-ventricular pause.
(2) An occasional auricular silence.
(3) Regularly occurring auricular silences—for example, one
silence in ten, nine, eight, seven, six, five, four, three, and two
auricular beats.
(4) A 2:1 rhythm.
(5) A 3:1 rhythm.
(6) Complete heart-block.

The compression of the bundle was subsequently con-
firmed in Erlanger’s experiments by careful autopsies and
histological examinations carried out by Retzer.

The protocols of the seventeen experiments given in the first
communication show that a successful result was obtained
in sixteen cases, and that these were the experiments in
which the bundle had been damaged. The work has been
extended and amplified by the same writer in conjunction
with Hirschfelder in researches in which operations of a
similar nature were performed with similar results. Erlanger,†
in conjunction with Blackman and Cullen, has also been
to damage the bundle in seven dogs, the animals surviving the operation for a varying time, and
presenting various grades of partial block or permanent
and complete block. The experiments are of importance
in that they demonstrate the permanence of the result,
and suggest the absence of other paths of conduction,
which might only come gradually (Erlanger).
They also show the possibility of producing syncopæ
t attacks and epileptiform seizures experimentally by this
means. Heart-block has been similarly produced by
damage to the bundle by Tabor. Biggs has recently
produced it in the rabbit by incisions.

But at we justified in concluding that the auriculo-
ventricular bundle is the sole path of conduction in all
species of mammals, and in all individuals of such species?
It appears that we must regard the bundle as one of
several remains of the primitive tissue of which the
embryonic heart is composed, and that during the phy-
logenetic, if not during the ontogenetic, development of the
heart the connexion between auricles and ventricles was
at one time around the whole ring. The amphibian and
terrestrial and reptilian heart still shows such a junction. Now, it would be a very surprising fact, if such a fact should be eventu-
ally demonstrated, that complete segregation of
ners has occurred in mammalia, and that such segr-