

Echocardiography The Scientific Basis of Medicine

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<Opening titles>

< Gibson to camera>

Although it was introduced only 25 years ago, the technique of echocardiography has now become almost indispensable in the assessment of patients with heart disease. One would as soon, nowadays, do without the echocardiogram as one would without the chest x-ray or the ECG. The object of the technique is to detect boundaries or interfaces [...]

<Gibson narrates over animated illustrative diagram, uses indication stick>

[...] between media which have different physical properties. Such a boundary is shown diagrammatically here. It might, for example, be the boundary between a valve and blood or between blood and myocardium. A transducer is placed on the anterior wall of the chest which transmits ultrasound at a frequency of 2 $\frac{1}{2}$ megahertz. A thousand pulses a second are transmitted, each pulse lasting a micro-



second. The ultrasound then passes through the first medium, obviously at a velocity dependent on the velocity of sound of that medium.

On reaching the interface, a portion of the ultrasound is reflected, the greater part passing on through it. The difference between blood and myocardium, for instance, is small, when considered in terms of velocity of sound or density and therefore only very small amounts of ultrasound are reflected at such interfaces. However, a small amount returns to the transducer where it can be detected and shown and displayed on a cathode ray tube, shown diagrammatically at the top. The distance from left to right of the returning impulse represents the time taken to pass through the first medium and clearly if one knows the velocity of sound, one can calibrate this as distance. The height of the impulse represents the intensity of the returning echo. In this particular slide, it's shown diagrammatically as height but in the echocardiograms we shall subsequently be looking at, it shall be shown as the intensity of the spot.

<Gibson narrates over illustration of a dissected heart, uses indication stick>

This sums up the anatomy of the heart and represents a transverse slice through it. Here is the intra-ventricular septum with the left ventricular cavity behind it. Separating this from the left atrium shown here is this large structure which is the anterior cusp of the mitral valve. This here is the posterior cusp of the mitral valve. Forward from the plane of the picture, is the aortic root. A transducer is placed on the anterior chest wall, just alongside the sternum – the reason for this position is that it is essential that ultrasound can pass to the heart without passing through lung since the alveoli and lung form a virtually impenetrable barrier to the passage of ultrasound.

So one can see that from this position the ultrasound beam is going to pass through the intra-ventricular septum, the left ventricular cavity, the anterior cusp of the mitral valve and behind that will be the left atrial cavity. If one aims the beam a little lower, one can pass across a dimension of the left ventricle, immediately below the left mitral valve in this region here, from the septum here to the posterior wall here.



Historically, the first structure to be studied in any detail was the anterior cusp of the mitral valve. And here is an echocardiogram showing the anterior cusp.

<Gibson narrates over echocardiogram of anterior cusp, uses indication stick>

In all of the echocardiograms I shall be showing, anterior represents the chest wall, depth into the patient is shown downwards. Time goes from left to right as is shown by the ECG. Depth into the patients is marked off here in centimetres and the distance between these arrays of dots represents time intervals of half of a second. Here is the echo from the ventricular septum, behind it as we saw previously, the left ventricular cavity and here is the echo from the anterior cusp of the mitral valve with the left atrial cavity behind it, and these dense echoes here come from the posterior mediastinal structures and the lung.

So if we start with the QRS complex, representing the start of systole there is a rapid downward or posterior movement of the anterior cusp, which is what one would expect as the valve moves posteriorly to its closed position. It's then carried forwards during ventricular systole as the left ventricular cavity size gets smaller as blood leaves it and enters the aorta. There is then a rapid, early diastolic opening movement, shown here. The valve, instead of remaining, however, in the open position, throughout diastole shows this very characteristic movement of mid-diastolic closure so that by this stage it's virtually back in the closed position. It then reopens here with atrial systole and closes again at the onset of the next ventricular systole.

This mid-diastolic closure of the mitral valve was predicted as long ago as 1836 but its mechanism is still not really understood. Vortices in the left ventricle have been invoked, deceleration of blood is also probably important and finally it reflects the fact that the normal anterior cusp is a very thin, diaphanous structure and so disturbances of any of these factors might be expected to interfere with the normal diastolic closure rate.

00:06:32:11



<Gibson narrates over a further echocardiogram, uses indication stick>

If one moves the transducer a little, it's possible to pick up echoes from the posterior cusp of the mitral valve, shown here, very close to the posterior wall of the left atrium. You see its motion is a mirror image of the anterior cusp shown here with rapid posterior movement early in diastole, again a mid-diastolic closure and reopening during atrial systole.

A second point is that if one records the diastolic movement of the valve at its efficiently fast time base, shown here, you'll notice that the diastolic closing velocity is not constant but gradually increases throughout the diastolic closing period.

So, that sums the main features of the normal mitral-diastolic closure. It's this part of the cardiac cycle which is particularly abnormal in patients with rheumatic mitral valve disease.

<Gibson narrates over a further echocardiogram, uses indication stick>

Here is the echocardiogram from such a patient. Again, these echoes here represent the intra-ventricular septum, here is the echo of the anterior cusp of the mitral valve with the left atrial cavity behind it. Notice that one gets the usual posterial movement during systole, the usual anterior opening movement shown here, but instead of the normal diastolic movement which would be in this direction, the valve is held in the open position throughout diastole until the next QRS complex when it closes. So the mitral-diastolic closing is very abnormal in rheumatic mitral valve disease.

A number of factors are involved – left ventricular filling is obviously slow if there's mitral stenosis and the normal vortices caused in the ventricle are not developed, and secondly the anterior cusp itself is thickened and held in the open position by a mitral-diastolic gradient across it. Notice in this particular slide, the cusp echo itself is of normal thickness and its overall amplitude and movement is normal. This is the sort of echocardiogram that comes from a patient who would do well with a closed mitral valvotomy.



<Gibson narrates over a further echocardiogram, uses indication stick>

This is from a patient with a disorganised and calcified mitral valve. Again, you see the anterior cusp here with a very much reduced diastolic closure rate but notice how thick the closure is here, apparently over a centimetre thick, showing these multiple layers which often go with the presence of calcification. So it's possible using the technique of echocardiography to not only get some idea of the presence of rheumatic mitral valve disease but also to get an indication of the anatomy and the sort of valve that the surgeon is likely to find at operation.

<Gibson narrates over a further echocardiogram with diagram, uses indication stick>

It's possible to quantify this disturbance of diastolic closure by measuring the diastolic closure rate shown diagrammatically here simply by constructing a tangent to the valve movement during diastole. One knows the speed with which the paper is going and therefore it's possible to calibrate this slope in terms of a closure rate, here 20mm per second.

<Gibson narrates over a further echocardiogram with diagram, uses indication stick>

After mitral valvotomy the diastolic closure rate often increases, in this particular patient to 50 mm a second. Measurement of the diastolic closure rate is quite a reliable way of following progress in individual patients with pure mitral stenosis following operation. But it's not a good way of quantifying the severity of mitral stenosis in a patient and we shall discuss later other ways in which this can be done using echocardiography.

00:10:38:00

<Gibson narrates over a further echocardiogram, uses indication stick>



Echocardiography can also be used to look at other abnormalities of the mitral valve. This is the mitral valve echo from a patient with ruptured chordae tendineae causing severe mitral regurgitation. Here is the anterior cusp echo, notice early diastolic opening movement, normal closure, normal reopening movement with atrial systole but instead of remaining in the closed position here during systole, there are multiple echoes stretching right back to touch the posterior left atrial wall. These appearances are very characteristic of ruptured chordae, causing severe mitral regurgitation, and in association with other studies of the left ventricular cavity, it enables this condition to be diagnosed and assessed non-invasively and patients referred to surgery without cardiac catheterisation.

<Gibson narrates over a further echocardiogram, uses indication stick>

The next echocardiogram shows another type of mitral valve abnormality. Again, diastole is normal with normal closure, normal reopening with atrial systole but you'll notice with ventricular systole, there is prolapse here of both anterior and posterior cusps from their usual systolic position which would be here. This syndrome is often accompanied by a mid-systolic click shown on the phonocardiogram here. And you notice that the timing of this mid-systolic click exactly corresponds to that of the prolapsed posterior cusp. Again, this is a diagnosis that can now be made quite specifically using echocardiography and catheterisation is no longer necessary.

00:09:57:10

<Gibson narrates over a further echocardiogram, uses indication stick>

This is another condition in which mitral valve movement may be very abnormal, hypertrophic cardiomyopathy. These mass of echoes here come from the enormously thickened interventricular septum. Here is the posterior wall of the left ventricle. The left ventricular cavity itself is small. This echo here is from the anterior cusp and this from the posterior cusp of the mitral valve. You see the normal opening movement, normal reopening with atrial systole, but during ventricular systole,



instead of them staying back in this region here as you would expect, the mitral valve is carried bodily forwards so it touches on the septum. This is described as the systolic anterior movement, or S.A.M., and is very characteristic of hypertrophic cardiomyopathy accompanied by a gradient between the left ventricular cavity and the aorta. It's this systolic anterior movement that's responsible for the systolic murmur. As you see the onset of the systolic murmur corresponds in timing to the onset of the systolic anterior movement – exactly the same time one gets the characteristic dip in the carotid pulse. Although such a systolic anterior movement such as this can occur rarely in other conditions, when accompanied by the features of a small left ventricular cavity and a very much increased septal thickness, these features make the diagnosis of hypertrophic cardiomyopathy virtually certain.

<Gibson narrates over a further echocardiogram, uses indication stick>

Mitral valve movement could also be abnormal in other conditions. This is the mitral valve echogram of a patient with severe aortic regurgitation. You notice that the overall movement of the cusp is normal, both in diastole and also in systole but superimposed on the echo is this fluttering movement. This reflects the fact that the normal anterior cusp is situated immediately below the aortic root and so it's exactly in the jet of blood coming back from the aortic root into the left ventricle during diastole. An echocardiogram like this is often found in a patient with a mid-diastolic murmur at the apex, the so-called Austin Flint murmur. If one has this echocardiogram, one can be quite certain that the patient does not have mitral stenosis and the mid-diastolic murmur is, in fact, an Austin Flint one. It may well be that the actual genesis of the mid-diastolic murmur is related in some way to this fluttering movement of the anterior cusp, although the exact relation between the two has not been definitely worked out. One sees an echocardiogram like this in a patient with chronic aortic regurgitation.

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<Gibson narrates over a further echocardiogram, uses indication stick>



If the aortic regurgitation is acute and severe, such as might result, for example from acute endocarditis of the aortic valve, one sees a rather different picture. This is the echocardiogram of a girl who developed acute staphylococcal endocarditis of the aortic valve during pregnancy. Here is the mitral valve echo and notice that one has a very early closure during diastole and no subsequent reopening. This is premature mitral valve closure and reflects the fact that on account of the severe aortic regurgitation, the left ventricular pressure rises rapidly above that in the left atrium and causes the mitral valve to close. These appearances are very characteristic of acute, severe, aortic regurgitation, suggesting that the patient is going to need early surgery.

<Gibson narrates over chest x-ray, uses indication stick>

It's quite possible to detect other abnormalities using echocardiography. This was a patient who presented with mitral stenosis. His chest x-ray, as you see, shows dilated pulmonary arteries, pulmonary vascular congestion and curly lines at the bases. Cardiac catheterisation showed a gradient between the left atrium and the left ventricle and it seemed quite clear cut. There was no diastolic murmur, but of course it's quite well recognised that mitral stenosis can often be silent particularly when there's severe pulmonary hypertension.

<Gibson narrates over a further echocardiogram, uses indication stick>

The next slide, however, shows the echocardiogram. And you notice the septum here, displaced posteriorly by the pulmonary hypertension, but much more important, the diastolic closure rate is totally normal. So one could be sure, before surgery, that whatever this patient had, they did not have rheumatic mitral stenosis. And the patient went to operation with a diagnosis of cor triatriatum; actually this proved to be wrong – it was a supra-valve diaphragm, above the mitral valve, separated from it only by a few millimetres and beneath this, when it was removed at the time of surgery, there was a totally normal mitral valve.

<Gibson narrates over a further echocardiogram, uses indication stick>



The next slide is another condition which could easily be confused with mitral stenosis. This was a patient who presented with a mid-diastolic murmur and systemic embolism. As you see the phonocardiogram here shows a loud first sound but instead of the usual mitral echocardiogram, one sees this mass of echoes behind the anterior cusp, coming forwards during diastole and going back into the left atrial cavity during systole. These appearances are very characteristic indeed of a left atrial myxoma. And again this is a diagnosis which can be made nowadays by echocardiography without submitting the patient to angiography.

<Gibson narrates over angiogram, uses indication stick>

The next slide is from an old angiogram from such a patient. Here a catheter is passing down the ascending aorta into the left ventricular cavity, shown here. There's some mitral regurgitation so the left atrial cavity here is outlined and I would like you to notice this filling defect here, which represents the tumour itself, immediately behind the anterior cusp, here. So if one imagines the echo beam passing in the direction shown by the pointer, so you can see how it passes through the anterior cusp and through the tumour which is behind it during diastole but goes back into the left atrium again during systole, along with the mitral regurgitation.

<Gibson narrates over a further echocardiogram, uses indication stick>

The next slide shows that other valves apart from the mitral valve can be studied. This is a patient with mitral stenosis. Here is the mitral valve, recorded at a rather slower time base than previously, but nevertheless the diastolic closure rate is clearly reduced. Here is the interventricular septum in front of it and this echo here comes from a normal tricuspid valve. This was significant in this patient, as a raised venous pressure with large V waves suggested the presence of tricuspid regurgitation and obviously the question arose as to whether it was organic or functional. If one sees a tricuspid wave of this sort then one can totally exclude organic tricuspid valve disease as a cause of a raised venous pressure.



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<Gibson narrates over a further echocardiogram, uses indication stick>

Next slide shows echoes from the aortic root. The aortic root, one sees the anterior and the posterior borders of it here. And within it are echoes from two of the aortic valves cusps which come together during diastole and separate during systole to form this rather characteristic box-like structure. This anterior echo comes form the right coronary cusp and the posterior one usually comes from a non-coronary cusp although it can come from the right coronary cusp. Immediately behind the aorta is a cavity here which represents the left atrium whose size can be measured as the distance between the posterior border and the back here. In front of the aortic root is the outflow tract of the right ventricle.

<Gibson narrates over a further echocardiogram, uses indication stick>

Sometimes one can detect abnormalities of the aortic valve cusps. This is the aortic echocardiogram of a patient with severe regurgitation involving the aortic valve. And as you see the normal box-like structure has been completely replaced by these diffuse echoes here. These come from vegetations on the valve and show the normal structure has been totally destroyed. Sometimes one can identify the particular cusp on which the vegetation will be found and you can tell the surgeon accordingly.

<Gibson narrates over a further echocardiogram, uses indication stick>

It's also possible to identify abnormal structures within the left aortal cavity itself. This is the echocardiogram from a child who presented as mitral stenosis. Again we see the aortic root with anterior and posterior borders but behind the posterior border, within the left atrial cavity itself is this abnormal echo which separates from the aorta during systole and comes to lie alongside it during diastole. Cardiac catheterisation in this child showed a gradient between the wedge and the left ventricular pressures, however the mitral valve echocardiogram was normal and these are the classical appearances of cor triatriatum.



<Gibson narrates over a further echocardiogram, uses indication stick>

If one angles the transducer a little forward from the aortic valve, one can pick up echoes from the pulmonary valve. The orientation of the pulmonary valve is very unfavourable to detecting echoes and usually one can detect only one cusp, shown here in its systolic position and here opening during its diastolic position. It's of some importance, as we shall see later, in patients with congenital heart disease to be able to detect the pulmonary valve because if one can show that the anterior great vessel is to the left then this definitively excludes de-transposition in a sick infant. Using echocardiography, therefore, one can detect all 4 cardiac valves.

<Gibson narrates over a further echocardiogram, uses indication stick>

One can also get evidence about a pericardial infusion. And indeed this is one of the major applications of the technique. This is the echocardiogram from a 24-year-old girl with Hodgkin's disease involving the mediastinum. Here we see the interventricular septum, the left ventricular cavity and the posterior heart border shown here moving forward during systole and backward during diastole. Behind the heart is this echo-free zone here and between the heart itself and these posterior mediastinal echoes. This zone here represents the fluid and you notice that one has 3 characteristic signs; one has moving heart border and echo-free zone and behind that are mediastinal echoes which are stationary. Sometimes it's possible by manipulating the gain control on the instrument to make an apparent space appear behind the posterior wall. But if there is no pericardial effusion, then behind this, motion will be transmitted to the posterior mediastinal structures. Echocardiography is now the medium of choice for diagnosing pericardial effusion, it's much quicker and easier than putting air into the pericardium or putting dye into the right atrium. It can be done repeatedly and the transducer can be put in the region where one intends to put the needle when one's aspirating the effusion and one knows then just how deep one's going to have to go before one will tap fluid. It's made all the difference to dealing with pericardial effusion.



00:24:54:00

<Gibson narrates over a further echocardiogram, uses indication stick>

A very major application of echocardiography is studying left ventricular function. This picture is taken from one of Inge Edler's early papers, who was a pioneer of the technique. The lower half is a cadaver heart showing the ventricular septum here, the anterior cusp of the mitral valve and the posterior wall of the left ventricle here. Above are the echoes obtained from it. You notice that echoes come from the front and the back of the septum, the mitral valve cusps and the posterior wall of the left ventricle. If one points the echo beam across the left ventricle in the living subject [...]

<Gibson narrates over a further echocardiogram, uses indication stick>

[...] one can get a very similar picture. Here is the left ventricular echocardiogram from a normal subject recorded at a fast time base. The QRS complex is shown there. These two echoes come from the front and the back of the septum, echo here from the back of the anterior and the posterior cusp of the mitral valve, and the endocardium here of the posterior wall of the left ventricle. It's possible therefore to measure a dimension between the septum here and the endocardial surface of the posterior wall of the left ventricle shown here. Obviously, a number of possible dimensions can be measured in the left ventricle and in order to standardise measurements it is essential that part of the mitral valve apparatus is included in the record.

<Gibson narrates over diagram of computerised cineangiogram, uses indication stick>

This shows diagrammatically a computerised cineangiogram. Each of these lines represents the outline of the opacified left ventricular cavity in successive cine frames taken at 20 second intervals. What I'd particularly like to stress is the way a normal left ventricular contraction is a very uniform process. There is concentric inward movement all the way round the cavity so that if one puts an echo beam across the



ventricle in this sort of region here then it will tell you something about the cavity as a whole. So if one is going to have any idea of left ventricular function simply by measuring a single dimension, one has to make allowance for the fact that contraction pattern may not be uniform.

<Gibson narrates over computer-generated chart, uses indication stick>

It's possible not only to measure the dimension at n-systole and n-diastole, as we previously discussed, but also to measure it all the way through. This involves the use of a simple computer technique. One runs a pencil follower along the echo representing the septum and another along that representing the posterior wall. The computer then generates a set of co-ordinates for each of these two echoes and subtracts one from the other, thus enabling the left ventricular dimension to be plotted out, shown on the lowest trace here. This is from a normal subject. 0 time corresponds to the QRS complex of the ECG, time goes along the bottom, the difference being .1 of a second. At n-diastole the left ventricular dimension is just over 4.5 cm., you notice that it drops during systole, increases rapidly at first during the rapid phase of ventricular filling, during mid-diastole there's little movement, corresponding to the period of diastasis and finally there's a further increase in late diastole representing the contribution of left atrial systole.

Once one has the figures in the computer of course, one can do other things to them. One can, for instance, look at the rate of change of dimension which is shown here in the middle trace. Here, as a negative deflection, one sees the rate of change as the dimension drops to about 15cm a second as the cavity size gets smaller in systole. In diastole the peak rate of increase of dimension reaches approximately 20cm per second and you notice that the filling pattern is shown very clearly on this trace with the initial peak corresponding to the early rapid phase of filling, the period of diastasis when the filling is slow and a late peak here corresponding to left atrial systole.



Sometimes, if one's interested in left ventricular systolic function it's useful to know the rate of change of unit length of diameter, so-called VCF or velocity of circumferential fibre shortening and that's shown in the top trace, here. Notice in this normal subject, the VCF reaches well below 2 which is the lower limit of normal, so one can say that the rate of contraction in this normal subject is well within what one would expect.

00:30:07:30

<Gibson narrates over graph, uses indication stick>

One can compare measurements made by echocardiography from those made by angiography in the same patients. This is from a study in which 23 patients were studied at cardiac catheterisation. Vertically we plot the echocardiographic dimension and horizontally we plot the angiographic one, and you see that there is excellent correlation between the two, even though the measurements weren't made absolutely simultaneously but with a gap of perhaps 10 or 15 minutes between the two.

<Gibson narrates over cineangiogram, uses indication stick>

Echocardiography can be used to study a number of different types of left ventricular disease. Here is the digitised cineangiogram of a patient with very poor left ventricular function – you see the cavity is greatly dilated and the overall amplitude of wall movement is very low. The ejection fraction which expresses this type of disturbance was less than 20% in this patient.

<Gibson narrates over a further echocardiogram, uses indication stick>

This shows the echocardiogram. Again, the interventricular septum here and the posterior wall. A very large cavity is shown by the increased distance between these two echoes, the low amplitude of wall movement is obvious and in addition you can see this patient has pulsus alternans with alternative large and small beats here.



Notice the velocity of the large beat, here, is greater than that of the small one although they both start from the same n-diastolic diameter.

<Gibson narrates over cineangiogram, uses indication stick>

One type of left ventricular disease, however, that the echocardiogram cannot detect is that due to incoordinate contraction. These are the diastolic frames from a patient with ischaemic heart disease which show a very obvious area here of disturbed contraction. The approximate path of the echo beam would be across there and therefore, using echocardiography, one could have no idea of what was going on at the apex. So the echo alone cannot be used to detect incoordinate contraction simply from looking at the left ventricular contraction. If one's going to study patients with incoordinate contraction one must have additional information. It's also possible, using this technique of digitised cineangiography to compare peak rates of wall movement made by the two techniques of echocardiography and angiography.

<Gibson narrates over angiogram, uses indication stick>

This is the angiogram of a patient with mitral and aortal regurgitation and this line here represents the approximate path taken by the echo beam. It's possible to programme the computer to plot out the rates of change of wall movement along that line exactly as in the echocardiogram which is shown here.

<Gibson narrates over computer-generated diagram, uses indication stick>

Again, time goes from left to right, 0 time corresponding to the QRS complex. Again, you see a reduction in diameter during systole with an increase during diastole. The usual division between the rapid filling phase and diastasis is lost in this patient on account of the valvular regurgitation. And, exactly as before, one can plot the peak rates of wall movement during systole and during diastole [...]

<Gibson narrates over graph, uses indication stick>



[...] and compare them with the corresponding values from the same patient derived by echocardiography. This is a comparison between the two, the echo estimates are plotted vertically and the angiographic ones are plotted horizontally and you see there is very satisfactory agreement between peak rates of wall movement as well as in measurements of the dimension itself made by echocardiography.

Now, having made these measurements, are they of any practical significance?

<Gibson narrates over further graph, uses indication stick>

Well, if one compares during systole the shortening rate here, shown as VCF, plotted along the bottom, with the peak rate of rise of left ventricular pressure as measured by a catheter tip micromanometer at cardiac catheterisation, the echo and the angio measurements being made simultaneously, you will see it is a very close relationship between the two. So it looks as though one can get a good idea of left ventricular function echocardiographically of the same type of function as is measured by peak left ventricular function by Dp/Dt. Obviously, measurement of left ventricular function requires cardiac catheterisation, whereas measuring the shortening rate by echocardiography does not.

00:35:00:00

<Gibson narrates over graph, uses indication stick>

It's also possible to look at diastolic rates of wall movement. This is the echocardiogram for a patient with rheumatic mitral stenosis, you see here the reduced diastolic closure rate of the anterior cusp of the mitral valve. Here is the interventricular septum and here the posterior wall of the left ventricle. And I'd like you to notice how slowly these two move apart during diastole.

<Gibson narrates over computer-generated graph, uses indication stick>



The next shows the computer plot out of this patient. Again, we have reduction during systole and a very slow rate of increase during diastole. This shows up particularly clearly in the rate of change of dimension. The lower limit of normal is round about 10cm per second, in this patient the peak is only 4, and secondly the filling pattern is changed, instead of the usual rapid early peak followed by diastasis, there is a long period here where more movement continues. So one can demonstrate these two abnormalities of filling in patients with mitral stenosis. And this, in combination with an echocardiogram of the mitral valve itself has made cardiac catheterisation virtually unnecessary in patients with rheumatic mitral valve disease.

<Gibson narrates over chart, uses indication stick>

Here we put together the differences one can see in patients with various forms of heart disease in peak filling rate. In normal subjects the peak is around 10cm per second, very significantly reduced in patients with mitral stenosis and very significantly increased in patients with mitral regurgitation due to ruptured chordae tendineae. It's normal in patients with aortic regurgitation, even the stroke volume may be very large, and left ventricular disease.

The advantage of this approach which is looking at the physiology of the left ventricle, rather than at the anatomy of the mitral valve itself is that it can be extended to unfamiliar situations.

<Gibson narrates over chart, uses indication stick>

Here for instance we look at the peak rate of wall movement after the surgical replacement of mitral valve. Three types have been used – inverted aortic Homograft, the Bjork-Shiley prosthesis and the Starr-Edwards prosthesis. And for each patient we plot the peak rate of diastolic wall movement expressed in cm per second. Normal, you remember, was round about 10, so all these patients, to some extent, have mitral stenosis, perhaps rather worse in patients with Starr-Edwards than the Bjork-Shiley or homograft. If one finds a patient, following mitral valve



replacement, who has a normal diastolic rate of wall movement, it's very suggestive evidence that that patient has a paraprosthetic leak.

Another type of abnormality of left ventricular function that the echocardiogram has enabled us to study [...]

<Gibson narrates over echocardiogram, uses indication stick>

[...] has been that of the septum. The echo, in fact, has been the first method of studying the septum in a satisfactory way. Normally, as we saw, the septal echo here moves downwards or backwards during systole as the posterior wall echo moves upwards. This of course is what one would expect as the left ventricular wall gets smaller during injection. But this patient, you notice, had alternate ventricular ectopic beats resulting from digitalis overdose, and in the ectopic beats, although posterior wall movement is normal, movement of the septum is very abnormal indeed. It balloons forwards, right into the right ventricular cavity instead of moving backwards as it should into the left ventricular cavity. So effectively, with abnormalities of contraction reverse septal movement can occur, which might be expected to have a very severe effect on left ventricular function as a whole.

<Gibson narrates over anatomical diagram, uses indication stick>

Another major application of echocardiography is in the study of left ventricular anatomy in association with the great arteries and the AV valves in congenital heart disease. This is a transverse slice through the heart showing diagrammatically here the aortic route which posteriorly is continuous with the mitral valve and anteriorly is continuous with the interventricular septum. And these two continuities are very important when assessing patients with congenital heart disease since they may be disturbed.

<Gibson narrates over echocardiogram, uses indication stick>



One can demonstrate this by doing a sweep. One starts with the transducer aimed at the aortic root, shown here as two parallel echoes, with the left atrial cavity behind. One then gradually angles the transducer so that it's pointing down towards the apex of the heart. As you see, the anterior border of the aorta, here, turns into the interventricular septum and the posterior border of the aorta is continuous with the cusp of the mitral valve. So one can demonstrate, using echocardiography, these two continuities between the base of the heart and further down. A number of abnormalities in these structures can occur in congenital heart disease.

<Gibson narrates over further echocardiogram, uses indication stick>

This is the echocardiogram of a patient with an atrial septal defect. Notice the posterior wall movement is normal but the septum is moving in the reverse direction. Moving anteriorly during systole and posteriorly during diastole. This reverse septal movement is very characteristic of conditions characterised by an increase in the volume load of the right ventricle. Notice also the anterior cusp of the mitral valve, here, and its diastolic position is parallel to the septum, all the way through diastole. And this is very characteristic of an ostium primum as distinct from an ostium secundum atrial septal defect.

41:12:12

<Gibson narrates over further echocardiogram, uses indication stick>

If one is dealing not with an ostium primum but with a total AV canal or AV communis, then the relationship between the mitral valve and the septum can be even more disturbed. Here is the mitral valve echo and here the septum. And you notice that the mitral valve echo appears to be passing through the septum. Of course it's not really doing this, it merely reflects the fact that the ultrasound beam is about a centimetre across and is therefore picking up echoes from the interventricular septum itself and also from the mitral valve which is passing through the high ventricular septal defect which can exist in this condition.



<Gibson narrates over further echocardiogram, uses indication stick>

Here is another condition where the relationships between the great arteries and the septum are abnormal. Again it's a sweep, here is the anterior and posterior border of the aorta. The posterior border of the aorta is continuous with the anterior cusp of the mitral valve but the interventricular septum, shown here, stops and there is a gap between that and the anterior border of the aorta. This is very characteristic of aortic override such as can occur in Fallot's tetralogy but may also occur in truncus arteriosus and some forms of double outlet right ventricle. Again, one sees the anatomy and one can demonstrate very clearly by echocardiography that aortic override has occurred.

<Gibson narrates over further echocardiogram, uses indication stick>

It may be very important in some types of congenital heart disease to demonstrate the presence of an interventricular septum. One can't assume that an echo in the usual position of the septum, showing usual motion of the septum, is in fact an interventricular septum. It may also be a bulbar ventricular septum which separates a primitive ventricle from an outlet chamber, and this is a very important distinction surgically. So if one is going to be precise about an interventricular or posterior septum one has to show that it has an AV valve behind it and an AV valve in front of it. So in this patient one can say that this structure here is unequivocally an interventricular or posterior septum.

<Gibson narrates over further echocardiogram, uses indication stick>

This, of course, is very important in the condition of primitive ventricle where there is no interventricular septum. Here, you see, there are two AV valves, an anterior one here and a posterior one here, but there is no septum between them. The posterior cusp here of the anterior valve meets the anterior cusp of the posterior one during early diastole. And one can be quite certain from this echocardiogram that this patient has no interventricular septum. This can often be a very difficult diagnosis to make by angiography and it's now our practice, before submitting patients suspected



with having VSD to surgery, to be certain they are not suffering from primitive ventricle using the technique of echocardiography.

<Gibson narrates over further echocardiogram, uses indication stick>

Finally, one can use the technique of echocardiography to demonstrate aortic mitral discontinuity. This is from a patient with a double outlet right ventricle and represents a sweep from the mitral valve here to the aortic root shown here. But you notice the mitral valve just stops there, there is an anterior AV valve there which is continuous with the posterior border of the aorta. So one can be certain in this patient that there is no continuity between the posterior AV valve and the great vessel.

<Gibson to camera>

In summary therefore, echocardiography, I've tried to show, has a great deal to contribute in the assessment of patients with ordinary heart disease. Using the technique, one can get some idea of anatomy, both of valves and also of the way intercardiac structures are related to one another and also of physiology, particularly with reference to left ventricular function. I'm sure there are many developments in store which include two-dimensional methods of looking for example at the left ventricle and producing results looking very like angiography.

The subject has gone a long way in 25 years and it seems that in the next 25 years very considerable further progress will be made.

<End credits>