Wellcome Film Project

The Value of Intracardiac Electrography

The Scientific Basis of Medicine

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<Opening titles>

<Krikler to camera>

Electrocardiography is very much a technique of this century. It was developed during the early 1900s, and by the 1920s many of the deductive lessons that we still use had already been learnt.

<Krikler narrates over photograph of early apparatus and tracing. Then using indicator stick narrates over diagrams, further tracings, photographs and x-rays, interspersed with talk to camera>

If we, however, think back to what our forefathers in this technique had to use, we would soon realise the difficulties that caused them to produce tracings that we can now be proud of, such as one that we now see and that was recorded by Sir Thomas Lewis as long ago as 1912. However, though the ECG tells us much, it tells us quite a little about various important aspects of the intervals that we are accustomed to

reading. <*Next diagram*> If we look at the ECG, the P-R interval, the QRS and the T wave, seen here in relation to the pressure-volume change that we see in the heart, we note that the P wave is simultaneous with atrial activation, and the QRS complex is recorded during ventricular depolarisation, and subsequently repolarisation occurs. But it is what happens here in the P-R interval that we do not see on the ECG tracing.

<*Next diagram*> The electrocardiogram indicates the sequence of depolarisation and repolarisation. Starting from the sinoatrial node, whose activation is silent, we have impulses passing through muscle bundles to reach the A-V node, an area of slowing, and then the impulse passes through the bundle of HIS, through the bundle branches and the Purkinje network more quickly than through the A-V node in order to achieve ventricular depolarisation. <*Next diagram*> Looking at the tissues more closely, we see here the internodal tracts of atrial muscle bringing the impulse to the A-V node, to its three regions and through the central slowing N region and we'd like to know what happens there as well as what happens in the bundle of His. This is all silent in the ECG until we come down to the Purkinje myocardial junction and ventricular depolarisation which we then see represented as the start of the QRS complex.

<*Next diagram*> Let's look here at the silent region, the P-R interval, disturbed only by the P wave, which seems to reflect so little. It doesn't show sinoatrial nodal activation nor do we see that on an intracardiac electrogram, though we do see high right atrial, low right atrial and we can see left atrial activation. We don't see the A-V node being activated but the impulse passes through it. We can sometimes find His bundle activation and usually should depict this on our tracings. It is rarer to see bundle branch activation. And the Purkinje myocardial junction is not seen on our tracings though the ventricular activation is. If we now look at what the intracardiac electrogram shows, we can see stimulus and atrial activation if we use pacing, high and low atrial electrograms, an A-H interval between the atrium and the bundle of His giving an idea of nodal activity. We can sometimes see His to bundle branch activation and, of course, we see the onset of ventricular activation in the form of a deflection and we see this both as the Q wave of the surface of the ECG and the V wave of the intracardiac electrogram.

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<*Next diagram*> In order to study the situation with intracardiac electrography, we clearly need electrodes to be placed in appropriate parts of the heart, connected through a junction box for a recording or stimulation to an oscilloscope, where we can watch what happens, and then an ECG machine for permanent recording and we use an 8 channel device for this purpose. We can use a tape recorder for permanent recording and playback at a later date, and by connection between our ECG machine and a synchronizer with a suitable stimulator, we can pace or induce suitable stimuli that enables us to make appropriate physiological deductions and measurements <*to camera*>.

Now, with such an apparatus, under special circumstances, we take the patient to the laboratory and we pass the electrodes and we make recordings and measurements at the very time. <*Next photograph*> We use a stimulator such as is shown here, which enables us to set intervals for steady pacing as well as for the introduction of one or more additional stimuli and this enables us to measure refractory periods and also enables us to set up or stop tachycardia.

<*Next photograph showing apparatus being used in hospital theatre*> Here we see what the situation is like in actual practice. This was recorded during a study when intracardiac tracings were made at open heart surgery. And we see here the electrocardiogram which plays out at 4 times the normal ECG speed, that is at 100 millimetres a second, and above, over here, you see the stimulator. This enables us to make recordings from electrodes, <*next diagram*> which we pass and you'll see how this can be done either from the femoral vein or from an arm vein, passed beyond the tricuspid valve ring and then pulled back appropriately for the key landmark, the recording from the His bundle. That is the landmark which is absolutely essential in order to define what happens above and below the watershed and tells us what is supraventricular and what is within the intraventricular conducting system including the bundle of His.

<*Next diagram*> Here we see how this is depicted and we pass the electrode into the ventricle and produce a ventricular electrogram. We pull back into the absolute correct position and this shows us the three impulses, the A-H and V waves, shown best down here, recorded precisely opposite the tricuspid valve giving us the A-H interval and the A-V interval, the crucial measurements for intracardiac recordings together with the simultaneous surface ECG for correlation. Should the electrode be pulled back too far as, for example, here into the right atrium, we will only see an atrial electrogram and not the H and V deflections.

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<*X-ray*> Here with an x-ray apparatus, we've taken a photograph of what we see with an image intensifier and it enables us under vision to place electrodes, for example, in the coronary sinus, or here the right atrium for pacing or recording, or across into the right ventricle for recording and across the bundle of His or elsewhere in the right atrium.

<*Next tracing*> And we see on this tracing the example. Here, let us concentrate on the His bundle electrogram: the A wave of the atrium, the H wave from the bundle of His, the V from the right ventricle and this is shown in relation to the separate right ventricular recording, and above – the atrial recording. And a single surface ECG recording here: lead 1. How much one can see here <*indicates H wave*> and how little one can see here <*indicates lead 1*> of importance will become clear as we go through tracings <*next tracing*> taken in actual examples of different rhythm and conduction disturbances. But firstly, an example showing continual pacing with the pacing artefact, shown not only on the surface leads but in the intracardiac leads as well, and the A wave, the small H deflection in this case, and the V wave. So, we pace regularly to overcome autonomic influences and then we introduce pre-timed beats that enable us to carry out the physiological studies that we wish.

<*Next tracing*> Let us look at a normal sequence of activation, well shown here: from high right atrium we see the A wave, which is a little further on in the His bundle electrogram which reflects low right atrial activation. And we see that the left atrium,

here recorded from the distal coronary sinus, is depolarised after the low right atrium. And then, of course, the surface ECG leads which act as our markers of what we know. If we were to move the coronary sinus electrogram a bit more proximally, we will see less atrial activation but much more ventricular activation and here we get a good indirect idea of left ventricular depolarisation. These are physiological tracings taken in sinus rhythm, <*next tracing*> but in the next tracing, we see an atrial extrasystole which follows after one sinus beat and which precedes another. We can locate its origin to the left atrium by virtue of the fact that the first A wave is seen in the coronary sinus electrogram. Thereafter the high right atrium and the low right atrium are depolarised, something we would not have been able to deduce from any of our surface ECG recordings. We can also see when we have a ventricular extrasystole, which we can diagnose as such from its morphology in the surface leads, that its retrograde impulse comes back to the low right atrium first, that is via the His bundle, and thereafter comes to the coronary sinus and goes up to the high right atrium. So, we see another additional item when retrograde atrial activation occurs, it comes through the His bundle unless it comes through an anomalous tract – of which more later.

<*Next tracing*> We also use pacing for studies of conduction disturbances and this is an example where we have been pacing regularly and you can see the pacing artefacts here. As we pace at this particular rate, we see that the A-H intervals lengthen progressively until finally there is a dropped beat and then there is the restoration of the original A-H interval after the dropped beat. We have seen a Wenckebach block sequence in the atrioventricular node with no activation at a critical rate and this we use as an index of A-V nodal conduction function.

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<*Next tracing*> In congenital complete heart block, which is located within the A-V node, these continuous tracings simultaneously of an intracardiac recording from the His bundle electrogram and here from a surface lead show the total dissociation but also that the subsidiary focus that leads to ventricular activation is within the bundle of His, that is the impulse is blocked within the A-V node and we're dealing with a

high subsidiary focus. On the other hand, when we deal with complete heart block of trifascicular nature, that is, where there is bilateral bundle branch block, we see that the atrial activation is always linked with His bundle depolarisation but that the ventricular activation is quite separate from a true idioventricular focus, which in addition produces a wide, bizarre QRS complex. This is helpful in showing the sort of patient who needs a pacemaker as is much more commonly required in this rather than in congenital block. But this is not always quite so simple <*next tracing*> for here we see a patient with complete heart block. Here we see that there are narrow QRS complexes, but if we look at the His bundle electrogram and the simultaneous tracings, we see that there are 2 His bundle deflections.

<*To camera*> These 2 HIS bundle deflections show that we're dealing with intra-Hisian block, something that we could never diagnose from the surface electrocardiogram – the normal QRS complex would give us the wrong idea. We were able to show in this case that the patient needed a pacemaker; we were dealing with acquired intra-Hisian block as a result of cardiac surgery. This is a fairly new discovery and something that we find of great importance where we're dealing with cases of doubt, where syncope, for example, complicates heart block with narrow QRS complexes.

<*Next tracing*> Now we turn to rhythm disorders. We would not use intracardiac electrography in order to diagnose atrial fibrillation which is evident in the surface tracing, but what we do see in these two intracardiac tracings, respectively a mid and a high right atrial tracing, organised into groups of muscle activity in one part to a greater extent than the other. The atria are much less silent in atrial fibrillation than one might deduce from the surface ECG. Also we see here something where intracardiac electrography has probably provided, with programmed electric stimulation, some of the most fundamental advances of understanding of arrhythmias and of their definition. This is an example of junctional reciprocating atrioventricular tachycardia, usually called paroxysmal supraventricular tachycardia and previously erroneously called paroxysmal atrial tachycardia. The first complex that we see here is a normal sinus beat as, indeed, is the second one. And these have occurred here and a stimulus has been introduced here and this has found one of the nodal

pathways refractory, the other one conductive slowly so that in this ladder diagram, we see slow conduction and then recovery with retrograde conduction back to the atrium with atrial depolarisation, but a circus movement is now established. A tachycardia has been set up by an induced atrial extrasystole. And this is the mechanism of the common-or-garden paroxysmal supraventricular tachycardia that so often occurs in young people. It can be started artificially in this way, but it very often starts naturally through extrasystole.

The other important lesson from this is the other side of the coin, namely that when this tachycardia is in progress, as we can see here from the ladder diagram, and we can see activation down through the bundle of His to the ventricles and then back again slowly to the atria and then once more down like this, we can see that the introduction of a suitably timed atrial extrasystole here, electrically-induced, blocked the pathways concerned in this re-entry mechanism and that the next sinus beat that we see here shows restoration of normal conduction. Here we have been able to show an important aspect of the mechanism of this type of tachycardia.

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<*Next diagram*> This particular arrhythmia is seen not only in intranodal tachycardia but also in pre-excitation. And the pre-excitation syndromes are most classically demonstrated by the Wolff-Parkinson-White syndrome. We have our electrodes placed as shown in this part of the diagram with the His bundle, coronary sinus, mid right atrial, high right atrial and right ventricular electrodes. And this is what we see. And this is the difference in the Wolff-Parkinson-White syndrome. If we look at the surface ECG leads, we see the broad, bizarre, curious complexes with a short P-R interval and the slurred upstroke, the delta wave. What this means can be shown best from the His bundle electrogram. The A-H interval is short, but even more important, the H-V interval is not only short, it is negative, -10 milliseconds.

<*To camera*> The only explanation for this set of findings is the fact that the impulse has come from the atrium to the ventricle other than through the normal pathways. And intracardiac electrography and programmed electric stimulation of the heart has

helped resolve the dilemma as to whether or not an anomalous pathway was necessary for this syndrome. We have been able to show, simultaneous with the development of anatomical techniques, that this is an essential substrate for the pre-excitation syndrome and that similar circumstances apply to the other pre-excitation syndromes with, for example, narrow QRS complexes.

<*Next diagram*> Here is the situation that we see in the Wolff-Parkinson-White syndrome. The QRS complex seen during sinus rhythm is the result of fusion of conduction down the normal pathways and down the anomalous pathway blending, producing this fusion complex. Where this anomalous pathway blocked, we would see the normal QRS complex with a normal P-R interval. This is what we also see in tachycardia where the impulse comes down the A-V node in the normal direction and goes back up the accessory pathway and the continuous circus movement is thus established. Were this ablated, we would see this. On the other hand, if this were not functioning, we would see the bizarre, broad, total delta wave which we sometimes see in atrial fibrillation when all conduction tends to be down the anomalous pathway.

<*Next tracing*> Induction of the tachycardia by an extra stimulus is well shown here. In A, we see two successive impulses that have been paced. The third impulse is paced earlier and produces a longer conduction time with a broad, bizarre QRS complex exaggeration of the pre-excitation anomaly. But if we pace slightly earlier, as we have done here, we now see block in the accessory pathway with conduction slowly down the normal pathway, and then retrograde conduction up the now receptive anomalous pathway, and a continuous reciprocating tachycardia has been established. Even earlier, stimuli would produce block either in the normal conducting tissues or the atrium itself might be refractory.

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<*Next tracing*> We see sometimes the need for two critically timed extrasystoles, produced here electrically. And, indeed, in this patient, two extrasystoles had been noted to occur spontaneously causing his tachycardias. Here two stimuli, 300 milliseconds apart, were insufficient to produce block in the accessory pathway and

the next sinus beats that we see here show the conventional pattern of pre-excitation with a normal short P-R interval and a well-shown delta wave.

We now again apply two of the extra stimuli with only minute degree of shortening of the interectopic interval. And now tachycardia has been established with narrow QRS complexes, typical of that seen in pre-excitation but quite different from that which we see during sinus rhythm in this syndrome.

<*Next tracing*> Another place where intracardiac electrography is useful and which can provide benefit with much less sophisticated equipment is the passive recording of a right atrial electrogram in a patient with paroxysmal tachycardia as seen here with broad QRS complexes, where one might not be sure whether one was dealing with a supraventricular tachycardia with aberration or with ventricular tachycardia. The finding of atrial complexes like this, dissociated from the ventricular activation here or in the intracardiac lead, is ample proof that the tachycardia is of ventricular origin and enables the appropriate therapy to be chosen.

<*Next tracing*> We revert here to one of the lessons that has been learnt from intracardiac electrography. Not only can we show in such a patient that drugs may stop the tachycardia, we may also show that when drugs fail to benefit the patient prophylactically, that if we choose the right position and type of pacemaker we may be able to programme it to switch off the tachycardia, and then we see here the start of the next normal sinus beat. And this has been an important advance in the therapy, in the prophylaxis too of paroxysmal tachycardias of this sort.

<*To camera*> Now let us come, as we end, to two aspects where we are learning things that we had not suspected and that we might not really have got to understand well without the benefit of this technique. The first tachycardia that I'm going to speak of may not seem very important and is very little known. <*Next diagram*> This is paroxysmal sinus tachycardia. How, you may say, can we have such a disorder? We can, indeed, have re-entry in the sinoatrial node, and looking at one's ECGs in retrospect, one sees many examples that one may not have recognised. Here we see, up above, sinus beats and then we see, on the right, tachycardia beats. In both

cases atrial activation sequence is identical and the P waves look the same, but if we introduce, as we have done here, a suitably timed high right atrial extra-stimulus, we have induced a tachycardia that perpetuates itself, i.e. we have induced a reciprocating tachycardia, and because of its characteristics, it is located in or around the sinoatrial node. Furthermore, if we look at the bottom panel, the same patient later on, we see the same sequence of activation as we did here but we induce a similarly timed atrial extra-stimulus and we stop it and normal sinus rhythm is restored. These tachycardias tend to be relatively slow and are often not complained of. They are perhaps of great physiological importance save that we may find that they lead to our final topic which is the question of sinoatrial block <*next tracing*>.

Sinoatrial block was originally described as long ago as 1912 by Thomas Lewis and one of his co-workers, but it was really only the advent of intracardiac pacing techniques that led to better diagnosis. This is one example of a diagnostic technique that is useful under these circumstances. The patient has undergone pacing, right atrial pacing, at a pre-determined rate, the pacemaker has been switched off and, as you'll see, the sinus node recovery time exceeds 3 seconds instead of the normal 1 to 1.2. This indicates disease of the sinoatrial node and helped us confirm the need for a pacemaker in this case.

<*To camera*> We turn from these techniques back to a brief reconsideration of electrocardiography. The electrocardiogram reflects what we have seen. We can deduce much. We can, indeed, deduce more now that we know what intracardiac electrography reveals, and possibly its greatest lesson of practical benefit to the general cardiologist, and to the general physician concerned with electrocardiography, is a better understanding of how to interpret the ECG.

<End credits>