His bundle electrograms

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Since the recording of the first human electrocardiogram by Waller in 1889 the science of electrocardiography has developed and advanced rapidly, but has been limited by the inability to record electrical activity from specialized areas of the heart. A significant contribution was made by Alanís, González, and López in 1958 when they recorded His bundle potentials in the isolated perfused dog heart using fine needle electrodes placed along the atrioventricular groove. The first His potentials to be recorded in humans were described by Giraud, Puech, and Latour (1960), and later by Watson, Emslie-Smith, and Lowe (1967). However, it was left to Scherlag and associates (1969) to develop a simple and safe electrode catheter technique for the consistent recording of electrical potentials from the His bundle site in man. This technique was later extended to the recording of atrioventricular nodal and right bundle-branch potentials (Damato *et al.*, 1969a) and has resulted in an extensive investigation of the specialized conduction tissue in normal and abnormal circumstances. As a result certain age-old concepts have now been proved correct or have had to be modified, and in particular the traditional criteria for interpretation of many dysrhythmias have been shown to be inadequate.

Catheter technique

In order to record the His bundle potential a bipolar or multipolar electrode catheter is introduced percutaneously into the femoral vein and under fluoroscopic control positioned across the tricuspid valve into the right ventricle. The proximal terminals of the electrode catheter are attached to an electrocardiograph amplifier and used either as a unipolar or bipolar system. His bundle activity is best recorded at high frequency settings of 40 to 500 cps at a paper speed of 100 or 200 mm/second. The position of the recording electrode is then adjusted until it is stable at the

base of the atrial septum in the middle of the tricuspid valve area where a sharp rapid biphasic or triphasic deflection should appear between the P and QRS complexes of a standard electrocardiogram recorded simultaneously. The recording of this His potential is safe and is usually obtained about 5 minutes after the catheter is placed in the femoral vein. The P-His interval, measured from the beginning of the P wave to the beginning of the His potential, represents conduction through the atrium and atrioventricular node; and the His-Q interval, measured from the beginning of the His potential to the beginning of the QRS complex, represents intraventricular conduction (Fig. 1). The generally recognized normal values for P-His interval are 80 to 140 msec and for His-Q interval are 35 to 55 msec.

Validity of measurement

As there are several possible sites for recording electrical activity during the PR interval, proof that this rapid deflection represents His bundle activity is imperative. By performing several interventions in isolated perfused dog hearts, Alanís *et al.* (1958) were able to validate their measurements. Crushing of the SA node resulted in His bundle activity followed by ventricular activity without any corresponding atrial potential. Production of complete heart block resulted in atrial activity followed by a constantly related His potential with an independent ventricular potential. Manoeuvres such as atrial stimulation, vagal stimulation, acetylcholine and epinephrine injection, or the production of asphyxia appropriately modified the P-His interval without affecting the His-Q interval.

In man validation of the His bundle potential is more difficult. The position of the recording electrode is at the established anatomical position of the bundle of His. During several successive cardiac cycles the configuration of the potential should not vary significantly, and the distance from the P wave should not vary by more than 5 msec. This configuration and relation to the P wave has been validated by direct placement of the

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FIG. 1 A recording of His bundle activity in a normal subject. H.B.E. = His bundle electrogram. Sinus rhythm at 80 beats/min is present. The P-His interval is 120 msec and the His-Q interval is 45 msec. Paper speed is 100 mm/sec.

electrode on the His bundle during cardiectomy under experimental conditions (Hoffman *et al.*, 1960). However, in this catheter position both atrioventricular nodal and right bundle-branch activity may also be recorded. Atrioventricular nodal activity can be easily identified and differentiated from either the bundle of His or right bundle-branch potentials. It is of longer duration (up to 50 msec), of lower amplitude, inscribed slowly, and often with notching on the upstroke. Increasing the heart rate by atrial pacing results in progressive prolongation of the P-His interval, the His-Q interval remaining constant (Damato and Lau, 1970). Though the P-atrioventricular nodal interval also increases with atrial pacing, the increase is only a fraction of the prolongation in the P-His interval. The distinction between right bundle-branch and His bundle activity is more difficult. The

P-right bundle-branch interval is also prolonged with atrial pacing. The right bundle potential is normally about 10 msec in duration and appears closer to the QRS complex, whereas the His potential is 15 to 20 msec in duration and appears closer to the P wave. However, if only one potential is recorded it is difficult to say for certain where it is recorded from. Narula, Scherlag, and Samet (1970d), by pacing at the site of the electrical activity, were able to distinguish these two potentials. Pacing the right bundle-branch resulted in left bundle-branch block, whereas pacing the bundle of His caused no change in the QRS configuration. The pacing stimulus to QRS interval was the same as the His-Q interval recorded during sinus rhythm. Thus, for absolute separation of the His bundle and right bundle-branch potentials, pacing at the recording site appears necessary.

Atrioventricular block

During the past few years it has become apparent largely from histological studies that the aetiology of complete atrioventricular block is usually due to pathology in the main bundle-branches rather than in the atrioventricular node (Lenegre, 1964; Lepeschkin, 1964). The electrocardiographic criteria for precise location of the site of the lesion in complete heart block have hitherto been unreliable, but the recording of His bundle electrograms now allows the cardiologist to make an accurate assessment of the site of the block. In 86 per cent of one series (Narula *et al.*, 1970c) the block was demonstrated to be distal to the bundle of His, the P-His interval being normal in all patients. In all 10 patients studied by Damato and Lau (1970), the block was found to be in His-Purkinje system, atrioventricular nodal conduction being normal. In our own experience acquired heart block appears to be almost uniformly due to bundle-branch disease though one patient with Ehlers-Danlos syndrome and congenital heart block was found to have block proximal to the bundle of His (Smithen and Sowton, 1971). It appears likely that congenital heart block is frequently due to a defect in the atrioventricular node.

His bundle recordings have been particularly useful in identifying mechanisms in the two types of second degree heart block. Mobitz Type I block (Wenckebach) is characterized by a progressive prolongation in the PR interval until there is a non-conducted P wave. His bundle records show that this dysrhythmia, either spontaneous or produced experimentally by atrial pacing, is usually due to block in the atrioventricular node and that

intraventricular conduction is normal (Damoto *et al.*, 1969c; Narula *et al.*, 1970a). Mobitz Type II block is characterized by a constant PR interval with periodic non-conducted P waves. It usually occurs with bundle-branch block but sometimes with a narrow QRS complex. The clinical observations of Langendorf and Pick (1968), and the experimental observations of Watanabe and Dreifus (1967), suggest that Type II block represents bilateral bundle-branch disease. His bundle electrograms have confirmed that in cases of Mobitz Type II with bundle-branch block, the site of block is distal to the His bundle, the atrioventricular nodal conduction being entirely normal (Narula and Samet, 1970). In Mobitz Type II block with narrow QRS the site of block may be either in the atrioventricular node, bundle of His, or the bundle-branches.

Not all cases fit this simple concept, and Narula and Samet (1970) described two cases of Mobitz Type I block with normal atrioventricular nodal conduction and block in the His-Purkinje system. They suggested that the distinction in the second degree blocks should be made on the basis of the site of block such as nodal or His-Purkinje system rather than Mobitz Type I or Type II. From a clinical point of view, this is extremely important for it is the blocks in the His-Purkinje system that seem to advance to complete heart block and not those in the atrioventricular node.

The site of heart block in patients with acute myocardial infarction was recently studied by Rosen and associates (1970b). In patients with diaphragmatic myocardial infarctions, the block was found to be proximal to the His potential, while in anterior myocardial infarctions the block was distal. Since 90 per cent of the time the right coronary artery supplies the atrioventricular node, it is not surprising to find that the block occurring with diaphragmatic infarcts, usually due to right coronary

artery occlusion, is in the atrioventricular node. Rosen's findings confirm the view that heart block following acute anterior infarction is due to bundle-branch disease. The difference in clinical behaviour between heart block in anterior and diaphragmatic infarctions can thus be explained by the different anatomical site of the lesion.

A very fascinating case of what appears to be second degree atrioventricular block on a standard electrocardiogram was reported by Rosen, Rahimtoola, and Gunnar (1970c). His bundle electrograms revealed that the apparent block was due to multiple non-propagated premature His bundle depolarizations with concealed conduction in the atrioventricular junction. The authors called this dysrhythmia pseudo-atrioventricular block because there was no evidence of an intrinsic conduction abnormality.

Dysrhythmias

The interpretation of certain dysrhythmias may be considerably aided by the use of bundle of His electrograms. The zone of concealment in both atrial fibrillation and flutter has been found to be proximal to the bundle of His (Lau *et al.*, 1969). Of more importance from a therapeutic point of view is the distinction between aberrantly conducted and ventricular ectopic beats. The electrocardiographic criteria suggested by Marriott and Sandler (1966) may be extremely misleading particularly in the absence of P waves as occurs in atrial fibrillation. A His bundle recording will conveniently allow this distinction because ventricular ectopics will not be preceded by a His deflection whereas aberrant beats will, the His-Q interval being the same as for the normal complexes. Recently a patient presented to us with atrial fibrillation, on large doses of digitalis. The standard electrocardiogram (Fig. 2) revealed several wide QRS complexes which were felt to be ven-

FIG. 2 Rhythm strip (lead VI) of a patient with coronary artery disease taking digitalis (see text). Atrial fibrillation is present. The wide QRS complexes were interpreted as ventricular ectopic beats, but aberrantly conducted supraventricular complexes could not be excluded.

tricular ectopics, and the clinical impression was that of digitalis toxicity. However, a His bundle electrogram revealed that each wide QRS complex was preceded by a His potential and the His-Q interval was the same in all complexes (Fig. 3). Because of this, digitalis was continued rather than stopped, diuretics were introduced, and shortly thereafter the aberrant beats disappeared.

The distinction between supraventricular tachycardia with aberration and ventricular tachycardia can also be different. His bundle recording can be of value in this situation by recording His potentials in front of QRS complexes in supraventricular dysrhythmias and not in ventricular dysrhythmias.

By recording from the specialized conduction system, Damato and Lau (1969) have been able to show that middle and low 'nodal' rhythms usually arise from the bundle of His. During these 'His bundle rhythms', a single His deflection and no nodal deflection precedes each QRS complex. The His-Q interval during tachycardia is the same as that during sinus rhythm. Retrograde conduction from the His bundle through the atrioventricular node to the atrium has been shown, and in some cases nodal and His activity are independent of each other, while His and ventricular deflections have a constant relation.

Paroxysmal supraventricular tachycardia has been shown recently to be usually caused by re-entry in the atrioventricular conduction system (Bigger and Goldreyer, 1970; Gettes and Yoshonis, 1970). The site of re-entry was

studied by Goldreyer and Bigger (1971) in 5 patients with episodes of paroxysmal supraventricular tachycardia. In all 5 patients His bundle potentials were recorded during tachycardia. Every episode of tachycardia was initiated by an atrial premature depolarization with prolonged conduction from atrium to His bundle. However, in 2 patients a premature depolarization induced supraventricular tachycardia but failed to propagate across the entire atrioventricular node to stimulate the bundle of His. Since His bundle depolarization was not required for supraventricular tachycardia initiation, it can be inferred that the site of re-entry in these patients was within the atrioventricular node.

His bundle electrograms have proved useful in the study of the genesis of Wolff-Parkinson-White syndrome. In 2 cases, Castellanos and co-workers (1970) were able to show ventricular activation which preceded a His potential, suggesting that ventricular pre-excitation was due to a bypass of the His bundle. The same group also demonstrated that during supraventricular tachycardia in Wolff-Parkinson-White syndrome antegrade conduction consistently occurred through the His bundle whereas retrograde conduction did not (Castillo and Castellanos, 1970). They concluded that the reciprocating tachycardias in this syndrome were probably a result of two separate anatomical communications, but re-entry within the atrioventricular node was not excluded.

FIG. 3 *His bundle recording during the rhythm seen in Fig. 2. A His bundle potential precedes each QRS complex regardless of configuration. The His-Q interval is 50 msec. The second and fourth complexes are normally conducted and the first and third complexes are abnormal. Paper speed is 100 mm/sec (see text).*

Drug studies

One of the more important applications of His bundle electrograms has been the study of the effect of certain cardiac drugs on atrioventricular conduction in man. With this technique it has been possible to show that isoprenaline and atropine shorten atrioventricular nodal conduction (Damato *et al.*, 1969b) and digitalis prolongs it (Damato and Lau, 1970). None of these drugs affect intraventricular conduction. We studied the effect of certain beta-adrenergic blocking agents on atrioventricular conduction using bundle of His recordings (Smithen, Balcon, and Sowton, 1971). Propranolol, in doses within the known therapeutic range, produced a significant increase in atrioventricular nodal conduction of up to 17 per cent without affecting intraventricular conduction. Dextropropranolol produced a much smaller but still statistically significant change in the P-His interval without affecting the His-Q interval and practolol produced no significant effect on either interval in the doses used.

The effects of lignocaine and procainamide on atrioventricular conduction have been studied by Rosen *et al.* (1970a). In the usual clinical doses lignocaine had no effect on atrioventricular and intraventricular conduction whereas procainamide usually prolonged both these intervals.

Future developments

Though a great deal of information has already been obtained, application of this technique will help solve new problems in the future. The recording of left bundle-branch potentials (Narula *et al.*, 1970b; Lau, Bobb, and Damato, 1970) along with His bundle, atrioventricular node, and right bundle-branch potentials will provide better understanding of the normal and abnormal activation process. The problems of ventricular pre-excitation and re-entry mechanisms, and the effect of new pharmacological agents on atrioventricular conduction can be studied. In addition to this type of information relating to tachydysrhythmias, there are several questions concerning slow rhythms under active investigation. Do patients with sinus node disease have abnormalities of atrioventricular or intraventricular conduction? Do patients with bundle-branch block have conduction defects in the other bundle-branch? Will it be possible to predict which patients with bilateral bundle-branch disease will develop complete heart block and when?

The answers to these and other questions will undoubtedly make the recording of His

bundle electrograms in man one of the most useful tools to become available to cardiovascular investigators and clinicians in recent years.

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