Editorial

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New frontiers in arrhythmias

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Understanding of the mechanisms responsible for disorders of cardiac rhythm has been increasing rapidly in the past 10 years. A great stimulus to these advances in knowledge has been the recognition in Coronary Care Units of the high mortality associated with some arrhythmias. The principal inroads that have been made into the high mortality of patients with acute myocardial infarction who are treated in hospital have been the prompt recognition and treatment of dangerous arrhythmias. Similarly, following cardiac surgery, particularly open-heart surgery with bypass, technically satisfactory surgical procedures may be compromised by the occurrence of disorders of rhythm. In recent years the keystone in successful management of the acute phase of myocardial infarction and also in the control of postoperative arrhythmias has been constant monitoring of the electrocardiogram. It is therefore a great pity that no uniform lead system has been generally adopted for this purpose. One of the most important problems in arrhythmias is to differentiate between beats (or rhythms) of supraventricular origin which have been conducted to the ventricles with aberration and those representing ventricular ectopy, since the correct treatment may often be different and even conflicting. In 1965, Sandler and Marriott pointed out that the best way of differentiating between supraventricular beats conducted with aberration and ventricular ectopic beats was from the morphology of the QRS complexes in lead VI. The vast majority of supraventricular beats conducted with aberration show the pattern of right bundle-branch block with a triphasic pattern in lead VI (rsR', rSR', or rSR') whereas ventricular ectopic beats usually show either a monophasic R wave or a qR complex. Marriott and Sandler (1966) have reviewed the differentiation of anomalous beats in atrial fibrillation. Unfortunately in the setting of the Coronary Care Unit or in the postoperative surgical recovery room, lead VI is quite unsuitable, since it entails connecting all four limbs to the monitoring equipment. Marriott and Fogg (1968, 1970) have described a bipolar chest lead system which largely overcomes these difficulties and still retains the diagnostic advantages of lead VI. This lead system is illustrated in Fig. 1. The positive electrode is placed in the normal position of the VI electrode (i.e. in the fourth right intercostal space close to the sternum) and the negative electrode is placed beneath the outer fourth of the left clavicle. The earth electrode is placed beneath the outer fourth of the right clavicle. Marriott and Fogg have termed this the MCL1 (modified chest left arm lead). The positive electrode can alternatively be placed in the normal V6 position to give an MCL8 lead, or on the left side of the abdomen where it imitates lead III and is termed the M3 lead.

One of the primary advantages of this system is mechanical, since when lead MCL1 is used for routine monitoring the lead wires can be conveniently draped over the right shoulder, thus leaving the praecordium free for clinical examination or, if necessary, for applying the paddles of a defibrillator. The diagnostic advantages of this lead system are numerous. First, lead MCL1 helps to differentiate between aberrant conduction of supraventricular stimuli and ventricular ectopic beats or rhythms.

Second, a right chest lead is very convenient for distinguishing between right and left ventricular ectopic beats. This may be important, for according to K. W. G. Brown (quoted by Marriott and Fogg, 1970), right ventricular ectopic beats almost never trigger ventricular fibrillation, whereas primary ventricular fibrillation may often be heralded and triggered by left ventricular extrasystoles.

Third, a right chest lead is also excellent for differentiating between left and right bundle-branch block and this may sometimes
bundle of His originates from the 'tail' of the AV node and is divisible into the 'penetrating' segment and a 'branching' segment. The penetrating segment runs through the central fibrous body where it has no contact with the common myocardium and is relatively immune from the cardiomyopathies and coronary artery disease. It is, however, susceptible to pathological processes arising from the surrounding fibrous structures. The branching portion of the bundle of His extends from the point where it begins to give off the most posterior fibres of the left bundle-branch to where it divides into the right bundle-branch and the anterior fibres of the left bundle-branch (Fig. 2). Rosenbaum et al. (1968) point out that there is no true bifurcation of the bundle in man and refer to the separation of the right bundle-branch and the anterior division of the left bundle-branch as the 'pseudo-bifurcation'.

Thus the intraventricular conduction system consists of three fascicles: the right bundle-branch, and the anterior and posterior divisions of the left bundle-branch. The most robust of these three fascicles is the posterior division of the left bundle-branch. A great advance in our knowledge of the clinical electrocardiogram has been the recognition that conduction block may occur in each of these three fascicles alone, or in any combination of two, or in all three simultaneously.

Disturbances of intracardiac conduction

Knowledge about the anatomy, electrophysiology, and histopathology of normal and abnormal intracardiac conduction has increased rapidly in the past few years. In the first place, Rosenbaum, Elizari, and Lazzari (1968) and Rosenbaum et al. (1969) in Buenos Aires have established that the normal human intraventricular conduction system consists of three fascicles, the right bundle-branch and the anterior and posterior divisions of the left bundle-branch, and have established the electrocardiographic patterns of left hemiblock; that is, block of the left anterior or of the left posterior division of the left bundle. A second great advance in knowledge has come from the development of a technique for recording the electrical activity of the human conduction system by an electrode catheter.

Anatomy of atrioventricular conduction system

Rosenbaum et al. (1968) have shown that the AV conduction system is not quite as conventionally pictured. The thin cylindrical
Isolated block of the posterior division of the left bundle-branch is rare since it is a thick structure with a rich blood supply. However, isolated block of the slender anterior division of the left bundle-branch (left anterior hemiblock) is fairly common. When this occurs, the main QRS forces are displaced upwards and to the left, producing an A QRS of $-60^\circ$ or even more leftward but usually without significant QRS widening. Left anterior hemiblock is now recognized as the commonest cause of left axis deviation in the clinical electrocardiogram. In patients over the age of 40, the most frequent cause of left anterior hemiblock is anterior myocardial infarction. Some patients presenting with typical angina but without infarction, show left anterior hemiblock in their electrocardiogram, and this may be an indication of disease of the left anterior descending coronary artery. In younger patients the more common causes are aortic valve disease, and the cardiomyopathies. Left anterior hemiblock is recognized as occurring in some types of congenital heart disease, particularly the endocardial cushion defects. There is some evidence that this is due to relative hypoplasia of the anterior division of the left bundle-branch (Feldt, DuShane, and Titus, 1970). Left posterior hemiblock alone is a very uncommon isolated lesion, largely because of its robust structure. It results in the main QRS forces being directed rightwards and inferiorly with an A QRS of around $+120^\circ$ and it may be difficult to distinguish from right ventricular hypertrophy.

**Right bundle-branch block with left anterior hemiblock**

A combination of right bundle-branch block and left anterior hemiblock occurs fairly frequently. It has been referred to in published material as 'right bundle-branch block with left axis deviation'. Its frequency reflects the vulnerability of the two fascicles, their anatomical proximity, and their largely shared blood supply. The addition of left anterior hemiblock to right bundle-branch block results in an A QRS of $-60^\circ$ to $-120^\circ$, often with a q1S3 pattern. In patients over 40 to 50 years of age, the aetiology is commonly coronary artery disease, when anterior infarction has simultaneously involved the right bundle-branch and the anterior division of the left bundle-branch. In other patients, cardiomyopathy and calcific aortic valve disease are recognized causes. There is, however, a definite group of patients with right bundle-branch block and left anterior hemiblock who show no other evidence of heart disease. When followed many of these develop Mobitz Type II partial heart block and complete AV block with Adams-Stokes attacks. The aetiology in this group is bilateral bundle-branch fibrosis of uncertain causation, and Rosenbaum et al. (1968) have termed this syndrome 'Lenegre's disease', since the pathology was first clearly described by Lenegre (1964). There is a further group of elderly patients, usually over the age of 70, with right bundle-branch block and left anterior hemiblock, also with no other clinical evidence of heart disease, who, unlike patients with Lenegre's disease, rarely develop AV block. The cause in this group is sclerosis of structures adjacent to the conduction system and since this was first described by Lev (1964), Rosenbaum et al. (1968) have termed this group 'Lev's disease'. Precise differentiation between these two eponymous syndromes must clinically be largely retrospective.

**Right bundle-branch block with left posterior hemiblock**

Right bundle-branch block with left posterior hemiblock is a less common association of block of two of the three main fascicles. It may occur during acute myocardial infarction or it may be encountered in patients with no other evidence of heart disease. Rosenbaum et al. (1970a) have emphasized its importance since it almost always progresses to complete heart block with Adams-Stokes attacks. They give the following electrocardiographic criteria for its recognition. It should be considered whenever right bundle-branch block is associated with an A QRS of $+120^\circ$ with an S1Q3 pattern provided right ventricular hypertrophy or a vertical heart with emphysema can be excluded. The diagnosis is strongly supported by the presence of tall R waves in leads II and III. In sequential electrocardiograms right bundle-branch block may come and go and left posterior hypertrophy may alternate with left anterior hemiblock with a changing PR interval. In patients with no other evidence of heart disease, these findings strongly suggest the presence of bilateral bundle-branch block due to Lenegre's disease. Other causes are coronary artery disease (i.e. acute myocardial infarction) or cardiomyopathies.

In Rosenbaum et al.'s (1970a) experience about 5 to 10 per cent right bundle-branch block with left anterior hemiblock eventually develop complete heart block whereas the combination of right bundle-branch block and left posterior hemiblock constitutes the QRS pattern most consistently heralding the development of complete heart block.
Narrow ventricular ectopic beats
It is a well-recognized electrocardiographic finding that the QRS-T complexes of beats of apparently AV junctional origin may have a different contour from sinus beats. Such differences in contour must represent a change in the order of the spread of excitation to different parts of the ventricles. Various explanations for this have been suggested. Pick (1956) suggested that the aberrant contour resulted from an eccentric location of the AV junctional pacemaker, the impulse of which reached the ventricles through the 'paraspecific' or 'preferential' pathways of Mahaim instead of by the normal conduction pathway. Since Mahaim's fibres connect the AV node direct to parts of the ventricle, bypassing the bundle of His, this explanation was plausible, until it was shown that, contrary to classic belief, the AV node was largely devoid of pacemaking cells (Matsuda, Hoshi, and Kameyama, 1958; Sano et al., 1958; Hoffman and Cranefield, 1960). Preferential conduction over Mahaim fibres cannot therefore explain aberrant conduction of AV junctional beats. Rosenbaum et al. (1970b) have suggested that in fact these aberrant beats are actually ventricular in origin and that they arise from the proximal parts of the three main conducting fascicles. In support of this hypothesis they point out that such aberrant beats show a relatively small, constant, and well-defined group of QRS complexes, which appear to be either incomplete left bundle-branch block or else incomplete right bundle-branch block with either left anterior hemiblock or left posterior hemiblock. Fig. 2 indicates the sites from which such ectopic beats may arise. Since they will use fast conduction pathways, the QRS complexes will be little if at all widened, but the usual order of excitation of the ventricles will be altered and the resulting beats will appear aberrant. The frequent occurrence of fusion beats strongly supports that such ectopic beats are ventricular in origin and, while the hypothesis waits final experimental proof, it seems the most intellectually satisfying explanation so far put forward for this common electrocardiographic finding.

Electrode catheter recordings from the AV conduction system
Until a few years ago, knowledge of normal and abnormal AV conduction in man was based on electrophysiological studies carried out in the experimental animal and by deductions made from clinical records. Giraud, Puech, and Latour (1960) and Watson, Enslie-Smith, and Lowe (1967) had recorded electrical activity from the AV conduction system in man during catheterization of abnormal hearts. However, Scherlag et al. (1969) introduced a new technique for recording depolarization of the bundle of His in intact man by an electrode catheter. Briefly, a bipolar electrode catheter is introduced percutaneously into the femoral vein and is fluoroscopically positioned across the tricuspid valve. The proximal terminals of the electrode catheter are fed into the AC input of an electrocardiographic amplifier to record bipolar electrograms from the conducting tissues simultaneously with a surface lead, usually either lead II or VI. After introduction, the electrode is slowly withdrawn across the tricuspid valve until a rapid biphasic (or triphasic) deflection is recorded between the atrial and ventricular electrogram and within the PR interval of the surface electrocardiogram. This deflection indicates depolarization of the bundle of His. Records are made at paper speeds of between 100 and 200 mm/sec. Measurements of the intervals of such records are recorded in milliseconds. Later, Damato et al. (1969a), using a multipolar catheter with six electrodes, were able to record and identify waves due to depolarization of the AV node (N deflection) and the right bundle-branch in addition to the His bundle deflection. The His bundle deflection (H) enables the PR interval to be divided into two parts; first from the onset of atrial activity to the H deflection, which largely reflects conduction time through the AV node, and second the interval between the H deflection and the onset of the QRS complex, which measures conduction time from the bundle of His to the onset of ventricular activation. The first interval is variously called the PH interval (or AH interval) and the second is termed the HG interval (or HV interval). In many investigations on human AV conduction a bipolar pacing catheter has been introduced via an arm vein to enable the atrium to be paced at varying rates. Damato et al. (1969b) reported a physiological study of human AV conduction using a His bundle electrode (HBE) technique. They found that in normal subjects the HG interval remained constant, whereas the PH interval was altered by any procedure which altered the PR interval. For example, increasing the heart rate by right atrial pacing progressively increases the PH interval while the HG interval remains constant. At any given paced heart rate, digitalis lengthens the PH interval whereas isoprenaline or atropine shortens it. None of these drugs change the HG interval.

Recently, Rosen et al. (1971) have investi-
gated in humans bundle-branch and ventricular activation in both ventricles, by simultaneously recording His bundle electrograms with right bundle-branch electrograms and electrograms from the left bundle by means of a tripolar catheter introduced across the aortic valve. They showed that in man the proximal parts of the right and left bundle-branches were activated almost simultaneously, but that ventricular activation actually starts in the left midseptal zone.

**His bundle electrograms in AV block**

It is now possible to study in man the site of conduction delay or block in disease by His bundle electrograms. These show that in the majority of cases of first degree AV block the lengthened PR interval is due to a lengthened PH interval without change in the HQ interval, thus reflecting disease in the AV node.

It has been known for many years that second degree AV block occurs in two different forms which have quite different physiological behaviour; they are termed Mobitz Type I and Mobitz Type II second degree AV block, respectively.

In Mobitz Type I or the Wenckebach phenomenon there is progressive prolongation of the PR interval in successive cycles until the P wave is blocked. Damato and Lau (1970) have shown by His bundle electrograms that the PH interval is progressively prolonged until the P wave is no longer followed by an H deflection and ventricular activation fails to occur, indicating that the failure of transmission is in the AV node. In the less common Mobitz Type II, the PR interval is fixed and failure of transmission to the ventricles occurs suddenly. Watanabe and Dreibus (1967) suggested as the result of experiments in the isolated rabbit heart that failure of conduction of Mobitz Type II block occurred in or below the His bundle. His bundle electrograms have fully confirmed that this is true in humans, for PH intervals and HQ intervals for conducted beats remain constant until sudden failure of transmission to the ventricles occurs and while the PH interval remains normal no QRS follows the H deflection when a P wave is blocked. When 2:1 AV block occurs, His bundle electrograms may be essential to distinguish between Mobitz Type I and Mobitz Type II block. When second degree AV block is associated with block of either the left or right main bundle, bilateral bundle-branch block should be suspected.

In chronic complete AV block, Damato and Lau (1970) have shown in all 10 cases studied that the non-conducted P waves were all followed by an H deflection, whereas the widened QRS complexes of the slow idioventricular rhythm were not. These findings offer electrophysiological confirmation of the histopathological studies of Mahaim (1931), Yater (1938), Lenegre (1964), and Davies and Harris (1969) that the majority of cases of chronic complete AV block are due to bilateral bundle-branch disease. These observations do not, of course, apply to all cases of complete AV block, particularly those with QRS complexes of normal duration, such as occur in congenital heart block or heart block complicating inferior myocardial infarction.

**AV junctional rhythm**

Until comparatively recently what we now call AV junctional or junctional rhythms, tachycardias, or beats were termed ‘nodal’. Zahn (1913) originally divided these into upper, middle, and lower ‘nodal’ according to whether the retrograde P’ wave preceded, was embedded in, or followed the QRS complex. Though it was implied that the AV node contained upper, middle, and lower groups of pacemaking cells, it was recognized that the relative speed of antegrade and retrograde conduction of impulses from these groups of cells would also help to determine the site of the retrograde P’ wave in relation to the QRS in the clinical electrocardiogram.

It has already been mentioned that modern electrophysiological studies have failed to confirm the presence of pacemaking cells in the AV node, except perhaps in the NH region where it joins the His bundle. For this reason most authors have dropped the term ‘nodal’ in favour of AV junctional or junctional. Damato and Lau (1969) using His bundle electrograms have studied cases of ‘nodal’ rhythm and AV dissociation, and claim to have located the pacemaker controlling the ventricles in the bundle of His, suggesting the term ‘His bundle rhythm’. Perhaps their most convincing evidence is that the H deflection in the His bundle electrogram in ‘lower nodal’ rhythm precedes both the QRS complex and the N deflection due to depolarization of the AV node. In AV dissociation, atrial activity is followed by an N deflection from the AV node, while the H deflection is linked to the QRS, which, except during ventricular captures, is independent in time.

Many other advances have followed the ability offered by this new technique for recording from the AV conduction system. For example, Damato and Lau (1970) have shown that in atrial flutter with 2:1 AV block, the non-conducted P wave is not followed by an H deflection, thus showing that failure of conduction occurs in the AV node. Similarly
in atrial fibrillation the ‘zone of concealment’ in digitalized patients is shown to lie in the AV node.

**Pseudo AV block**

From time to time records are encountered in which, during apparently normal sinus rhythm, the PR interval is suddenly prolonged and then promptly returns to its previous value, or second degree AV block may occur with failure of conduction of a P wave. It had first been suggested by Langendorf and Mehlman (1947) that this might be caused by concealed junctional extrasystoles, which, though failing to reach either the atria or ventricles, rendered the AV junction relatively or completely refractory to the next sinus impulse. Rosen, Rahimtoola, and Gunnar (1970) have now demonstrated by His bundle electrograms that this explanation is correct and have termed this situation ‘pseudo AV block’.

**Wolff-Parkinson-White syndrome**

There is still some controversy as to whether the pre-excitation complex of the Wolff-Parkinson-White syndrome represents a fusion beat between an impulse reaching the ventricles by a fast accessory pathway and the normal impulse using the slower normal AV route. Attempts to study this problem have been made using His bundle electrograms. Castillo and Castellanos (1970) have shown that in the pre-excitation syndrome the delta wave precedes the H deflection in the His bundle electrogram, thus suggesting a fusion beat, but they admit that their findings do not disprove the concept of synchronized sinus-ventricular conduction of Sherf and James (1969).

**Distinction of aberrant ventricular conduction from ventricular ectopy**

The importance of distinguishing between supraventricular beats and rhythms with aberrant ventricular conduction from ventricular ectopy has already been stressed. Though differentiation can often be deduced from a right chest lead, it is never possible to be 100 per cent certain. Recording of His bundle electrograms may offer this certainty, for if the H deflection is related to and precedes the QRS complex, a supraventricular origin is proved. On the other hand, if the H deflection is buried in or follows the QRS complex, this demonstrates a ventricular origin. Illustrative records have been published by several authors, including Puech *et al.* (1970) and Damato and Lau (1970).

Clearly direct recordings from the bundle of His in man have great potential, both for research into arrhythmias and in routine diagnosis. Unfortunately the technique demands specialized equipment and some expertise from the operator. It would obviously be a great advance if a way could be found to record the H deflection from the surface electrocardiogram. Since this can sometimes be done in children (though not at present in adults) it is at least an ideal worth striving for.

**Mechanism of supraventricular tachycardia**

For many years it has been generally accepted that recurrent attacks of supraventricular tachycardia are due to an ectopic pacemaker, situated either in the atria or the AV junction, which has suddenly acquired a rapid rate of spontaneous diastolic depolarization. Very similar attacks of supraventricular tachycardia are a common feature of the Wolff-Parkinson-White syndrome, and there is now much evidence that these are due to a circus movement involving two pathways connecting the atria and ventricles. The cardiographic features of WPW are generally believed to be due to an accessory AV pathway connecting the atria and ventricles, probably most often a bundle of Kent, in the right or left atrioventricular groove. Conduction in the accessory pathway is faster than in the normal AV pathway resulting in a short PR interval and pre-excitation of part of the ventricle. The initiating beat of an attack of supraventricular tachycardia in WPW usually does not show pre-excitation, indicating that for some reason the accessory pathway was temporarily refractory. By the time the normal sinus impulse has reached the ventricle, the accessory pathway has become excitable again and conducts the impulse in a retrograde direction back to the atria. In this way a circus movement or reciprocating tachycardia is initiated, the QRS complexes not showing pre-excitation. Strong support for this view has been produced by many workers, including Durrer *et al.* (1967), who were able to initiate or terminate attacks of supraventricular tachycardia in patients with the Wolff-Parkinson-White syndrome, by appropriately timed electrical stimuli delivered via an intracardiac pacing catheter to either the atria or the ventricles.

A similar mechanism for attacks of supraventricular tachycardia in the absence of WPW was originally suggested by Iliescu and Sebastiani (1923) and this mechanism was strongly supported by Barker, Wilson, and Johnston (1943). However, this view gained little credence, presumably because only one pathway was known to connect the atria and
ventricles in subjects without the WPW syndrome.

Moe, Preston, and Burlington (1956) produced strong experimental evidence in animals for the presence of a dual AV conduction system. They suggested that the fibres of the AV node could be longitudinally dissociated at least in its upper part, the pathways uniting in the lower node to form a final common pathway. If this were true in humans, the anatomical basis would exist for reciprocating tachycardia apart from the WPW syndrome. Indeed the occurrence of reciprocal beats and reciprocal rhythms in clinical tracings demands the presence of two AV pathways to explain their occurrence (Schamroth and Yoshonis, 1969).

Many authors have recently advanced both clinical and electrophysiological evidence that the underlying mechanism in many cases of supraventricular tachycardia is a reciprocating tachycardia, involving two separate pathways in the AV node. For example, in a study of six successive cases of supraventricular tachycardia, Bigger and Goldreyer (1970) found, using sophisticated electrophysiological techniques, that a re-entry mechanism for the attacks rather than a rapidly discharging focus was the only acceptable explanation. More recently (Goldreyer and Bigger, 1971) the same authors have shown, using His bundle electrograms, that the site of re-entry is the AV node.

The existence of two separate pathways could only become manifest if they had different physiological properties. It is simplest to envisage them as having different refractory periods. A premature supraventricular beat, such as an atrial extrasystole, may find one pathway in a relative refractory phase and the other still in the absolute refractory phase. The impulse will then be conducted slowly along the relative refractory path and reach the final common pathway at a time when the second pathway has recovered excitability. This excitation wave will then divide into two fronts, one completing the journey to the ventricles down the final common pathway and the other returning to the atria by the second pathway. Such an event occurring once is known as an ‘atrial echo’. Alternatively, a circus movement may be started, resulting in a reciprocating tachycardia.

One would therefore expect that supraventricular tachycardia should start with an atrial extrasystole falling in the relative refractory period and having a long P'R interval. In practice this is found to be invariably so, whereas atrial extrasystoles (in the same patients) falling outside the relative refractory period do not initiate supraventricular tachycardia. Moreover, the first few cycles of supraventricular tachycardia alternate in length, as would be expected from a reciprocating mechanism, but not from a rapidly discharging ectopic focus.

A final point, which may well have therapeutic implications, is that Bigger and Goldreyer were able at will both to initiate and terminate attacks of supraventricular tachycardia in their patients by a single appropriately timed electrical stimulus to the atrium.

References


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