Studies of heart block with His bundle electrograms

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A total of 24 patients with heart block revealed by scalar electrocardiography was studied using the His bundle electrogram. Four groups of disturbed conduction are described which include various types of heart block, partial and complete, at different sites, single and multiple, in the specialized conducting system. These are (1) intra-atrial block with delayed or blocked internodal conduction, (2) atrioventricular nodal block due to delayed or blocked conduction in the AV node, (3) His-Purkinje-system block due to delayed or blocked conduction in the fibres of the His-Purkinje-system, (4) multiple site AV block due to disturbances of conduction in the AV node and His-Purkinje-system in combination. His-Purkinje-system block is characterized by associated intraventricular block, and its relation to bilateral bundle-branch block and trifascicular block is discussed. In some instances concealed conduction disturbance, both anterograde and retrograde, is revealed by the His bundle electrogram, including facilitation of conduction in the main bundle of His. The His bundle electrogram defines more clearly the nature and extent of disturbed conduction and it may contribute to the choice of treatment, either by drugs or by artificial pacing, temporary or permanent.

The atrioventricular conduction tissues have two fundamental properties, the propagation of impulses and the formation of abnormal beats. Both properties are interdependent and influence each other. They have been extensively studied in animals using microelectrode techniques to measure transmembrane action potentials of myocardial fibres and of the specialized conducting tissues (Alanís, González, and López, 1958; Alanís *et al.*, 1959; Hoffman *et al.*, 1963). More recently, extracellular potentials generated by depolarization of the atrioventricular conduction tissues during the passage of impulses have been recorded in man by the technique described by Scherlag *et al.* (1969). Thus, disturbances of impulse formation and of impulse conduction can be studied more precisely under clinical conditions than has been possible hitherto. The method, which is simple in a properly equipped laboratory and is without undue risk or discomfort to the patient, has been employed in the study of atrioventricular block and the tachyarrhythmias and in measurements of normal sinus conduction (Damato *et al.*, 1969; Narula *et al.*, 1970b; Narula and

Samet, 1970; Narula *et al.*, 1971; Castillo and Castellanos, 1970; Bekheit *et al.*, 1971).

This paper is concerned with the study of abnormal conduction and impulse formation in the atria and junctional tissues. The use of the His bundle electrogram permits more precise location of the site of impulse delay than is possible with standard surface electrocardiograms, and the mechanisms involved can be shown more accurately. These factors are of great physiological importance and point to the need for revision of current concepts of interpretation of Wenckebach block (Mobitz I) and Mobitz II block, based on duration of refractoriness (Wenckebach, 1899, 1906; Mobitz, 1924). The His bundle electrogram shows that multiple sites of block of varying degrees can occur in the conduction system (Narula *et al.*, 1970b) and, moreover, as we will show, it indicates that conduction disturbances concealed in standard leads may become revealed. There is necropsy evidence that Adams-Stokes attacks are particularly associated with isolated disease in the fibres of the His-Purkinje-system (Davies and Harris, 1969). The His bundle electrogram permits location of the site or sites of such disease, and thereby may have practical clinical application, for example when surface leads show trifascicular block (Rosenbaum, Elizari, and

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TABLE Classification of heart block

Type of heart block	Case No.	Sex and age	Disease	Syncope	Standard electrocardiogram			Bundle of His electrogram		
					PR (msec) (normal < 200)	QRS (msec) (normal < 100)	QRS axis	PA (msec) (normal 10-45)	AH (msec) (normal 50-125)	BH (msec) (normal 15-25)
GROUP I										
Intra-atrial block (PA)										
(A)										
1st-degree	1	F 48	Sarcoidosis	—	250-260	70	+ 58°	70-85	125-130	20
GROUP II										
AV nodal (AH) block										
(A)										
1st-degree	2	F 52	Idiopathic heart block	+	250-265	80	+ 40°	40	170-180	20
	3	F 64	Coronary heart disease	+	285-290	80	+ 12°	30-40	215-220	20
	4	F 46	Idiopathic heart block	+	220-230	70	+ 68°	20-30	150-160	15
	5	M 58	Coronary heart disease	+	330-340	80	+ 90°	10-15	275-280	15
(B)										
With blocked beats partial/complete	6	M 52	Idiopathic heart block	—	345-505	80	+ 30°	35	270-430	20
	7	F 80	Idiopathic heart block	+	225-230	80	- 30°	25	175-180	20
GROUP III										
His-Purkinje-system (HV) block										
(A1)										
1st-degree normal PR	8	M 69	Coronary heart disease	+	130-140	120	+ 61°	5	60-70	20
	9	M 50	Cardiomyopathy	—	170-180	70	- 75°	20-30	70-80	15
	10	F 67	Coronary heart disease	+	190-200	110	- 28°	15-20	110-120	20
	11	M 63	Coronary heart disease	+	175-185	140	- 10°	35-40	75-80	15
	12	M 57	Coronary heart disease	+	190-195	140	- 15°	30-40	80-90	20
	13	M 54	Coronary heart disease	—	170-180	110	+ 60°	20-30	70-80	25
	14	M 69	Coronary heart disease	—	190-200	105	- 34°	10-20	100-110	20
(A2)										
1st-degree prolonged PR	15	M 52	Coronary heart disease	—	220-230	170	- 60°	30-40	100-105	20
(B)										
Blocked beats partial/complete	16	F 62	Idiopathic heart block	+	185-190	160	- 34°	25-35	75-85	15
	17	M 63	Idiopathic heart block	+	190-315	160	- 55°	25-30	65-75	15
GROUP IV										
Combined AV nodal + His Purkinje (AH + HV) block										
	18	F 67	Idiopathic heart block	+	—	120	+ 54°	10	175-180	45-65
	19	M 19	Aortic and mitral valve disease	—	230-240	90	S ₁ S ₂ S ₃	20-25	140-150	25
	20	M 69	Coronary heart disease	—	230-240	110	- 50°	20-30	130-140	15
	21	M 66	Coronary heart disease	+	225-235	110	- 50°	25-35	130-140	15
	22	M 56	Coronary heart disease	—	250-260	140	- 40°	10-20	170-180	15
	23	M 54	Coronary heart disease	—	220-230	80	S ₁ S ₂ S ₃	20-30	140-145	15
	24	M 58	Coronary heart disease and cor pulmonale	—	215-225	100	+ 138°	20-30	135-145	20

		Remarks
HV (msec) (normal 35-45)	Abnormal beats	
50-55	-	—
45	+	Concealed conduction; isoprenaline
35	+	Concealed conduction; isoprenaline
40	+	—
45	-	—
40	-	Wenckebach block; isoprena- line
40	+	2:1 block; concealed conduc- tion
70	-	Right bundle-branch block
80	-	Left anterior hemiblock; digoxin
65	-	Left bundle-branch block; digoxin
65	-	Left bundle-branch block; digoxin
70	-	Left bundle-branch block; digoxin
70	-	Intraventricular block; digoxin
80	-	Left bundle-branch block
85	+	Left bundle-branch block
80	-	Left bundle-branch block; Mobitz II pacemaker
100-210	-	Right bundle-branch block; left anterior hemiblock; 2:1 and 3:1 block
—	+	Complete heart block; AH 1st-degree; right bundle- branch block
70	-	AH 1st-degree; HV MPS 1st-degree
70	-	AH 1st-degree; HV 1st-degree; left anterior hemiblock
60	-	AH 1st-degree; HV 1st-degree; left anterior hemiblock
70	-	AH 1st-degree; HV 1st-degree; left bundle-branch block
60	-	AH 1st-degree; HV 2:1 block
60	+	AH 1st-degree; HV 1st-degree; right bundle-branch block

Lazzari, 1970) or suggest bilateral bundle-branch block (Lenegre, 1964; Schuilenburg and Durrer, 1970). This study shows first, disturbances of conduction of sinus impulses due to block, single and multiple, partial and complete in the intra-atrial and junctional tissues, and second it is concerned with concealed conduction disturbances both antero-grade and retrograde, all of which are revealed by the His bundle electrogram. Before presentation of our results the His bundle electrogram will be described briefly.

There is good evidence that the sinus impulse reaches the AV node by internodal preferential pathways (James, 1963) and enters the AV node from the right atrium (Scherf and Cohen, 1964). Thereafter the sinus impulse is normally delayed in some unknown way, probably the result of varying duration

FIG. 1 (Table, Case 1.) First-degree block (Group I, A). The PR interval is prolonged, 250-260 msec. Delayed conduction in the internodal fibres is the chief contributory factor, as the PA interval is conspicuously prolonged to 70-85 msec (normal ≤ 45 msec). AV nodal and His-Purkinje-system conduction were just above upper limits for normal values for AH interval (normal ≤ 125 msec), and HV intervals, 50-55 msec (normal 35-45 msec). Distance between two light vertical lines 40 msec. The standard leads showed no abnormality apart from first degree AV block. Records from a woman aged 48 years with advanced sarcoidosis.

of action potentials of the atrial fibres immediately adjacent to the AV node (Scherf and Cohen, 1964). Conduction in the AV node and the His-Purkinje-system is myogenic, though in the former it is influenced by the autonomic nervous system. The His bundle electrogram is recorded from an electrode catheter placed astride the atrioventricular conduction fibres. It is so called because of the prominent potential produced by the main bundle of His (BH wave) either as a biphasic or triphasic deflection. The initial deflections (A wave) represent depolarization of atrial fibres immediately adjacent to the AV node, and the interval between the first rapid deflection of the A wave and the beginning of the BH wave is a measurement of conduction time in the AV node (Hoffman *et al.*, 1963). Depolarization of the AV node itself may be marked by a notched deflection (N wave) near the termination of the A wave (Damato *et al.*, 1970), but in our tracings this could not be

identified with certainty. The BH wave is followed by rapid deflections of initiation of ventricular depolarization (V wave) and the interval between the beginning of the BH wave and the first rapid deflection of the V wave represents conduction time through the main bundle of His to the Purkinje myocardial junction. If the electrode catheter is advanced too far into the right ventricle depolarization of the right bundle-branch may be recorded (RB wave). It may simulate the BH wave but can be differentiated readily from it by pacing the atrium or main bundle of His (Narula, Scherlag, and Samet, 1970c). The nomenclature of the deflections of the His bundle electrogram has not been formalized as yet and the symbols employed in this paper are those used by the pioneer workers already mentioned. In summary, the His bundle electrogram measures AV node conduction (AH interval) and His-Purkinje-system conduction (HV interval) and when correlated

FIG. 2 (Table, Case 3.) First-degree AV nodal block (Group II, A). The PR interval in scalar lead is prolonged, 285–290 msec. The AH interval is abnormally prolonged to 215–220 msec (normal, ≤ 125 msec). Intra-atrial conduction, PA interval, 30–40 msec, is normal (≤ 45 msec). His-Purkinje-system conduction is also normal, HV interval 35 msec (normal, 35–45 msec). Delayed conduction occurred exclusively in the AV node. Distance between two light vertical lines 40 msec. The standard leads showed low T wave voltage with first-degree AV block. Tracings recorded from a woman aged 64 years with coronary disease.

with a surface lead, the distance between the beginning of the P wave of the standard electrocardiogram and the beginning of the A wave of the His bundle electrogram (PA interval) is a measurement of internodal atrial conduction.

Subjects and methods

A total of 24 patients was studied, 17 men and 7 women (age range 19 to 80 years). All of them had organic heart disease. Coronary artery disease was present in 14 patients and rheumatic heart disease in one. Seven patients were considered to have isolated fibrosis of the specialized conducting tissues and the remaining 2 had congestive cardiomyopathy and advanced sarcoidosis, respectively. The standard electrocardiograms were abnormal in all of them, and in particular showed either atrioventricular block or intra-ventricular block, or both types of conduction disturbance. The clinical data, electrocardiographic findings, and the measurements determined from the His bundle electrograms are summarized in the Table.

The details of the method by which the His bundle electrograms were recorded have been described by us previously (Bekheit *et al.*, 1971). Long strips of not less than one hundred cycles were recorded at a paper speed of 100 mm a second from which the measurements were made. The normal values for the intervals PA (25–45 msec), AH (50–125 msec), BH (15–20 msec), and HV (35–45 msec) were used for controls in our measurements (Narula *et al.*, 1970a; Bekheit *et al.*, 1971). Selected tracings are illustrated in Fig. 1–13.

Results: classification of abnormal conduction

The His bundle electrograms record abnormal conduction under resting conditions without any form of intervention, and they illustrate various types of heart block, partial and complete, at different sites in the conducting system both single and multiple. For purposes of description the various types of block have been divided into four main groups according to the site of block. It is suggested, however, that such a classification may have practical clinical significance in addition to allowing a more precise understanding of the mechanisms of abnormal conduction.

Group I: Intra-atrial block with delayed or blocked internodal conduction (Table, Case 1) The conduction disturbance may represent abnormal delay in the preferential internodal pathways occurring simultaneously. The sinus impulse may be delayed causing a prolonged PA interval (type A) or it may be blocked (type B). An example of type A intra-atrial block only is available in our series.

FIG. 3 (Table, Case 6.) AV nodal Wenckebach block (Group II, B). The bundle of His tracings are continuous. Note progressive prolongation of AH interval from 270–430 msec. His-Purkinje-system conduction remains normal, 40 msec (normal, 35–45 msec). The blocked beat (BA) is not followed by a BH wave, indicating that the site of impaired conduction and block is exclusive to the AV node. Distance between two light vertical lines 40 msec. Standard leads were normal apart from AV block. The tracings are from a man aged 52 years without definite cardiac symptoms.

First-degree intra-atrial block with delayed internodal conduction, Fig. 1 (Table, Case 1) The tracing was recorded from a woman aged 48 years with advanced sarcoidosis. The surface electrocardiogram showed a prolonged PR interval (250–260 msec). The bundle of His electrogram revealed prolongation of the PA interval from 70–85 msec (normal 25–45 msec) with normal conduction in the AV node

FIG. 4 (Table, Case 7.) 2:1 AV nodal block (Group II, B). The PR interval of the conducted beats is prolonged to 245 msec. The His bundle electrogram confirms that the delayed conduction is exclusive to the AV node, as the AH interval is abnormally prolonged to 180 msec (normal ≤ 125 msec). The blocked beats (BA in His bundle electrogram) are not followed by a BH wave. The impulse is therefore blocked in the AV node. His-Purkinje-system conduction is normal, HV interval 40 msec (normal, 35-45 msec). Distance between two light vertical lines 40 msec. Tracings recorded from a woman aged 80 years.

(AH interval 125 msec) and a slightly prolonged HV interval, 50-55 msec (normal 35-45 msec). This is explained by delay in the conduction of the sinus impulse through the internodal fibres. The slight prolongation of the HV interval denotes, in addition, a delay in conduction in the main bundle of His.

Group II: Atrioventricular nodal block (Table, Cases 2-7) In atrioventricular nodal block the delayed or blocked conduction is confined to the AV node. The His bundle electrogram does not discriminate between

abnormal conduction located at the atrio-nodal junction (N region) and the junction of the distal end of the AV node with the main bundle of His. It is convenient to subdivide AV nodal block into two types, (A) first-degree AV nodal block, characterized by prolongation of the AH interval, and (B) AV nodal block with blocked impulses, partial or complete, in which the blocked impulses are not followed by a BH wave.

(A) *First-degree AV nodal block* Four patients had conduction disturbances of this

type (Table, Cases 2-5). All of them had a history of syncopal attacks, suggesting that partial or complete AV nodal block may have occurred intermittently. The characteristic features were (1) prolonged PR interval in the surface leads greater than 200 msec (range 220-340 msec); (2) prolonged AH interval greater than 120 msec (range 155-280 msec); (3) normal BH, HV, and QRS intervals. The QRS axis of the surface electrocardiograms ranged from $+12^\circ$ to $+90^\circ$ consistent with normal conduction in the distal part of the His-Purkinje-system. The tracing (Fig. 2, Case 3) was recorded from a woman aged 64 years with syncopal attacks. The PR interval of the surface electrocardiogram was prolonged to 290 msec. The His bundle electrogram revealed that the AH interval was prolonged to 220 msec. The PA, BH, HV, and QRS intervals were normal. Thus, the His bundle electrogram showed that the prolonged PR interval was entirely due to delayed conduction in the AV node.

(B) Atrioventricular nodal block with blocked sinus impulses Two patients had conduction disturbances of this type (Table, Cases 6 and 7). When sinus beats were conducted the criteria for diagnosis were the same as in Group II (A), but when they were blocked the A waves were not followed by BH waves. This type of block includes varying degrees of partial heart block, including Wenckebach block (Mobitz I block). The essential facts are that the AV node is the site of block and conduction is intact in the His-Purkinje-system. An example of Wenckebach block, Fig. 3 (Table, Case 6) was recorded from a man aged 52 years. A long strip from lead II of the surface electrocardiogram showed a prolonged PR interval and episodes of Wenckebach block (Mobitz I block). The PR interval varied from 280-505 msec in the conducted sinus beats. The His bundle electrogram showed prolongation of the AH interval within the range 230-420 msec. The HV intervals of the conducted sinus beats were normal. Blocked sinus impulses were not followed by BH waves. The His bundle electrogram confirmed, therefore, that the Wenckebach block was located entirely in the AV node, and the His-Purkinje-system conduction was normal.

An example of 2:1 AV nodal block (Fig. 4) was recorded from a woman aged 80 years (Case 7) who had a history of syncopal attacks over a period of two years. Surface electrocardiograms indicated varying degrees of AV block. The conducted sinus beats had a prolonged PR interval (225 msec). Fig. 4 shows a His bundle electrogram recorded during a

FIG. 5 (Table, Case 9.) First-degree His-Purkinje-system block with normal PR interval (Group III, A1). The PR interval measures 180 msec. Intra-atrial conduction and AV nodal conduction are normal, PA interval 30 msec, and AH interval, 70-80 msec. The HV interval is prolonged to 80 msec (normal 35-45 msec), confirming delayed conduction in His-Purkinje-system. Distance between two light vertical lines 40 msec. The standard leads show left anterior hemiblock (-75°). Tracings were recorded from a man aged 50 years with congestive cardiomyopathy.

static phase of 2:1 AV block. The AH interval of the alternate conducted sinus beats was prolonged to 180 msec but their HV interval was normal. The A waves of the blocked sinus impulses were not followed by BH waves. Thus, the delay and block in conduction was located in the AV node.

Group III: His-Purkinje-system block (Table, Cases 8-17) In His-Purkinje-system block the delay or blocked conduction is confined to the main bundle of His, or its bundle-branches and their ramifications. It is of topical interest in view of its relation to bilateral bundle-branch block as a common

FIG. 6 (Table, Case 15.) First-degree His-Purkinje-system block with prolonged PR interval and complete left bundle-branch block (Group III, A2). The PR interval is prolonged to 220 msec. Intra-atrial and AV nodal conduction are normal, the PA interval, 30–40 msec (normal ≤ 45 msec), and AH interval, 100–105 msec (normal ≤ 125 msec). The prolonged HV interval, 85 msec (normal, 35–45 msec), confirms that there was delayed conduction in the His-Purkinje-system. Distance between two light vertical lines 40 msec. The standard leads also confirm abnormal conduction in the left bundle of His. The tracings were recorded from a man aged 52 years with coronary heart disease.

mechanism of complete heart block (Lenegre, 1964) and the significance of the hemiblocks as indicators of disease of the Purkinje fibres (Rosenbaum, Elizari, and Lazzari, 1968). Our cases had either right or left bundle-branch block with a prolonged QRS interval, or left anterior hemiblock when the QRS interval was normal. As in Group II, it is convenient to subdivide the His-Purkinje-system block into two types: (A) first-degree His-Purkinje-system block characterized by prolongation of the HV interval, and (B) His-Purkinje-system block with blocked sinus impulses which includes various types of partial and complete heart block in which the blocked impulses are not followed by V waves.

(A) *First-degree His-Purkinje-system block* (Table, Cases 8–15) The characteristic features were (1) prolonged HV interval greater than 45 msec (range 65–90 msec), (2) normal PA and

AH intervals, and (3) intraventricular block either right or left bundle-branch block, left anterior hemiblock, or indeterminate intraventricular block. The implication of intraventricular block is that the disease process which delays His-Purkinje-system conduction extends proximally to involve the main bundle or the contralateral branch. However, if the His-Purkinje-system block is due to an isolated lesion of the main bundle of His, intraventricular conduction may be normal and the block may be revealed by changes in the duration of the BH wave which may be duplicated (BH and BH waves, Narula *et al.*, 1970a).

Before describing our tracings, some comments on the duration of the PR interval may be useful. The PR interval of conducted beats in the electrocardiogram comprises the PA, AH, and HV intervals of the His bundle electrogram. Assuming minimal values of 25 msec for the PA interval and 50 msec for the

FIG. 7 (Table, Case 16.) Partial His-Purkinje-system block (Mobitz II) with complete left bundle-branch block (Group III, B). Tracings recorded with a pacemaker in situ (Cordis standby pacemaker). Records are from a woman aged 62 years with a history of syncopal attacks. The PR interval of conducted beats is normal, 180–190 msec. The HV interval is prolonged to 80 msec (normal, 35–45 msec), indicating delayed conduction in His-Purkinje-system. Intra-atrial and AV nodal conduction are normal. Distance between two light vertical lines 40 msec. The standard leads show complete left bundle-branch block. The last two beats (S) of the second strip represent paced beats. Impaired conduction was exclusively limited to His-Purkinje-system.

FIG. 8 (Table, Case 17.) Partial-His-Purkinje-system block with complete right bundle-branch block and left axis deviation (Group III, B). The blocked beats (BA) in the His bundle electrogram are followed by a BH wave confirming that the site of block is in the His-Purkinje-system. All sinus impulses are conducted normally through the atria and AV node, the PA interval being 20–30 msec (normal ≤ 45 msec), and the AH interval, 65–75 msec (normal ≤ 125 msec). In the conducted beats the HV interval is prolonged from 100–210 msec (Wenckebach His-Purkinje-system block). Distance between two light vertical lines 40 msec. The standard tracings show complete right bundle-branch block with left axis deviation. The tracings were recorded from a man aged 63 years with a history of syncopal attacks.

AH interval, then the HV interval would require to exceed 125 msec to cause prolongation of the PR interval to more than 200 msec in conventional surface tracings ($PR = PA + AH + HV$). It follows, therefore, that when the HV interval exceeds the upper normal value of 45 msec but measures less than 125 msec, first-degree His-Purkinje-system block may often be concealed within the normal PR interval of the scalar electrocardiogram, depending upon the normal range of variation of the PA and AH intervals at the

time the tracing is recorded. Similarly, first-degree His-Purkinje-system block may also be concealed in atrial fibrillation. In the Table, therefore, the cases have been subdivided into those with a normal PR interval, A₁ (Cases 8–14), and those with a prolonged PR interval, A₂ (Case 15).

An example of first-degree His-Purkinje-system block with normal PR interval and left anterior hemiblock (Type A₁), Fig. 5 (Table, Case 9) was recorded from a man aged 50

FIG. 9 (Table, Case 19.) First-degree combined AV nodal and His-Purkinje-system block (Group IV). The PR interval is prolonged, 230–240 msec. Intra-atrial conduction time is normal, PA interval, 20 msec (normal, ≤ 45 msec). Delayed conduction is present in the AV node and His-Purkinje-system, AH interval 140–150 msec (normal ≤ 125 msec), and HV interval, 70 msec (normal, 35–45 msec). Distance between two light vertical lines 40 msec. The standard leads showed $S_1 S_2 S_3$ syndrome and first-degree AV block. The tracings were recorded from a man aged 19 years with rheumatic mitral and aortic valve disease.

years with congestive cardiomyopathy. The standard electrocardiogram showed a PR interval measuring 170 msec with a normal QRS interval of 70 msec and left anterior hemiblock (QRS axis -75°). The His bundle electrogram showed His-Purkinje-system block with prolongation of the HV interval to 70 msec. The PA and AH intervals were normal, measuring 30 and 70 msec, respectively. The His bundle electrogram therefore revealed His-Purkinje-system block which was concealed within the normal PR interval of the surface tracing. In association with left anterior hemiblock this indicates involvement of the specialized conducting tissues, presumably by the pathological process of the cardiomyopathy.

An example of first-degree His-Purkinje-system block with prolonged PR interval and left bundle-branch block (Type A2), Fig. 6 (Table, Case 15) was recorded from a man aged 52 years with coronary artery disease. The standard electrocardiogram showed prolongation of the PR interval to 220 msec and complete left bundle-branch block (QRS interval 170 msec). The His bundle electrogram showed prolongation of the HV interval to 85 msec. The PA and AH intervals were normal. In conjunction with left bundle-branch block the HV interval delay is suggestive of bilateral bundle-branch block.

(B) His-Purkinje-system block with blocked impulses (Table, Cases 16–17) The criteria for

FIG. 10 (Table, Case 2.) First-degree AV nodal block (Group II, A) with concealed conduction in the AV node. The first, third, and fourth complexes are conducted sinus beats. The PR interval is prolonged to 260 msec in beats 1 and 4. The second beat is an abnormal beat preceded by a BH wave but not by an A wave, indicating that its focus of origin is in the junctional tissues. His-Purkinje-system conduction is normal, HV interval, 45 msec. The first-degree block is entirely due to AV nodal delay, AH interval 170 msec (normal ≤ 125 msec). The AH interval in beat 3 is more prolonged to 270 msec due to concealed delayed conduction in the AV node after penetration by the preceding junctional impulse. Distance between two light vertical lines 40 msec.

conducted beats are the same as for Group III (A). Blocked sinus impulses penetrate the AV node, and the A wave in the His bundle electrogram is followed by a BH wave but not by a V wave, i.e. the site of block is in the His-Purkinje-system. When the block is partial and the PR interval is normal, the standard electrocardiogram may correspond to Mobitz II block.

An example of His-Purkinje-system block with blocked sinus impulses and normal PR interval of the conducted beats, Fig. 7 (Table, Case 16), was recorded from a woman aged 62 years with a history of Adams-Stokes attacks over a period of four years. The standard electrocardiogram showed various degrees of AV block with blocked beats and left bundle-branch block (QRS interval 185 msec). The His bundle electrogram was recorded after a permanent inhibited demand pacemaker was *in situ* (Cordis 'Stanicor' blocking standby pacemaker). The HV interval of the sinus beats was prolonged to 80 msec, indicating that the disturbed conduction

was in the His-Purkinje-system. The PA and AH intervals were normal, as was the His bundle duration. When pacemaker beats occurred no BH wave was recorded. The association of syncope with conspicuous prolongation of the HV interval and left bundle-branch block suggests that bilateral bundle-branch block is the mechanism involved.

An example of His-Purkinje-system block with blocked sinus impulses, 2:1 and 3:1 His-Purkinje-system block, Fig. 8 (Table, Case 17), was recorded from a man aged 63 years with syncopal attacks over a period of two months. The electrocardiogram showed right bundle-branch block (QRS interval 160 msec), left anterior hemiblock (QRS axis -55°), and 2:1 and 3:1 AV block. The PR interval of the conducted beats ranged between 190 and 315 msec. Thus, the left posterior division of the main bundle of His was the only pathway by which the sinus impulses could have reached the ventricles. The His bundle electrogram showed prolongation of the HV interval of the conducted beats (range

FIG. 11 (Table, Case 3.) First-degree AH type AV block with concealed conduction disturbance in the AV node and His-Purkinje-system (Group II, A). Long tracings showed sinus arrhythmia with prolonged PR intervals of varying duration. The delayed conduction is due to prolonged AH intervals, 155 msec in first beat and 135 msec in third beat. The second beat is a fusion beat due to synchronization of the sinus impulse with a junctional beat. The junctional impulse activates the ventricles normally but results in increased delay of the sinus impulse in the AV node so that the AH interval is prolonged to 170 msec and complete block of the sinus impulse in the His-Purkinje-system (BH wave). Distance between two light vertical lines 40 msec.

190–210 msec), even when the PR interval was normal, i.e. Wenckebach His-Purkinje-system block was present. The PA, AH, and BH intervals were normal throughout, indicating that conduction within the atria and in the proximal part of the junctional tissues was intact. When the sinus impulses were blocked, the BH deflections were not followed by V waves, confirming that the His-Purkinje-system was the site of complete block. In association with right bundle-branch block and left anterior hemiblock, this indicates that trifascicular block was the mechanism underlying the syncopal attacks.

Group IV: Multiple site AV block (Table, Cases 18–24) When the conducting system is diseased, impaired conduction may occur in the AV node and the His-Purkinje-system

in combination. Group IV therefore includes cases of AV block with various types of AV nodal and His-Purkinje-system block which can only be revealed by the His bundle electrogram. All patients had some form of intra-ventricular conduction disturbance, left bundle-branch block was present in 3, right bundle-branch block in 2, and 2 had $S_1 S_2 S_3$ syndrome.

A tracing was recorded of multiple site block with first-degree AV nodal and His-Purkinje-system block, Fig. 9 (Table, Case 19), from a man aged 19 years with rheumatic aortic and mitral valvular incompetence. In addition, there was calcification of the aortic valve. The standard electrocardiogram showed a prolonged PR interval (230 msec) and the $S_1 S_2 S_3$ syndrome. The His bundle electrogram showed that the first-degree heart block

FIG. 12 (Table, Case 7.) Combined AV nodal block and His-Purkinje-system block (Group IV). The first and second beats represent conducted sinus beats with delay in the AV node, AH interval prolonged to 180 msec (normal ≤ 125 msec). The second ventricular complex shows complete left bundle-branch block. The second blocked P wave is followed by a BH wave, confirming that the impulse has penetrated the AV node and is blocked in the His-Purkinje-system. The third ventricular complex shows a complete left bundle-branch block in spite of long preceding ventricular cycle length, due to effects of penetration of the preceding sinus beat deep into His-Purkinje-system (phase 4 depolarization, see text). Distance between two light vertical lines 40 msec.

was due to a combination of first-degree AV nodal block (AH interval 150 msec) and first-degree His-Purkinje-system block (HV interval 70 msec). Since the aortic valve was calcified, involvement of the conducting pathways by the calcific process may explain the abnormal conduction in the AV node and His-Purkinje-system.

Results: Concealed conduction disturbances

Concealed conduction was first described by Scherf and Shookoff (1925). Since then it has been more widely recognized and its mechanisms better defined (Langendorf, 1948; Langendorf and Pick, 1956; Hoffman, Cranefield, and Stuckey, 1961). The His bundle electrogram may reveal conduction disturbances both anterograde and retrograde in the atrio-ventricular and His-Purkinje-systems, which are concealed on conventional surface tracings. Among the tracings of the 24 patients in this study we found 4 examples of concealed conduction revealed by the His bundle electrogram. Two patients were classified in Group II A (Cases 2 and 3) and one in Group II B (Case 7). The fourth patient was classed in Group IV (Case 18).

First-degree AV nodal block with concealed conduction in the AV node (Fig. 10, Table, Case 2) The tracing was recor-

ded from a man aged 52 years. The His bundle electrogram confirmed that the prolonged PR interval (250–265 msec) of the surface electrocardiogram was entirely due to prolongation of the AH interval (170–180 msec). The HV and QRS intervals were normal. Thus, first-degree AV nodal block was present. When abnormal beats occurred they were preceded by BH waves indicating that their site of origin was in the junctional tissues. The velocity of conduction of the abnormal beats to the ventricles was the same as the sinus beats (HV interval 45 msec), but their ventricular complexes were aberrant due to incomplete recovery of the Purkinje myocardial fibres. The AH intervals of the immediately following sinus beats occurred in time, but with a greatly prolonged AH interval (270 msec). The explanation appears to be that the abnormal impulse was conducted in a retrograde direction from its site of origin and penetrated deeply into the AV node, but was blocked within it and failed to reach the atria. Consequently, the sinus rhythm was not disturbed, but increased delay in AV nodal conduction of sinus impulses immediately following the abnormal beats occurred. Thus, prolongation of the PR interval after the abnormal beats was due to concealed delayed conduction in the AV node which was shown by the His bundle electrogram.

FIG. 13 (Table, Case 18.) Combined first-degree AV nodal block with complete orthograde His-Purkinje-system block and intact retrograde conduction (Group IV). The standard tracing shows complete AV block, atrial rate 100 a minute, ventricular rate, 37 a minute. The His bundle electrogram (continuous strips) confirms that sinus impulses are conducted through the AV node and blocked in the His-Purkinje-system. The AH interval is prolonged to 175–180 msec (normal ≤ 125 msec), i.e. first-degree atrioventricular nodal block (Group II). The third ventricular complex occurs late in atrial diastole and is followed by a BH wave, indicating facilitation of retrograde conduction at the site of the block. The prolonged BH wave, 45 msec, indicates delayed conduction in the main bundle of His. Distance between two light vertical lines 40 msec.

First-degree AV nodal block with concealed conduction in the AV node and His-Purkinje-system block, Fig. 11 (Table, Case 3) The tracing was recorded from a woman aged 64 years with coronary artery disease and syncopal attacks over a period of seven years. The standard electrocardiogram showed sinus arrhythmia with varying duration of the PR interval. Long cycle lengths were terminated by junctional escape beats which frequently synchronized with sinus impulses, giving rise to fusion beats. The QRS interval was normal (80 msec). The His bundle electrogram indicated that the varying PR interval of the conducted sinus beats was entirely due to varying duration of the AH intervals within the range 135–155 msec. The HV interval was normal (35 msec). Thus, first-degree AV nodal block was present and the varying degrees of delayed conduction in the AV node appeared to be related to varying vagal tone associated with sinus arrhythmia. When escape beats occurred they were preceded by BH waves, confirming that their site of origin was in the junctional tissues, and they were conducted to the ventricles with the same HV intervals as the sinus impulses. A rapid deflection was recorded after the V wave of each junctional beat but distinct from it, and though it differed in shape from the BH wave of sinus beats it can reasonably be assumed that it represents depolarization of some part of the His-Purkinje-system (BH wave). On this basis, the following explanation is offered to account for competition of the sinus and junctional impulses for the conduction pathways. The junctional impulse occurred at a critical time when the sinus impulse had already penetrated the AV node but had not gained access to the His-Purkinje-system. It was therefore conducted normally to the ventricles. The junctional impulse was also conducted in a retrograde direction and penetrated the AV node causing delayed conduction of the immediately following sinus impulse so that its AH interval was prolonged to 170 msec, i.e. concealed conduction in the AV node has been revealed. Nevertheless, the delayed sinus impulse passed through the AV node and was blocked in the His-Purkinje-system, so that concealed His-Purkinje-system block was also revealed. The shape and duration of the BH wave suggest delayed conduction in the main bundle of His with complete block in its distal part or in both bundle-branches.

AV nodal block with blocked impulses and concealed conduction in the His-Purkinje-system (Fig. 12, Table, Case 7)

The tracing was recorded from a woman aged 80 years with syncopal attacks over a period of two years. The electrocardiogram showed varying degrees of AV block and prolongation of the PR interval of conducted sinus beats up to 225 msec. The His bundle electrogram was recorded during phases of varying 2:1 AV block (Fig. 4) and 3:2 AV block. When higher degrees of AV block occurred concealed conduction disturbances were revealed in the His-Purkinje-system, and also in the Purkinje-myocardial fibres (the gate), as well as increased delay in the AV node. The AH interval was prolonged to 180 msec. Conduction in the proximal part of the His-Purkinje-system was normal (HV interval 35 msec). The fourth sinus impulse was conducted through the AV node and its A wave was followed by a BH wave but not by a V wave, i.e. there is concealed block in the His-Purkinje-system. In spite of the long ventricular cycle length (2410 msec) the ventricular complex of the fifth sinus impulse showed conspicuous intraventricular block (QRS 180 msec). The explanation appears to be that the preceding blocked sinus impulse penetrated deeply into the His-Purkinje-system, with phase four depolarization of the Purkinje-myocardial fibres accounting for the intraventricular block. The His bundle electrogram showed therefore AV nodal block and Purkinje-myocardial system block, due to either manifest or concealed conduction of sinus impulses.

Multiple site block with first-degree AV nodal block, complete His-Purkinje-system block, and right bundle-branch block of orthograde impulses, and concealed retrograde conduction of idioventricular beats (Fig. 13, Table, Case 18) The tracing was recorded from a woman aged 67 years with Adams-Stokes attacks over a period of 5 years. The standard electrocardiogram showed complete heart block, atrial rate 100 a minute, ventricular rate 37 a minute. The ventricular complexes measured 120 msec and resembled right bundle-branch block, suggesting that the idioventricular focus was located in the contralateral left ventricle. The His bundle electrogram showed that each sinus P wave was followed by an abnormal BH wave measuring up to 65 msec in duration, and the BH waves were not followed by V waves so that the site of complete block was the His-Purkinje-system. The AH interval was also abnormally prolonged (180 msec). Thus the sinus impulses were delayed in the AV node and probably also in the main bundle of His, but were

completely blocked in the His-Purkinje-system. However, when idioventricular impulses occurred late in atrial diastole they were followed by an abnormal deflection (BH wave) which is distinct from the V wave of the idioventricular beat. The explanation appears to be that the BH wave represents depolarization of the main bundle of His in a retrograde direction by the idioventricular impulse. That this is the likely mechanism is supported by the premature occurrence of the BH wave. Thus, concealed retrograde conduction has been revealed by the His bundle electrogram. This observation has important physiological implications for it supports the concept that facilitation of retrograde conduction in complete orthograde AV block is independent of retrograde atrial activation (Cohn and Fraser, 1914; Scherf and Schott, 1953; Fletcher and Morton, 1968). The explanation may be that the preceding sinus impulse induces electrophysiological changes at the site of complete block in the His-Purkinje-system, which facilitate the passage of the retrograde idioventricular impulse when this occurs at a critical stage in atrial diastole.

Discussion

All the patients in this study had organic heart disease. Necropsy studies of the specialized conduction fibres in patients dying of heart block have confirmed the importance of localized changes in the fibres of the AV node and His-Purkinje-system (Lev, 1961; Zoob and Smith, 1963; Lenegre, 1964; Rosenbaum *et al.*, 1968; Davies and Harris, 1969). The proximal tissues of the conducting system are closely related to the fibrous skeleton of the ventricles and may be involved in its degenerative processes, for example by spread of the calcific process in diseased mitral and aortic valves as in Case 18. The His-Purkinje-system may be involved by ischaemic processes as in Case 22, or the infiltrative processes of a cardiomyopathy as in Case 9. Chronic heart block however may develop without any apparent cause, and the explanation may be in the nature of the highly specialized conduction fibres. Nothing is known about their rate of growth with increasing age, but it seems likely that if cardiac enlargement develops it may outpace the capacity of the Purkinje fibres to adapt to the increased muscle mass leading to stretching rupture and fibrous replacement with resultant impaired conduction. Thus the His bundle electrogram reveals more precisely the quality and quantity of AV block than is possible with conventional surface tracings. Some questions pertaining to its use will now be discussed.

The outstanding complication of heart block is recurrent Adams-Stokes attacks and these are regarded as an indication for artificial pacing either temporary or permanent. However, Adams-Stokes attacks rarely occur in the presence of the examiner and the description of a witness to the attack alone may be available. Moreover, if the period of cardiac arrest is less than 5 seconds, unconsciousness may not supervene and the electrocardiogram may not show blocked beats during several recordings. The His bundle electrogram may show a localized block in the His-Purkinje-system, even if the PR and QRS intervals are normal, as in Case 9, i.e. it may reveal a latent intraventricular block. In addition, Adams-Stokes attacks may occur in patients with atrioventricular nodal block (Group II) as in Cases 2-7, all of whom had had at least one previous Adams-Stokes attack, and in Group III His-Purkinje-system block (Cases 8-17). Nothing is known about any difference in prognosis between these two sites of block so far as sudden death is concerned, but the His bundle electrogram will differentiate them and this may have therapeutic implications. Intra-atrial internodal block and atrioventricular nodal block (Groups I and II) are likely to respond to sympathomimetic drugs, whereas His-Purkinje-system block is unlikely to be affected by such drugs, and immediate pacing may be the treatment of choice. Lastly, it is significant that in Groups III and IV where His-Purkinje-system block was present, standard electrocardiograms showed intraventricular block, suggesting that a disease process affecting a bundle-branch may spread proximally to involve the main bundle or the contralateral bundle resulting in bilateral bundle-branch block. Thus, the occurrence of intraventricular block in a patient with a history of syncope probably indicates extensive involvement of the Purkinje fibres, even if the PR interval is normal. Finally, apart from its potential clinical value in assessment of heart block the His bundle electrogram can reveal electrophysiological changes such as concealed conduction, as in Cases 2, 3, 7, and 18, which add to the basic understanding of impulse conduction and impulse formation in the human heart.

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