Depression of Escape Pacemakers Associated with Rapid Supraventricular Rate in Patients with Atrioventricular Block

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SUMMARY

Intermittent failure of escape pacemakers to maintain an effective rate when the supraventricular rate was rapid was observed in three patients with complete, or high degree atrioventricular block. In two of these patients asystole or slowing of escape pacemaker rate to less than 20 per minute was reproduced by atrial pacing at rates from 125 to 150 per minute. We localized the site of the block and the site of escape pacemaker proximal to the bifurcation of the His bundle in all patients. We assume that a critical increase in atrial rate facilitates penetration of the nonconducted supraventricular impulses into the area of block causing discharge of the escape pacemaker. We suggest that such concealed discharge of escape pacemakers should be considered as a cause of slow and variable rate of escape pacemakers in patients with block proximal to the bifurcation of the His bundle.

Additional Indexing Words:
Concealed conduction
Asystole
Concealed discharge
Syncope
His bundle electrograms
Atrial pacing

THE MAINTENANCE OF CARDIAC FUNCTION in patients with complete or high degree atrioventricular (A-V) block depends on adequate function of the escape pacemakers. The inherent discharge frequency of escape pacemakers in patients with complete A-V block usually ranges between 30–45 beats per minute—a rate sufficient to protect most patients from a critical decrease in cardiac output. Sometimes, however, unexpected slowing or standstill of escape pacemakers occurs spontaneously and leads to syncope or other manifestations of low cardiac output. The causes of such spontaneous failure of the escape mechanism are not known. In this paper, we report three patients with high grade A-V block in whom we attributed critical slowing of the escape pacemakers to the increased supraventricular rate. To explain this association we propose the hypothesis that a supraventricular impulse which fails to propagate through the A-V junction may nevertheless disturb the function of the escape pacemaker located distal to the site of block. Our observations suggest that such events may occur when the site of an escape pacemaker is proximal to the bifurcation of the His bundle.

Case Histories

Case 1

S.H. was a 38-year-old man admitted to the University of Kentucky Medical Center Hospital (UKMCH) on 1/26/71 following two syncopal episodes during the week before the admission. The patient had had a slow heart rate since adolescence. The earliest available electrocardiogram, (ECG) recorded in 1965 (fig. 1), showed right bundle branch block (RBBB) and 2:1 A-V block with a P-R interval of 0.16 sec. In 1969 (fig. 1) the ECG was unchanged except for lengthening of the P-R interval in the conducted beats to 0.24 sec. In 1970 the ECG showed complete A-V block. The RBBB pattern of the escape beats was identical to the pattern in the conducted beats in the previous years (fig. 1). The ventricular rate on admission was 34 beats per minute with regular rhythm. Abnormal physical findings included a blood pressure of 220/90 mm Hg, varying intensity of first heart sound, and a grade 2/6 ejection systolic murmur at the lower left sternal border. The ECG on admission showed complete A-V block with an unchanged QRS pattern in the escape beats. While the patient’s ECG was monitored, the rate of the escape pacemaker varied spontaneously between 13 and 34 beats per minute. Carotid sinus massage decreased the atrial rate and increased the escape rate (fig. 2). This phenomenon could be repeatedly demonstrated. A permanent transvenous demand pacemaker was implanted and the patient was discharged free from symptoms.

Comment

These clinical observations suggested that the rate of the escape pacemaker was influenced by abrupt changes in the rate of supraventricular impulses and prompted the studies performed in two subsequent patients described below.

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Case 2

B.P. was an 81-year-old woman admitted to the UKMCH on 11/22/72 with a history of nausea, vomiting, and two syncopal episodes on the day before the admission. The patient gave no past history of heart disease and was not receiving cardiac drugs. She was disoriented; her heart rate was 40 beats per minute with a regular rhythm; blood pressure was 154/64 mm Hg. Abnormal physical findings consisted of cannon waves in the neck, variable intensity of the first heart sound, and a grade 3/6 ejection systolic murmur heard over the entire precordium and transmitted to the neck. Urine examination revealed proteinuria, pyuria, and bacteriuria. The chest roentgenogram showed a heavily calcified mitral valve ring. This was corroborated by echocardiogram which also suggested fibrosis of the aortic root. The ECG showed sinus rhythm with atrial rate of 120 beats per minute and complete A-V dissociation. The rate of the ventricular escape pacemaker was 40 beats per minute. The duration of the QRS complex was 0.08 sec, and the ECG pattern suggested left ventricular hypertrophy. She was treated for the urinary tract infection and the ECG was monitored. The A-V block disappeared and all sinus beats were conducted with a P-R interval of 0.19 sec (fig. 3, upper two traces). However, on the eleventh day she again developed intermittent complete A-V block with the rate of the escape pacemaker ranging from 12 to 40 beats per minute. The sinus rate was always more rapid when A-V block was present than when A-V conduction was normal (fig. 3, lower two traces). The configuration of the escape complexes was the same as that of the conducted beats. A temporary transvenous pacemaker was inserted into the right ventricle but the response to pacing was not consistent due to unstable electrode position. Therefore, this pacemaker was withdrawn into the right atrium, and a second transvenous pacemaker was inserted into the right ventricle. This provided an opportunity to test the effect of varying atrial rates on the A-V conduction. Prior to atrial pacing, sinus rhythm was present with 2:1 A-V block, and the atrial rate was 105 beats per minute. When the atrial pacing rate ranged from 110 to 120 beats per minute, the 2:1 A-V block persisted, but when the atrial pacing was increased to 125 beats per minute, A-V conduction ceased and no escape pacemaker emerged (fig. 4). This intervention was repeated several times with the same results. The longest period we allowed ourselves to observe ventricular asystole was nine seconds. When the ventricles were paced no retrograde conduction to the atria was recorded.

On the following day we repeated atrial pacing under the protection of a demand ventricular pacemaker and recorded His bundle electrograms with a tripolar electrode catheter introduced via the right femoral vein and positioned across the tricuspid valve. Informed consent was obtained prior to the study. Bipolar pacing electrodes were positioned along the lateral wall of the right atrium for atrial pacing and the right ventricular apex for ventricular pacing. Pacing at various rates with stimulus strengths twice diastolic threshold was accomplished with a battery powered pulse generator. * His bundle electrograms were recorded on a multichannel recorder† with filter frequencies set at 40 and

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*Medtronic 5880.
†Honeywell model 1912 visisorder.

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Figure 1
Electrocardiograms of Case 1 (S.H.).

Figure 2
Strips of electrocardiogram (lead II) of Case 1 (S.H.). In the upper strip complete A-V block with A-V junctional escape pacemaker. In the middle strip carotid sinus pressure applied during the period under the solid black line is associated with lengthening of P-P and shortening of R-R. The lowest strip recorded after cessation of carotid sinus massage shows shorter P-P and longer R-R intervals. The second complex in this strip is probably a ventricular extrasystole.

Figure 3
Electrocardiogram of Case 2 (B.P.) is suggestive of left ventricular hypertrophy. Strips of lead II, recorded at 5 min intervals show 1:1 A-V conduction (upper) and apparent complete A-V block with A-V junctional escape rhythm (lower). The P-R interval is shorter (0.52 sec) during complete A-V block than during 1:1 A-V conduction (0.64 sec).
500 Hz on photographic paper moving at a speed of 100 mm per second. His bundle electrograms were recorded simultaneously with two standard ECG leads and validated by an increase in A-H interval during rapid atrial pacing.

During sinus rhythm with 1:1 A-V conduction (fig. 5, upper panel) the A-H interval (100 msec) was normal and the H-V interval (65 msec) was prolonged. During atrial pacing at 150 beats per minute, all atrial impulses were blocked below the site of recording of the His bundle spike (fig. 5, lower panel). Ventricular pacing at a rate 40 beats per minute was used to support circulation when it was noted that no escape pacemaker emerged after 6 sec of asystole.

Comment

In this patient increase in atrial rate was associated with an increased A-V block, apparently due to concealed conduction of the supraventricular impulses into the A-V junction. The consistent slowing of ventricular rate after each increase in atrial rate suggested that the patient's syncope could have been precipitated by a critical increase in supraventricular rate. Atrial pacing at a rapid rate completely suppressed A-V conduction and appeared to interfere with the impulse formation or conduction of the escape pacemakers.

Case 3

C.C. was an 84-year-old man admitted to UKMCH with a history of angina pectoris, congestive heart failure, slow heart rate, and frequent ventricular extrasystoles. During the previous two years he had been treated with diuretics and potassium salts. Physical examination revealed an alert man in no acute distress. His heart rate was 45 beats per minute and the rhythm was irregular; blood pressure was 170/70 mm Hg. Auscultation revealed a few rales at the bases of both lungs and a grade 2/6 ejection systolic murmur at the left lower sternal border. The chest roentgenogram showed moderate cardiomegaly. The ECG showed an ectopic atrial rhythm with a rate of 125 beats per minute, complete A-V block, and an escape rhythm with a rate of 35 beats per minute. The escape beats showed a pattern of left bundle branch block (LBBB). While the ECG was monitored in the cardiac care unit, the ventricular rate decreased to 15 beats per minute. A temporary transvenous ventricular pacemaker was inserted and the effect of atrial rate on the function of the escape pacemaker was tested in the same manner as in patient 2. Prior to atrial pacing (fig. 6, top tracing), atrial rate was 125 beats per minute and ventricular rate was 33 beats per minute. The escape rhythm was slightly irregular but the R-R intervals did not vary by more than 100 msec. Each escape QRS complex was preceded by a His bundle spike. The H-V interval was 85 msec. Pacing the atria at 125 beats per minute did not alter the rate of the escape pacemaker (fig. 6, middle tracing), but when the atrial pacing rate was increased (fig. 6, bottom tracing), the escape complex failed to appear at the expected time. However 900 msec later another escape complex arose from a more distal site. This complex had different QRS configuration and was not preceded by a His bundle spike. Such abrupt transition from a more rapid escape pacemaker with a QRS complex preceded by H spike to a slower escape pacemaker with a QRS complex not preceded by H spike occurred on five consecutive occasions when the atrial pacing rate reached a critical level which ranged from 130 to 150 beats per minute.

Comment

In this patient with apparent complete A-V block, increase in atrial rate caused slowing of the ventricular rate and shift of the escape pacemaker to a more distal site.

Discussion

Two of our patients (patients 1 and 3) had an apparent permanent complete A-V block while in the third (patient 2) the complete A-V block was induced by critical increase in atrial rate. In all three patients, the spontaneous rate of the escape pacemaker intermittently fell to less than 20 beats per minute during

![Figure 4](https://circ.ahajournals.org/doi/10.1161/01.CIR.50.4.257)

Electrocardiogram of Case 2 (B.P.), simultaneously recorded leads I, II, and III. Prior to initiation of atrial pacing sinus rhythm with 2:1 A-V conduction was present. The atrial pacing stimulus interval (X-X) (0.48 sec) is shorter than the interval between spontaneous atrial beats (0.50). The first atrial capture is followed by ventricular asystole resulting from complete A-V block and failure of escape pacemakers. Ventricular activity is re-established with ventricular pacing (Y).

![Figure 5](https://circ.ahajournals.org/doi/10.1161/01.CIR.50.4.257)

His bundle electrogram (HBE) of Case 2 (B.P.). Upper panel shows normal A-H (100 msec) and prolonged H-V (65 msec) intervals during 1:1 A-V conduction. Lower panel shows effect of rapid atrial pacing at a rate of 150 beats per minute. Atrial depolarizations (A) follow atrial pacing stimulus artifacts (S). His bundle spikes (h) are present after each atrial depolarization. (A-H interval = 125 msec); absence of ventricular complex V after h indicates block below the level of recording of His bundle spike. Ventricular complexes V follow ventricular pacing stimulus S₂.
monitoring. This event was always associated with increase in supraventricular rate. In the two patients studied by means of atrial pacing, the critical atrial rates which precipitated the apparent malfunction of escape pacemakers were 125 beats per minute in one and 130–150 beats per minute in the other.

Several studies have shown that increase in atrial rate may decrease the ventricular rate by decreasing the number of conducted beats. This phenomenon has been described in patients with type I A-V block, type II A-V block, and even in persons without A-V block. McHenry and Knoebel reported a patient in whom a critical spontaneous increase in atrial rate caused a sudden change from 1:1 A-V conduction to complete A-V block. Increased severity of A-V block associated with sinus tachycardia induced by isoproterenol has also been reported. Langendorf and Pick have postulated that the concealed conduction of sinus impulses within the A-V junction may cause not only repetitive blockade of supraventricular impulses but also discharge of the subsidiary pacemakers. However, we have not encountered reports of patients in whom an increase in atrial rate caused a long asystole without emergence of an escape pacemaker.

Postulated Mechanisms

The exact mechanism of suppression of escape pacemakers in these patients is not understood. We have no evidence to assume sudden changes in vagal tone, or extracellular potassium concentration. We considered the possibility that the ventricular slowing caused by increase in atrial rate was related to a change in cardiac output and in blood pressure leading to decrease in the coronary perfusion and ischemia of the A-V conducting tissue. Such mechanism proposed by Massumi et al. to explain paroxysmal A-V block cannot be excluded but we do not consider it likely because the apparent malfunction of the escape pacemakers persisted when the circulation was supported by ventricular pacing (fig 5).

We favor the hypothesis that rapid atrial pacing contributes in some manner to the discharge of the escape pacemaker. This could occur if the supraventricular impulses which failed to traverse the entire A-V junction suppressed the impulse formation, or condition of the A-V junctional escape pacemakers. Such concealed discharge of the escape pacemakers can be best explained by repetitive stimulation during the relative refractory period with a resulting slow decremental conduction and functional block. Moore illustrates such a phenomenon in figure 3 of reference 9. In this figure rapid stimulation of the right bundle branch causes a slow and apparently decremental conduction within the right Purkinje papillary muscle junction.

Concealed conduction and concealed discharge could explain our observations in the patient without permanent A-V block. However, in two patients with the apparent permanent complete A-V block, an additional phenomenon must be postulated to explain how the critical increase in rate enables the impulse to traverse the A-V junction in order to reach the site of the escape pacemakers. Such facilitation of A-V conduction could be attributed to the phenomenon of "summation." Cranefield et al. demonstrated that excitation threshold may be reached by summation of two weak subthreshold impulses elicited at opposite ends of a Purkinje fiber. A similar increase in stimulus strength could perhaps be caused by summation of two successive weak stimuli traveling in the same direction. This would occur if the second stimulus reached the site of block before the depolarizing effect of the previous subthreshold stimulus had completely subsided. The method of propagation in such situations could be electrotonic spread, decremental conduction in a tissue with incompletely reactivated rapid sodium conductance, or even a slow channel-dependent conduction in depolarized myocardium. In either case facilitation of conduction by rapid stimulation would be explained by less time available for complete repolarization after the transient subthreshold depolarization.

Site of Block and Site of Escape Pacemakers

Analysis of the ECG and His bundle recordings
DEPRESSION OF ESCAPE PACEMAKERS

shows that in two patients both the site of block and site of escape pacemaker were proximal to the bifurcation of the His bundle, and in one patient proximal to the bifurcation of the main LBB stem. In the first two patients, the QRS configuration of the escape pacemaker was the same as in the conducted beats prior to the development of complete A-V block. This finding indicates that in the patient with the narrow QRS complex the block was proximal to the bifurcation of the His bundle, and in the patient with the RBBB pattern proximal to the division of the LBB stem. The type II pattern suggests that the site of block was in the His bundle rather than in the A-V node. Heavy calcification of the mitral ring in the second patient also favors location of a lesion below the A-V node. In this patient, the normal A-H interval, the prolonged H-V interval in the conducted beats, and the type II block distal to the site of the Hs bundle spike suggest that the block was in the His bundle. In the third patient (C.C.), no ECGs were recorded before the appearance of A-V block and no conducted beats were present. However, the His bundle electrograms showed that the block was proximal to the site of the His bundle spike recording.

We do not know whether the similar anatomic site of block in all three patients indicates that the described phenomenon occurs only in patients with lesions in the main stem of the His bundle. However, we have not observed any changes in the rate of escape pacemakers following an increase in atrial pacing rate in patients with escape pacemakers distal to the bifurcation of the His bundle.

In addition to two patients described in this report, we tested the response of escape pacemakers to atrial pacing in three other patients with A-V block within the His bundle, and found that their escape pacemaker rate was not affected by rapid atrial stimulation. Neither was the rate of escape pacemaker affected by atrial pacing in another study of two patients with complete A-V block within the His bundle. The incidence of escape pacemaker depression induced by rapid atrial rate is not known. Further studies are needed to understand the precise mechanism of this phenomenon and to elucidate other possible factors which may affect the function of escape pacemakers in patients with A-V block.

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