Re-entry due to Manifest and Concealed His Bundle Ectopic Systoles

Report of a Case

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SUMMARY Concealed (C) His bundle ectopic systoles (H') have been shown in man to give rise to first and second degree atrioventricular (A-V) block and to simulate nonconducted atrial premature beats (P'). This report outlines a hitherto undescribed electrophysiologic consequence of H' in a 69-year-old man with arteriosclerotic heart disease and a Wenckebach type second degree A-V block in the His-Purkinje system. During a His bundle study, H' were shown to conduct either to the atria and ventricles with varying relationships to P' and QRS, or to conduct only to the atria, simulating nonconducted P' or atrial fusion beats. Both types of H' could initiate a re-entrant arrhythmia during retrograde conduction. Of particular interest are late coupled H' that failed to conduct to the ventricles and also failed to activate the atria because of prior capture by the sinus impulse (CH'). These CH' could also initiate re-entry by conducting retrogradely to engage the subatrial re-entry circuit. Evidence is presented to suggest re-entry occurs by way of a retrograde concealed accessory pathway and antegrade conduction in the atrioventricular node.

IN 1947, LANGENDORF AND MEHLMAN\textsuperscript{1} utilizing deductive analysis of the electrocardiogram (ECG) suggested that concealed junctional premature systoles could simulate first and second degree heart block and non-conducted atrial premature beats. Langendorf\textsuperscript{2} and Langendorf and Pick\textsuperscript{2} later proposed that concealed junctional premature systoles could also result in alternation of the P-R interval and pseudo-Wenckebach periods. However, it was not until 1970, when Rosen and associates,\textsuperscript{4} through recording of the His bundle electrogram, provided the first documented example of concealed His bundle ectopic systoles producing intermittent first and second degree atrioventricular (A-V) block. Damato and colleagues\textsuperscript{5} showed, in an experimental model, that induced His bundle extrasystoles could simulate first and second degree A-V block, high degree A-V block, nonconducted atrial bigeminal rhythm, and alternation of the P-R interval. Few clinical examples\textsuperscript{6-11} proved the probability of some of these experimental observations. The present report extends further the observations on electrophysiologic manifestations of His bundle ectopic systoles by describing a case in which both manifest and concealed His bundle ectopic systoles could initiate a re-entrant arrhythmia. A critical analysis of the conduction characteristics of the re-entry circuit strongly suggests retrograde conduction through an accessory pathway and antegrade conduction through the A-V node.

Case Report

J.B., a 69-year-old man, was admitted to the Veterans Administration Hospital for evaluation and treatment of frequent dizzy spells. The patient had a diagnosis of arteriosclerotic heart disease for the last ten years but there was no history of a documented myocardial infarction. For the last three years, he had received 0.25 mg of digoxin daily for possible congestive heart failure. Four months prior to admission, the patient noticed frequent episodes of dizzy spells. The ECG showed right bundle branch block, left axis deviation, and inversion of T waves in precordial leads. The rhythm was sinus with A-V conduction varying from first degree block to Wenckebach type second degree block and 2:1 A-V conduction (fig. 1). The patient records also revealed frequent junctional ectopic systoles with a QRS configuration similar to conducted sinus beats and retrograde P wave either preceding, superimposed, or immediately following the QRS. In addition, blocked premature junctional P waves (P') were also observed (figs. 1 and 2). Figure 2 will be explained in detail later.

Because of the Wenckebach type A-V conduction, the patient was originally suspected to have digitoxicity. However, the serum digoxin level obtained on admission was 1.2 ng/ml and serum electrolytes, blood area nitrogen, and creatinine were normal. The possibility of a silent myocardial infarction was ruled out by normal levels of serum enzymes and the absence of serial changes in the QRS and ST-T complexes. With the patient's recent symptoms in mind, an organic A-V conduction disorder, possibly at the His-Purkinje level, was suspected and an electrophysiologic study was performed after obtaining the patient's informed consent.

Electrophysiologic Studies

His bundle electrograms (HB eg) were obtained using a tripolar electrode catheter inserted percutaneously into the right femoral vein.\textsuperscript{12} A quadripolar electrode catheter was inserted into an arm vein and positioned against the lateral wall of the right atrium. The proximal two electrodes were
used to record a high right atrial electrogram (HRA eg) while the distal pair was used to pace the atrium. A bipolar electrode catheter was introduced through another arm vein and positioned in the coronary sinus to record the coronary sinus electrogram (CS eg) which would reflect left atrial activation. Atrial pacing was performed using a programmed digital stimulator that delivered rectangular impulses of 1.5 msec duration at approximately twice diastolic threshold. Ventricular pacing was accomplished in a similar way using the lowest milliamperage that permitted reliable capture. Refractory period studies were performed with the atrial extrastimulus method.19

Figure 3 shows that A-V nodal conduction time was at the upper limit of normal (normal range of A-H interval is 50-120 msec), while His-Purkinje conduction time was prolonged (normal range of H-V interval is 35-55 msec). Both 3:2 Wenckebach and 2:1 A-V block were localized distal to the recording site of the Hb eg. The record shows an increment in H-V interval of 100 msec during a 3:2 Wenckebach period.

Figure 4 shows a premature His bundle ectopic impulse (H') that conducts retrogradely to the atria but fails to conduct in antegrade direction. This beat is followed by a second His bundle deflection (H) that conducts in both antegrade and retrograde directions. The next H' is blocked in both directions and permits the reset sinus impulse to escape. Figure 5, records A and B, show a similar pattern but with a relatively late coupled H'. In record A, H' resulted in partial retrograde activation of the atria giving rise to an atrial fusion beat (FP). In record B, the sinus impulse anticipated the retrograde conduction of H' and completely captured the atria. The sequence of atrial activation during complete retrograde atrial activation (fig. 4), complete antegrade atrial activation (fig. 5, record B), and atrial fusion (fig. 5, record A) is clearly shown in HRA eg and the atrial electrogram in the Hb recording. A diagrammatic analysis of the rhythm disorder in figures 4 and 5 suggests that H' during its retrograde conduction to the atria entered a re-entrant pathway and conducted antegrade to the ventricles. A second re-entrant cycle conducted retrogradely to the atria before termination of the re-entrant activity. The diagram in figure 5 shows that the atrium is not a necessary link in the re-entry circuit.

Two findings in the electrophysiologic study favor re-entry initiated by H' rather than the alternative possibility of two consecutive H'. Although the polarity of H' was frequently similar to the antegrade H during the study, minimal changes in catheter position could completely reverse the polarity of H' while not affecting the polarity of H. This is illustrated in all three records in figures 4 and 5 and strongly suggests that the second H that follows H' represents antegrade His bundle activation.

The most convincing evidence of re-entry, however, was obtained during ventricular stimulation. This resulted in retrograde atrial activation followed by a re-entrant antegrade H and a second re-entrant P wave. This sequence gave rise to an interesting arrangement during continuous ventricular pacing (fig. 6). Record A shows that, at a pacing cycle of 650 msec, a retrograde P wave follows each paced impulse with alternating long and short QRS-P' intervals. The long QRS-P' is always followed by H. Shortening of the pacing cycle was associated with no change in the long QRS-P' interval, very slight increase of the A'-H interval, and no change in the interval from H to next A' which measured 150 msec in the Hb eg. These changes resulted in gradual lengthening of the short QRS-P' interval as the pacing cycle length shortened while the long QRS-P' interval remained constant. Sudden termination of ventricular pacing after a paced impulse with a long QRS-P' interval was always
followed by H and a second P' (second half of record B in figure 6). The diagram in figure 6 suggests that during ventricular pacing alternate paced impulses conducted retrogradely to the atria. During its retrograde A-V conduction, the impulse entered a re-entrant pathway and conducted antegrade to H, then retrogradely to the atria giving rise to a re-entrant P'.

Study of effective refractory periods of retrograde and antegrade pathways of the re-entry circuit by the ventricular extrastimulus method was not possible because of impaired ventriculo-atrial conduction. Figure 7 shows ECG tracings that correspond to the records in figure 6. It is clear that what looks like 1:1 ventriculo-atrial conduction with alternate conduction times in figure 7, records A and B, is a fortuitous arrangement due to 2:1 ventriculo-atrial conduction and re-entrant atrial activation. The mechanism of conduction during ventricular stimulation showed an obvious similarity to conduction of H' and lends direct support to the hypothesis of re-entrant activity in figures 4 and 5.

Characteristic changes in conduction times in both retrograde and antegrade pathways during H'-induced re-entry were observed in relation to cycle length changes. This is shown in figures 4 and 5 and is graphically illustrated in figure 8. The interval between the re-entrant antegrade H and re-entrant A' (H-A' interval) was taken to represent the retrograde pathway conduction time. Since A' was usually superimposed on V in the Hb eg, the H-A' interval was calculated as the interval between H and HRA eg minus the interval between A' on the Hb eg and HRA eg obtained during isolated retrograde atrial activation. The low-to-high right atrial conduction time consistently measured 50 msec. The H-A' interval was considered more representative of the retrograde pathway conduction time compared to the H'-A' interval, which could have included a variable conduction delay from the site of origin of H' to the takeoff of the retrograde pathway. Figure 8, diagram A, shows the relation of H-A' interval to preceding H'-H interval. The H-A' interval which measured 135–150 msec showed no significant change on shortening of the H'-H interval from 650 to 460 msec. Failure of re-entrant A' was not observed at cycle lengths seen in this study so that the effective refractory period (ERP) of the retrograde pathway could not be measured. The H-A' interval during spontaneous H'-induced re-entry was comparable to the H-A' interval during ventricular pacing induced re-entry (150 msec). Also, the failure of the retrograde pathway conduction time to increase on shortening of preceding cycles was similar during both spontaneous H' and ventricular pacing induced re-entry. On the other hand, the A'-H interval was taken to represent the antegrade pathway conduction time. Figure 8, diagram B, shows the relation of A'-H interval to preceding A-A' interval. The A'-H interval showed gradual lengthening from 320 to 500 msec with shortening of the preceding A-A' interval from 860–610 msec. At an A-A' interval of 590–610 msec, antegrade conduction failed. This value would represent the ERP of the antegrade pathway. When antegrade conduction failed, the surface ECG showed a non-conducted premature P' (see figure 2, records D to F).

Failure to sustain re-entry was always related to block in the antegrade pathway at A'-A' cycles shorter than 590–610 msec. The ERP of the A-V node measured by the atrial extrastimulus method was 580 msec, a value similar to the ERP of the antegrade pathway. The response of the antegrade pathway to preceding cycle length was only partially comparable to that of the A-V node during antegrade conduction of atrial premature stimuli (see figure 8, diagrams B and C). The major discrepancy was the relatively longer antegrade conduction time (A'-H interval) at long preceding A-A' cycles. Thus, the A'-H interval measured 350 msec following an A-A' cycle of 800 msec.

**Figure 3.** His bundle electrograms (Hb,eg and Hb,eg) showing a 3:2 Wenckebach A-V conduction in the His-Purkinje system. HRA = high right atrial electrogram.

**Figure 4.** Re-entry secondary to an ectopic His bundle systole (H'). A diagrammatic analysis of the arrhythmia is shown below. In this and subsequent figures, Hb,eg and Hb,eg = His bundle electrograms; HRA eg = high right atrial electrogram; SAN = sino-atrial node; A = atria; AVN = atrioventricular node; HPS = His-Purkinje system; V = ventricles.
while the A-H interval at a similar A-A cycle measured 115–120 msec. This discrepancy could possibly be ascribed to partial retrograde conduction to the A-V node at relatively long H-H' coupling intervals. On the other hand, the response of both A'-H and A-H intervals to short preceding cycles was more comparable, which would suggest retrograde A-V nodal block of H' at the shorter cycles. However, precise delineation of the role of retrograde concealed conduction of H' in lengthening the antegrade conduction time of re-entrant A' was difficult.

In view of the preceding interpretation, the second H' shown in figure 4 was considered as an ectopic H' rather than a re-entrant His bundle activation. In addition to showing a configuration basically similar to the first H', it is too immediately coupled to represent part of a continuous re-entrant activity. However, a continuous re-entrant activity was occasionally seen during the electrophysiologic study. Figure 9 shows a short run of re-entrant rhythm initiated by H' that follows a blocked atrial premature beat (X). The surface ECG simulates an accelerated junctional rhythm (atrial rate of 96/min) with 2:1 A-V block. It is interesting to note that the first His bundle ectopic beat showed a P'-QRS pattern simulating the so-called upper nodal rhythm. This is explained by a rather long H'-V interval of 180 msec due to a short H-H' coupling interval and concealed conduction of the premature atrial beat into the H'-V pathway. On the
other hand, the H'-A' interval is relatively short (140 msec).
In contrast, the fourth beat in figure 9 has a P/QRS pattern simulating middle nodal rhythm. This is explained by a slightly longer H-V interval.

Some, but not all, of the ECG rhythm strips shown in figure 2 correspond to records obtained during the electrophysiologic study. Thus, records A, B, and C in figure 2 correspond to figure 4; figure 5, record A; and figure 5, record B, respectively. Records D, E, and F, in figure 2 showed blocked premature P' secondary to H'. The arrhythmia shown in record I was not encountered during the electrophysiologic study. It represented a consecutive series of the rhythm disorder shown in records G and H. The arrhythmia is initiated by P', most probably secondary to H', and consists of a slow regular ventricular rhythm of 38 beats/min with a QRS configuration similar to conducted beats. Each QRS is followed by two retrograde P waves, a pattern similar to the one that followed paced ventricular impulses (see figure 7, record C). The arrhythmia most probably represents a His bundle escape rhythm showing retrograde atrial activation followed by a re-entrant P'. It is to be noted that this arrhythmia does not represent the ECG pattern of continuous re-entry shown in figure 9. A representative ECG that corresponds to this pattern could not be obtained during the patient's stay in hospital.

Clinical Course

A permanent transvenous right ventricular pacemaker was inserted in the patient's heart. He was advised to continue on the antifailure regimen. No treatment was given for the ectopic His bundle systoles. On follow-up visits, the patient was asymptomatic and reported complete disappearance of dizzy spells.

Discussion

The site of A-V block in this case could be either in the distal His bundle or somewhere along the posterior division of the left bundle, assuming the presence of constant block in the right bundle and anterior division of the left bundle. His bundle pacing which could have helped to solve this question was not done in this case. The H' could have originated at or anywhere above the site of A-V block. The change of polarity of H' without concomitant change in polarity of H by slight maneuvering of catheter position may suggest a site of origin of H' in the His bundle. Although there was marked variation of coupling of H', a parasystolic pattern could not be worked out.

The electrophysiological study illustrated that, depending on relative conduction delay in the antegrade and retrograde pathways, H' could show varying temporal relationship of P' and QRS, simulating the so-called upper, middle, and lower nodal rhythms. H' could also conduct retrogradely with failure of antegrade conduction simulating nonconducted atrial premature beats. With relatively short coupling, H' can block in both antegrade and retrograde directions and its occurrence can be completely concealed in the surface ECG (see fig. 4). The term concealed H' was originally suggested for those H' that fail to conduct to the ventricle and only partially conduct in the A-V node so that they can only be suspected from their effect on A-V conduction of subsequent atrial beats. Thus, in the strict sense, H'

FIGURE 7. Ventricular pacing simulating 1:1 ventriculo-atrial conduction with alternate conduction times. The arrhythmia is actually due to 2:1 ventriculo-atrial conduction and re-entrant atrial activation.

FIGURE 8. Graphic analysis of the effect of changes in preceding cycle length on retrograde pathway conduction time (H-A' interval) in A, antegrade pathway conduction time (A'-H interval) in B, and A-V nodal conduction time (A-H interval) in C.
that conduct retrogradely to the atria but fail to conduct antegraderly to the ventricles are not concealed, although they may be sometimes difficult to differentiate from blocked atrial premature beats. In the present study, both H' that are manifested by P' and QRS and those that only showed P' could initiate re-entry. Of particular interest is the observation that late-coupled H' that failed to conduct to the ventricles and failed to activate the atria because of prior capture by the sinus impulse could still initiate re-entry. In these instances, re-entry can be appropriately ascribed to concealed H'. The demonstration of re-entry without retrograde atrial activation is another piece of evidence that supports the hypothesis that the atria are not a necessary link in re-entrant rhythms.

A similarity can be drawn between the mechanism of ventriculo-atrial conduction during ventricular pacing as seen in figures 6 and 7 and the mechanism of retrograde A-V nodal Wenckebach during ventricular pacing described by Damato et al. Thus, it is possible in our case that ventriculo-atrial block of alternate paced ventricular beats was due to collision in the His-Purkinje system of the retrograde impulse and the re-entrant impulse. On the other hand, figures 6 and 7 illustrate another example of the occasional limitations of surface ECG in diagnosis of complex arrhythmias. In a recent report, several ECG rhythm strips were described as examples of dual pathways of retrograde impulse conduction in the human heart. Our case illustrates the potential pitfalls of this kind of deductive analysis.

A critical analysis of conduction characteristics of retrograde and antegrade pathways of the re-entry circuit strongly suggests that retrograde conduction took place via an accessory pathway, while antegrade conduction followed the normal A-V nodal pathway. During both paced ventricular beats and spontaneous H', retrograde conduction time showed no increment with shortening of the cardiac cycle. This behavior is distinctly different from the response of the A-V node and closely resembles that of bypass fibers of the A-V node during antegrade17 or retrograde conduction.18 The response of antegrade pathway to cycle length changes and its effective refractory period was comparable to that of the A-V node during premature atrial stimulation. This would suggest that antegrade conduction traversed the A-V node. An accessory pathway with this response pattern would probably stretch from the proximal part of the A-V node close to its atrial connections to somewhere in the His bundle or the nodal-His bundle (NH) region. This pathway would correspond to some of James's tracts.19 The demonstration that re-entry could take place without retrograde atrial activation also suggests that the pathway connected to the atrio-nodal (AN) region of the A-V node or to the atrium very close to the A-V nodal connections. The accessory pathway in the present case showed unidirectional conduction with consistent antegrade block which explains the absence of pre-excitation during sinus rhythm. The presence of bypass pathways of the A-V node that function only in retrograde direction has been recently reported. In one study, the presence of these bypass fibers was suggested in 15% of patients who showed bi-directional conduction.23

In the absence of extensive antegrade and retrograde extrastimulus studies, the alternative possibility of a functional dual A-V nodal pathway should be considered. The evidence in favor of this explanation would be the relatively long H'-A interval which was longer than antegrade A-H interval during sinus rhythm. Curves B and C in figure 8 could represent conduction characteristics of A-V nodal slow and fast pathways, respectively. The fast pathway could be used for retrograde conduction. However, the argument against this possibility was the demonstration of failure of H'-A1 to increase with decrease in H'-H' interval. This differs markedly from the response of a functional dual A-V nodal pathway. In the latter case, lengthening of retrograde conduction time is usually demonstrated on shortening of cycle lengths.24

A re-entrant arrhythmia initiated by H' that only conducts retrogradely to the atria will be difficult to differentiate from a supraventricular ectopic discharge. On the other hand, an accessory pathway capable of only ventriculo-atrial transmission can initiate and sustain re-entrant arrhythmias but remain clinically unrecognized.24 The unusual combination of a re-entrant arrhythmia that utilizes such a bypass and is triggered by concealed H' is highly unlikely to be recognized from the surface ECG.

In summary, our study adds a new dimension to the protean electrocardiographic manifestations of concealed His bundle ectopic systoles. Furthermore, it stresses once more the occasional limitations of surface ECG and the potential value of His bundle electrocardiography not only in unraveling complex disorders of the cardiac rhythm but also in documenting new electrophysiologic observations.

References
Parachute Accessory Anterior Mitral Valve Leaflet Causing Left Ventricular Outflow Tract Obstruction

Report of a Case with Emphasis on the Echocardiographic Findings

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SUMMARY

A case of left ventricular outflow tract obstruction caused by a parachute accessory anterior mitral valve leaflet is presented. The abnormality was detected in an asymptomatic patient by systolic murmur and thrill and was demonstrated by echocardiography, as well as by cardiac catheterization and surgery. These findings are presented. Several previously reported cases of a similar nature are reviewed. An identical case was not found in the English literature.

SUBAORTIC STENOSIS is usually associated with asymmetric hypertrophy of the ventricular septum (also called idiopathic hypertrophic subaortic stenosis, IHSS) or a subaortic diaphragm (fixed or discrete subaortic stenosis). We have documented a case in which the obstruction was caused by an accessory mitral valve leaflet which parachuted into the left ventricular outflow tract in systole.

Case Report

The patient was an asymptomatic nine-year-old boy who was seen by his physician with a complaint of enuresis. On physical examination, he was a well developed young male with no abnormalities except those related to the cardiovascular system. Peripheral pulses were of normal rate and quality. The blood pressure in the right arm supine was 86/60. There was a systolic thrill over the neck, suprasternal area, right upper sternal border and base. The second heart sound was finely split; both components were unremarkable, and no click was present. There was a grade IV/VI harsh systolic murmur at the upper right sternal border radiating well to the base of the heart and less well to the neck and back. There was a grade III/VVI systolic murmur at the lower left sternal border radiating to the apex but not axilla and having a high pitched squeak. It did not vary with position. No diastolic murmurs were present.

The chest X-ray showed slight prominence of the left ventricle, borderline heart size and normal pulmonary vasculature. The ECG and vectorcardiogram showed left anterior...
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Circulation. 1976;53:902-908
doi: 10.1161/01.CIR.53.5.902
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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