Ventricular Tachycardia with Narrow QRS Complexes (Left Posterior Fascicular Tachycardia)

By Howard C. Cohen, M.D., Edilberto Gozo, Jr., M.D., and Alfred Pick, M.D.

SUMMARY

Ectopic atrial, A-V junctional, and ventricular tachycardias in man have been associated with digitalis medication. Recently it has become possible to distinguish various locations of pacemakers within the specialized conduction system of the ventricles on the basis of the form of the QRS complexes in the standard electrocardiogram. Tachycardias originating in the left bundle branch and documented by right, left, and His bundle recordings have been produced in animals given excessive digitalis. We have noted a similar tachycardia in a patient with ischemic heart disease receiving digitalis during hypokalemia. The QRS complexes were 0.10 sec in duration and by contour suggested an ectopic focus located in the posterior fascicle of the left bundle; His bundle recordings were consistent with this diagnosis. As the ectopic rhythm became synchronized with a slightly irregular sinus rhythm, bidirectional depolarization of the His bundle with fusion His potentials could be demonstrated.

Additional Indexing Words:
A-V dissociation  His bundle potentials (retrograde)  Digitalis toxicity
His bundle potentials (fusion)  Fascicular block  Bundle-branch block

ROSENBAUM\(^1\) has located the origins of "narrow ventricular ectopic beats" by their contour in the standard electrocardiogram (ECG). This method of localization was used to explain the aberrant QRS complexes falling within a portion of the cardiac cycle in which ventricular refractoriness can no longer account for the aberration of a supraventricular impulse. His bundle electrograms may confirm the origin of such beats. His bundle potentials falling near the beginning of an aberrant QRS complex, too close for normal Hisian conduction and too early for retrograde conduction from the distal portions of the ventricles, indicate that the pacemaker lies high in the specialized conduction system of the ventricles.\(^2,3\) The QRS duration of such beats may, in the standard ECG, appear to be within, or slightly beyond, the normal range\(^1,2\) (see fig. 2 below). Indeed, left bundle-branch rhythms with His bundle potentials near the beginning of a slightly prolonged QRS complex have been produced experimentally by the administration of digitalis to dogs.\(^4\)

The purpose of this report is to present a clinical observation of A-V dissociation caused by emergence of such an ectopic ventricular rhythm, with minimal QRS prolongation in the standard ECG, that has been documented by His bundle recordings. The patient had ischemic heart disease and was receiving digoxin. Detailed analysis of temporal relations of atrial, His, and ventricular complexes permitted placement of the ectopic pacemaker...
in one of the fascicles of the left bundle. In addition, fusion of sinus and ectopic impulses within the ventricles or within the His bundle could be demonstrated.

His bundle electrograms were recorded through a tripolar electrode catheter with electrodes 2 mm wide and 1 cm apart. The catheter was placed transcutaneously into the femoral vein and positioned across the tricuspid valve.\(^5\) These filtered electrograms (40–500 Hz) were recorded simultaneously with standard ECG lead II on a photographic recorder at a paper speed of 100 mm/sec (Electronics for Medicine, Model DR-8).

**Case Report**

N. M., a 40-year-old black woman, was admitted to Michael Reese Hospital and Medical Center for chest pain. She had a 7-year history of intermittent angina pectoris, mild essential hypertension, and diabetes mellitus controlled with diet. Medication included hydrochlorothiazide, 50 mg/day.

During this hospitalization, laboratory tests were normal except for a fasting blood sugar of 164 mg% and serum potassium levels ranging from 3.3 to 4.0 mEq/liter. Digoxin medication was begun for treatment of congestive heart failure. After 4 days, at a time when serum potassium level was 3.6 mEq/liter, the heart rate became faster and at times irregular. Electrocardiograms including a His bundle recording were obtained. Oral potassium chloride solution, 60 mEq daily, was begun, and digoxin was discontinued. The arrhythmias disappeared within 48 hours, and the ECG stabilized.

**Description and Interpretation of ECG’s and His Bundle Recordings**

An ECG (fig. 1) 1 day before the increase in heart rate shows sinus rhythm at a rate of

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**Figure 1**

ECG during sinus rhythm showing the normal contour of ventricular complexes.

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71/min with a P-R interval of 0.16 sec. The ventricular complex with a QRS axis of $0^\circ$ shows primarily left ventricular hypertrophy. However, ST-T segments appear to be modified by ischemic alterations of the anterior and posterior walls and possibly by hypokalemia, since the T waves merge with enlarged U waves. Figures 2 and 3 were recorded after acceleration of the heart rate. In figure 2, P waves are discernible only in $V_6$ at a shortened P-R interval, suggesting that atria and ventricles are dissociated, P waves coinciding with QRS complexes in the other leads. Moreover, a fundamental change in the shape of the ventricular complexes has occurred. The QRS is slightly widened to 0.10 sec and its frontal mean axis has shifted to about $-80^\circ$, manifested by development of prominent S waves in leads I, II, aV$_L$, and aV$_F$ and a "late" R wave in aV$_R$. In the chest leads the most pronounced changes consist of the appearance of a late R wave in $V_1$ and S waves over the left precordium.

Concomitant with these QRS alterations, the ST-T segments no longer show features of either left ventricular hypertrophy or of ischemia. However, the hypokalemic alterations noted in figure 1 can still be recognized. It would therefore appear that ventricular action has become independent as a result of acceleration of a subsidiary ectopic pacemaker. This ectopic focus may be located either in the A-V junction with conduction delay in both the right bundle branch and the anterior fascicle of the left bundle branch, or may be located in the posterior fascicle of the left
bundle branch. The distinction between these alternatives could be made by analysis of long leads, pertinent portions of which are shown in figure 3.

In figure 3, three types of ventricular complexes are seen in varying relation to P waves. One type with a P-R interval of 0.16 sec resembles the sinus beats in figure 1; a second type with a very short P-R distance, or without discernible P waves, resembles the ectopic beats of figure 2; and a third type of more or less intermediate shape, with P-R intervals of 0.14 or 0.16 sec, represents ventricular fusion beats (F). Their occurrence militates against a junctional origin of the ectopic impulse and favors a pacemaker located in the posterior fascicle of the left bundle branch.

The occurrence of these three types of ventricular complexes is determined by variation of the sinus rate, from 71 to 96, as opposed to a regular ectopic discharge rate of 88 (fig. 3). When the sinus rate has slowed considerably, thereby becoming markedly out of phase with the ectopic pacemaker as in the beginning of V₁, transition of ectopic to sinus rhythm takes place abruptly as a result of total capture of the ventricles by a sinus impulse. As the sinus accelerates gradually ventricular fusion beats occur (fig. 3). When sinus and ectopic rates are about equal, temporary isorhythmic A-V dissociation is observed, the

Figure 3

Longer portions of the same record as figure 2 showing that the A-V dissociation is intermittent. Transition from sinus rhythm to left posterior fascicular tachycardia and back to sinus rhythm occurs abruptly or gradually, the latter via ventricular fusion beats (F).
two impulses meeting somewhere within the A-V junction (fig. 2). The different levels of their collision could be determined by recording His bundle potentials as illustrated in figures 4 and 5.

Figure 4 shows the development and the cessation of the A-V dissociation in part of a long continuous record of lead II, together with a lead recorded simultaneously from the area of the bundle of His. A-V dissociation starts with the second beat of the upper portion and ends with the next to last beat of the lowest portion. The first and last diphasic QRS complexes are ventricular fusion beats; all others in between are pure ectopic beats (see figs. 2 and 3).

Three types of His potentials are seen (H₁, H₂, and H₁₂). These can be identified as antegrade, retrograde, and bidirectional His depolarizations on the basis of the following considerations: H₁, which is small and precedes the two ventricular fusion beats at an A-H₁ interval of 90 msec, which is equal to the A-H interval in pure sinus beats recorded elsewhere in the long record (see beat 1 in fig. 5). H₁ therefore corresponds to antegrade His depolarization produced by the sinus impulse. However, the H₁-Q (H₁ to beginning of QRS in lead II) is 10 msec shorter than in the sinus beats, indicating that ectopic discharge occurred before complete capture of the ventricles by the antegrade impulse. All H₂ deflections, which are larger and narrower than H₁, occur at the same fixed short H-Q intervals of 10 msec in front of pure ectopic beats. At the same time the atrial deflections (P and A) gradually approach the QRS and merge with it so that H₂ may at times precede A.

![Figure 4](http://circ.ahajournals.org/)

*The transition from a ventricular fusion beat to left posterior fascicular rhythm and vice versa is demonstrated in a simultaneous lead II (upper tracing in each panel) and His electrogram (lower tracing). The three panels are continuous (see text). P = surface atrial potentials; A = atrial potentials in His electrogram; H₁ = antegrade bundle of His potential; H₂ = retrograde bundle of His potential; H₁,₂ = fusion bundle of His potential.*
Obviously, then, H has lost its relation to A and has become attached to the QRS. In other words, H₂ must represent retrograde activation of the His bundle by the ectopic impulse. Two beats are preceded by potentials H₁₂ because (1) their A-H interval equals A-H₁; (2) their H-Q interval equals H₂-Q, and (3) the H deflection is intermediate in shape between H₁ and H₂. Hence, here the His bundle must have been depolarized simultaneously from both directions or, in other words, H₁₂ stands for fusion His potentials.

In order to correlate the varying shape of H deflections and length of A-H-Q intervals with the sequence of His bundle and ventricular depolarization, eight pertinent beats were selected from a record partly illustrated in figure 4 with a diagrammatic representation of impulse propagation within the A-V junction and the trifascicular ventricular conduction system. Points of fusion of the sinus and ectopic impulses are indicated by opposing arrowheads (see text). AVN = atrioventricular node; X = approximate location of His bundle recording; HB = bundle of His; LPF = posterior fascicle of left bundle branch; LAF = left anterior fascicle of left bundle branch; RBB = right bundle branch; ⋄ = site of ectopic pacemaker; → = direction of conduction. All other symbols are the same as in figure 4.
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selected from the entire long record (partly illustrated in fig. 4) and are arranged in figure 5 according to the progressively shorter P-R intervals (measured in lead II). The drawing below the individual beats indicates the postulated extent of invasion of the specialized conduction system and its three fascicles by sinus and ectopic impulses. The solid circles in the left posterior fascicle (LPF) indicate discharge of the ectopic pacemaker, the arrows the direction of activation fronts, and opposing arrowheads the level of their collision. X is the approximate position of the His electrode which was kept constant throughout the entire record.

Thus, beat 1, a pure sinus beat, reveals the shape of the antegrade His potential (H1), a normal A-H interval of 90 msec, and a slightly prolonged H1-Q interval of 0.65 msec, resulting from a minor conduction delay distal to the point of the His recording. Nevertheless, a spontaneous discharge (escape) of the ectopic pacemaker was prevented in contrast to beats 2 to 8. In beats 2 to 4, A-H1 is unchanged but H1-Q progressively shortens to 55, 45, and 30 msec. This shortening indicates increasing prematurity of an ectopic escape beat relative to the sinus impulse.

In beat 2, a ventricular fusion beat, the ectopic impulse activates part of the left ventricle by the posterior fascicle, while the rest of the ventricle is reached by the sinus impulse via the other two fascicles.

In beats 3 and 4 both ventricles are entirely captured by the ectopic impulse which also begins to penetrate backward into the His bundle. However, its collision with the sinus impulse remains distal to the point of His bundle recording (X).

Beat 5 is the first with a larger H deflection. Its distance to QRS has reached its minimum of 10 msec, but A-H is still 90 msec. Consequently, point X must have been reached simultaneously by the sinus and ectopic impulses, which appear to have shared in His depolarization to about the same extent. Deflection H1,2 can therefore be considered to be a fusion His potential.

Subsequently, in beats 6 to 8 His depolarization is represented only by deflection of H2 type with a constant H2-Q interval of 10 msec. In beat 6 it still follows A; in beat 7, H2 and A coincide; and in beat 8, H2 precedes A which is buried in the QRS. This gradual reversal of the sequence of H2 and A with a constant H2-Q interval is further evidence that H2 indeed represents retrograde activation of the major part of the His bundle by the ectopic impulse.

Discussion

Our observation indicates that QRS complexes of ventricular origin may be of almost normal duration in the standard ECG, and thereby mimic supraventricular complexes with slightly aberrant intraventricular conduction. In contrast, previous investigations were often concerned with supraventricular beats that showed aberration and/or resembled ventricular ones.6-10

The tachycardia presented here, despite absence of significant QRS prolongation in the standard ECG, is ventricular. This conclusion is based on the presence of fusion beats, retrograde activation of the His bundle, and a contour compatible with a pacemaker in the posterior fascicle of the left bundle. The complexes interpreted as retrograde His bundle potentials are not part of the P waves because their relationship with these latter complexes varies, and at times they precede the P wave. They are unlikely to be right bundle-branch potentials because, in our experience, the position of our recording catheter does not give such prominent right bundle potentials, and these potentials were not present during sinus rhythm. The fact that retrograde potentials preceded ventricular complexes does not preclude a ventricular pacemaker. Rather, it merely indicates that the pacemaker was located high in the specific conduction system, close to the branching portion of the His bundle, so that retrograde conduction time into the bundle was shorter than antegrade conduction time down to the ventricles.

An escape rhythm with QRS complexes having duration and contour similar to our
case has been described in a patient with acute inferior wall myocardial infarction and A-V block. His bundle electrograms showed retrograde His potentials with H-R intervals of 3–5 msec. The ectopic pacemaker was considered to be in the posterior division of the left bundle branch. Contour changes similar to those of the ectopic beats in the case presented here can be produced by early or late aberrance of supraventricular beats, including conduction via paraspecific fibers or spontaneous diastolic depolarization, which may cause intraventricular conduction abnormalities. The presence of retrograde His potentials reduces these possibilities. Similarly, accelerated ectopic rhythms with minor degrees of ventricular aberration and intermittent A-V dissociation previously described under the term, non-paroxysmal “A-V nodal” tachycardias, might have been ventricular in origin.

Ventricular tachycardia produced by excess digitalis in patients was demonstrated at least 47 years ago by Reid. In that report, figure 1 shows a terminal S wave in lead I and QRS complexes that are almost entirely inverted in leads II and III. QRS complexes are 0.11 sec in duration. In our case the initial 0.02-sec forces are directed inferiorly whereas the vector during the next 0.04 sec shifts to −80°. These forces result from early depolarization of the posteriorinferior wall and later depolarization of the anterolateral wall of the left ventricle. The location of the ectopic pacemaker in the posterior division of the left bundle branch produces waves of depolarization that also arrive later in the right bundle branch and right ventricle than during normal His conduction. This results in the terminal R waves in V1 and terminal S waves in I, aV1, and left precordial leads (fig. 5). Though in part delayed, because the impulse travels mainly through the specialized conducting system, only a slight widening of the QRS complex is produced.

According to Rosenbaum’s classification of ventricular extrasystoles, this pattern fulfills the criteria for a left posterior fascicular pacemaker. Ventricular tachycardia with normal QRS duration has been described in a patient soon after myocardial infarction, furthermore, hypokalemia predisposes to acceleration of ectopic rhythms by digitalis. Thus, the patient reported here had at least three factors operative that have been associated with ventricular tachycardias: ischemia, hypokalemia, and medication with digitalis. We have shown that this pacemaker may be located in one of the fascicles of the ventricular conduction system and may become so synchronized with the sinus pacemaker that it captures part or all of the ventricles, and part or all of the bundle of His.

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