Sound Pressure Correlates of the Austin Flint Murmur
An Intracardiac Sound Study

P. Sudhakar Reddy, M.D., Edward I. Curtiss, M.D., Rosemarie Salerni, M.D., James D. O'Toole, M.D., Franklin W. Griff, M.D., Donald F. Leon, M.D., and James A. Shaver, M.D.

SUMMARY Mitral valve motion and pressure correlates of the Austin Flint murmur (AFM) were investigated in nine patients with aortic regurgitation using high fidelity catheter tip micromanometers and the mitral valve echocardiogram (MVE). External phonocardiography demonstrated a mid-diastolic murmur (MDM) in eight subjects and a presystolic murmur (PSM) in five. Maximum intensity of both AFM components was found in the left ventricular (LV) inflow tract; the murmur was not recordable in the left atrium (LA). In two patients, an apparent AFM was recorded in the intracardiac phonocardiogram when absent externally. Only one subject had a significant late diastolic "reversed" or LV to LA gradient; in this patient, presystolic mitral regurgitation was shown angiographically but no PSM was present and MVE revealed absence of atriogetic mitral valve re-opening. In two subjects, a PSM disappeared from the external phono when a "reversed" gradient occurred during the diastolic pause following a ventricular premature systole; this LV to LA gradient was associated with mitral diastolic regurgitation recordable in the left atrial phono. In two patients, LV inflow phono showed the MDM to begin 80–120 msec after the aortic second sound and during the D to E phase of the MVE. The rate of early diastolic mitral valve closure in patients (152 ± 24 mm/sec) was not significantly different from 13 normals (232 ± 30 mm/sec).

With regard to the genesis of the AFM, the present study concludes: 1) diastolic mitral regurgitation plays no role, and 2) antegrade mitral valve flow is required but simultaneous retrograde aortic flow may also be necessary.

IN 1862, AUSTIN FLINT described an apical presystolic murmur in two patients with "considerable" aortic regurgitation who had no evidence of organic mitral stenosis at autopsy. This great clinician believed the murmur of mitral valve obstruction to be limited to presystole. He attributed the functional murmur to "... distension of the [left] ventricle [so] that the mitral curtains are brought into coaptation and when the auricular contraction takes place, the mitral direct current passing between the curtains throws them into vibration and gives rise to the characteristic blubbling murmur." Since its original description, the Flint murmur has been broadened to include a mid-diastolic component.

Modern investigators have assigned the genesis of the presystolic component to two mutually exclusive causes — diastolic mitral regurgitation and increased velocity of antegrade flow across a closing mitral valve. The investigative techniques utilized by proponents of the former explanation have primarily been pressure measurement and cineangiography, and of the latter cause, echocardiography. Simultaneous high fidelity micromanometer pressure determinations and intracardiac phonocardiography have not previously been reported in patients with an Austin Flint murmur. Since these two techniques would be of significant value in attempting to resolve the discrepancies posed by the above explanations, the present study was undertaken to obtain information that could further elucidate the genesis of the Austin Flint murmur.

Materials and Methods

Nine patients with aortic regurgitation undergoing diagnostic cardiac catheterization were the subjects of this investigation. Informed consent was obtained from all subjects prior to study. The valvular lesion was isolated in six and combined with moderate and mild aortic stenosis in one and two patients, respectively. Aortic regurgitation was chronic in five and acute in four. The angiographic grade (scale of 0 to 4+)* of insufficiency was 4+ in five, 3+ in two, 2+ in one, and 1+ in one patient. All patients were in sinus rhythm; three had first degree atrioventricular block.

Prior to cardiac catheterization, external phonocardiograms were recorded at the cardiac base (2nd left intercostal space) and apex simultaneously with an apaxcardiogram, indirect carotid pulse tracing, and mitral valve echocardiogram. Mitral valve echocardiography was performed on a Smith Kline Ekoline 20 A diagnostic ultrasonoscope with a ¼ inch 2.25 MHz transducer having a 7.5 cm focal length interfaced to multichannel physiologic recorders.

The rate of diastolic mitral valve closure was measured as the Fo to F slope of the anterior leaflet according to the method of Madeira et al.† The closure rate was calculated in eight study patients and 13 normal subjects.

Right, retrograde, and transseptal left heart catheterization was performed according to standard techniques. Intracardiac phonocardiograms and micromanometer pressures were obtained from Millar Mikro Tip catheters. Audio output circuitry had a flat response from 70 to 2,000 Hz with a roll-off of 12 Db per octave below 70 Hz. Left ventricular and left atrial pressures were recorded from 8F and 5F Millar catheters, respectively. Micromanometers were

From the Department of Medicine, Division of Cardiology, University of Pittsburgh School of Medicine and Presbyterian-University Hospital, Pittsburgh, Pennsylvania.

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Address for reprints: P. Sudhakar Reddy, M.D., Director, Cardiac Diagnostic Laboratories, 3411 Presbyterian-University Hospital, 230 Lothrop Street, Pittsburgh, Pennsylvania 15213.

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†Millar Instruments, Inc., Houston, Texas.
calibrated externally for 0-100 mm Hg. Micromanometer
equisensitivity was established by placing both catheters in
the left ventricle. Compensation for gravitational effects was
obtained by reference to fluid-filled pressures recorded
through the side-hole of the No. 8 Millar catheter and a No.
9 Ross catheter.

The external phonocardiogram and mitral valve echo
were repeated during the catheterization as described above.
The following simultaneous traces were obtained in five
patients: 1) external phonocardiogram at the cardiac apex,
2) left atrial and left ventricular pressure from equisensitive
micromanometer catheters, and 3) intracardiac phono-
cardiogram sequentially recorded from the left atrium and
left ventricular inflow tract. In four of these, a simultaneous
left ventricular outflow tract intracardiac phonocardiogram
was also obtained. In the remaining four patients, various
combinations of recordings at these sites were accomplished.
Aortic and left ventricular cineangiograms in the RAO posi-
tion were obtained in all patients.

Results

External Phonocardiogram (fig. 1)

Eight patients had a mid-diastolic low to mid frequency
murmur at the cardiac apex. The onset of this component
was frequently difficult to identify confidently. However, its
low frequency content and tendency to peak in mid-diastole
permitted it to be distinguished from the high frequency de-
crescendo aortic regurgitant murmur. An apical presystolic
or ariosystolic component was present in five of these eight
patients. Precise delimitation of the offset and onset of mid-
diastolic and presystolic components was difficult when the
P-R interval was prolonged or the sinus rate was rapid; the
latter tended to be the rule in acute valvular lesions (fig. 2,
right hand panel). In one patient, an Austin Flint murmur
was neither audible nor recordable externally.

Intracardiac Phonocardiogram

Figure 3 shows micromanometer pressures and internal
phonocardiograms from the left ventricular outflow and in-
flow tracts and an external phonocardiogram from the apex.
The outflow tract phonocardiogram shows a high frequency
decrescendo murmur beginning with the aortic component
of the second sound. The internal phonocardiogram from
the left ventricular inflow tract, on the other hand, reveals a
lower frequency murmur following the aortic component of
the second sound by 80 msec. In other patients, the onset of
the mid-diastolic low frequency left ventricular inflow tract
murmur followed A2 by 80-120 msec. In all patients with ex-
ternally recordable apical mid-diastolic and/or presystolic
murmurs, a similar murmur was recorded in the left ventricu-
lar inflow tract which disappeared when the catheter
was withdrawn into the left atrium.

Figure 4 shows external phonocardiogram at the apex, in-
ternal phonocardiogram recorded through the transseptal
catheter, pressures recorded through the retrograde and
transseptal catheters, and the mitral valve echocardiogram.
In the left hand panel, the retrograde catheter is positioned
in the left ventricular outflow tract and the transseptal catheter
in the left ventricular inflow tract. In the right panel, the
transseptal catheter is withdrawn into the left atrium. Both
external and internal phonocardiograms in the left panel
show a low frequency mid-diastolic murmur which dis-
appears from the internal phono when the transseptal cathe-
ter is withdrawn into the left atrium (right panel).

Figure 5 shows an external phonocardiogram from the
apex, internal phonocardiogram from the left ventricular in-
flow tract, and left ventricular and central aortic micro-
manometer pressures; these traces were recorded in a patient
with aortic regurgitation and minimal aortic stenosis who
also manifested first degree heart block. An Austin Flint
murmur is not recordable externally but mid-diastolic and
atriosystolic murmurs are evident in the left ventricular in-
flow phonocardiogram. They closely resemble the usual ex-
ternal phonocardiographic appearance of the Austin Flint
murmur.

Pressure/Sound Correlates

Figure 6 shows recordings of left atrial and left ventricular
pressures using fluid-filled (left hand panel) and micro-
manometer (right hand panel) systems in a patient with aortic
regurgitation. The former system demonstrates a peak
LA to LV gradient of 20 mm Hg approximately 60 msec
after pressure crossover; the gradient becomes negligible
when recorded micromanometrically. When measurable left
atrial to left ventricular micromanometric pressure gra-
dients were present, they occurred in early diastole and
persisted when the left atrial catheter was advanced to the left
ventricular inflow tract (fig. 4). A significant late diastolic
left ventricular to left atrial pressure gradient was present in
only one patient. Figure 2 (left hand panel) illustrates
simultaneous micromanometric and phonocardiographic
traces in this subject. A mid-diastolic crescendo-decrescen-
do murmur in the apical phonocardiogram is unassociated
with a measurable early to mid-diastolic LA to LV pressure

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*Presystolic was used to denote timing of the murmur in relation to the onset
of left ventricular systole without implying a mechanism. However, when the
P-R interval was prolonged, augmentation of the diastolic murmur with atrial
systole was not presystolic; in this situation, the term ariosystolic was
preferred.
The mitral valve echocardiogram revealed two diastolic opening movements in six of eight patients. In one of the remaining two, lack of clearly definable atrio-geic re-opening was related to P-R interval prolongation of 0.34 seconds. In the other patient, lack of atrio-geic re-opening was due to development of a significant diastolic left ventricular to left atrial pressure gradient (fig. 2, left panel). In two patients where an apical low frequency murmur following a third heart sound was recorded externally, a murmur of earlier onset was recorded from the left ventricular inflow tract phonocardiogram. In figure 4 (left panel), the onset of the latter murmur follows the aortic component of the second sound by a silent interval of 80 msec and occurs during the D to E phase of the mitral valve phonocardiogram. The E to F and F to F slopes exceeded 50 mm per second in all patients; anterior and posterior leaflet motion during diastole were always opposite in direction, i.e., no patient had echocardiographic evidence of organic mitral stenosis. The rate of mitral valve closure calculated as the Fo to F slope of the anterior mitral leaflet was 152 ± 24 (SEM) mm/sec in eight patients with aortic regurgitation. This was not significantly different from the value of 232 ± 30 mm/sec found in 13 normal subjects. Five of the latter group

gradient. The absence of presystolic accentuation of the apical murmur is accompanied by a "reversed" LV to LA gradient occurring 100-120 msec prior to the onset of isovolumic systole, an absent first heart sound and failure of atrio-geic re-opening of the mitral valve. Several study patients had a small LV to LA gradient at end diastole associated with a presystolic murmur. However, its onset was always less than 50 msec prior to the beginning of isometric contraction (fig. 2, right panel), and it was not associated with angiographically demonstrable presystolic mitral regurgitation. In addition, presystolic murmurs were recordable in the absence of end-diastolic LV to LA gradients (fig. 4, right panel).

Echo-Phono Correlates

The mitral valve echocardiogram revealed two diastolic opening movements in six of eight patients. In one of the remaining two, lack of clearly definable atrio-geic re-opening was related to P-R interval prolongation of 0.34 seconds. In the other patient, lack of atrio-geic re-opening was due to development of a significant diastolic left ventricular to left atrial pressure gradient (fig. 2, left panel). In two patients where an apical low frequency murmur following a third heart sound was recorded externally, a murmur of earlier onset was recorded from the left ventricular inflow tract phonocardiogram. In figure 4 (left panel), the onset of the latter murmur follows the aortic component of the second sound by a silent interval of 80 msec and occurs during the D to E phase of the mitral valve phonocardiogram. The E to F and F to F slopes exceeded 50 mm per second in all patients; anterior and posterior leaflet motion during diastole were always opposite in direction, i.e., no patient had echocardiographic evidence of organic mitral stenosis. The rate of mitral valve closure calculated as the Fo to F slope of the anterior mitral leaflet was 152 ± 24 (SEM) mm/sec in eight patients with aortic regurgitation. This was not significantly different from the value of 232 ± 30 mm/sec found in 13 normal subjects. Five of the latter group
and only one patient with aortic regurgitation had slopes exceeding 250 mm/sec.

**Angiographic Correlates**

The only patient with a significant LV to LA pressure gradient during mid to late diastole had cineangiographically demonstrable diastolic mitral regurgitation. However, she had neither a presystolic Flint component nor atriogenic reopening of the mitral valve on echogram. The five patients with clearly identifiable presystolic components did not manifest diastolic mitral regurgitation during the left ventricular cineangiogram.

A PVC was induced during the left ventricular angiogram in one patient with a presystolic murmur who had no resting diastolic gradient. During the pause following the PVC, left ventricular diastolic pressure exceeded left atrial pressure in early to mid-diastole extending to late diastole; this was associated with diastolic mitral regurgitation and disappearance of the presystolic murmur on the simultaneously recorded external phonocardiogram.

One patient manifested findings of special interest. Figures 7 and 8 illustrate sound-pressure and echocardiographic correlates in an individual with varying diastolic intervals due to premature ventricular systoles. Figure 7A, B, and C represent sinus diastolic intervals. A double dome mitral valve echogram correlates with an Austin Flint murmur demonstrating presystolic accentuation. The Austin Flint murmur is recordable in the external and left ventricular inflow tract phonocardiogram but not in the left ventricular outflow tract phonocardiogram.
stead, a mitral regurgitant murmur appears in the left atrium but it is not externally recorded. Diastolic mitral regurgitation and peripheral runoff cause a gradual fall in aortic and left ventricular diastolic pressures. During this interval, left atrial pressure rises slowly due to dual sources of inflow (mitral regurgitation and pulmonary venous bed) and equilibrates with left ventricular diastolic pressure. At this point, atrial systole occurs resulting in re-opening of the mitral valve and an “a” kick in the left ventricular and aortic pressure pulses, strongly suggesting that antegrade mitral flow must have taken place. At this point, a presystolic murmur cannot be identified in either the surface or left ventricular inflow tract phonocardiograms. In this case, two alternatives must be considered regarding the cause of disappearance of the presystolic component. One is failure to achieve a critical antegrade mitral flow velocity. Another equally plausible explanation is lack of simultaneous aortic regurgitant flow.

Discussion

The Austin Flint murmur is not a universal finding in patients with aortic regurgitation and any hypothesis of its genesis must be able to explain this clinical observation. Flint noted German investigators had demonstrated the forcible injection of liquid through the mitral valve would cause coaptation of its cusps. He reasoned that the rapid ventricular distension produced by “considerable” aortic regurgitation would tend to bring the mitral leaflets into apposition, thus giving rise to a functional narrowing of the effective orifice. In addition to “considerable” regurgitation, ventricular enlargement also appeared to be required; the author was careful to state that other undetermined conditions might also be necessary.

Subsequent authors postulated that specific aortic cusps had to be involved; they suggested that if the regurgitant stream achieved a critical direction, the anterior mitral leaflet would be deflected into the path of antegrade aortoventricular flow. However, postmortem studies yielded no apparent agreement on the specific lesion required.

When direct recordings of left-sided intracardiac pressures became available, it was found that fluid-filled catheter systems showed a late diastolic or “reversed” pressure gradient from left ventricle to left atrium or pulmonary capillary wedge position. Lochaya and Cohen showed early left atrial to left ventricular and late diastolic left ventricular to wedge or left atrial pressure gradients in all 12 of their patients with a Flint murmur which they defined as a mid-diastolic rumble. This characteristic pressure record was not present in five other subjects with severe aortic regurgitation who did not have the Flint murmur. They felt their finding of an early diastolic gradient was compatible with the hypothesis that the regurgitant stream impinges upon the anterior mitral leaflet interfering with complete opening. In turn, the finding of a late reversed gradient was consistent with presystolic mitral regurgitation as the origin of the presystolic component even though most of their patients did not have a recordable murmur at this phase of the diastolic cycle.
FIGURE 7  Sinus Interval. A) External phonocardiogram from base and apex, apexcardiogram (ACG) and mitral valve echocardiogram (MVE). B) External phono (apex), internal phono and pressure from central aorta (CA) and left ventricular inflow tract (LVI). C) External phono from apex, internal phono and pressure from left ventricular outflow (LVO) and LA.

FIGURE 8  Long diastolic intervals following premature ventricular beat (PB). A) External phonocardiogram from base and apex, apexcardiogram (ACG) and mitral valve echogram (MVE). B) External phono (apex), internal phono and pressure from central aorta (CA) and left ventricular inflow tract (LVI). C) External phono from apex, internal phono and pressure from left ventricular outflow (LVO) and left atrium.
Our findings indicate diastolic mitral regurgitation is not related to genesis of the Austin Flint murmur. The intracardiac phonocardiographic evidence suggests this murmur is localized to the left ventricular inflow tract. In the presence of aortic regurgitation and an Austin Flint murmur, the only recordable intracardiac diastolic murmurs were found in the left ventricle. The murmur recorded in the left ventricular inflow tract tended to have the characteristics of the externally recordable Austin Flint murmur, i.e., onset after a significant silent interval following A2 and presystolic accentuation. There was no direct correlation between presystolic murmurs and the presence or absence of a significant end diastolic LV to LA pressure gradient; in addition to the finding that this component was only recordable in the left ventricular inflow tract and not in the left atrium, presystolic mitral regurgitation was not angiographically demonstrable. Actual significant mid to late reversed gradients were associated with internal phonocardiographic and cineangiographic evidence of mitral regurgitation. Most importantly, however, the internally manifest left atrial murmur was not present externally. When a Flint murmur was present initially, the occurrence of a significant reversed gradient caused it to disappear. These data are in agreement with Pridie et al.17 who found no association between the presence of an Austin Flint murmur and angiographically demonstrable diastolic mitral regurgitation.

Large left atrial to left ventricular early to mid-diastolic gradients recorded with fluid-filled catheters tended to be minimal or absent and inconstant when recorded with high fidelity catheters. In individuals, this inconstancy appeared to be respiratory related and may have been due to a problem peculiar to micromanometer catheters, that is, sensitivity to gravitational effects. Such gradients may actually exist but could be due to flow rather than obstruction;19 these small LA to LV gradients tended to persist when the left atrial catheter was advanced to the left ventricular inflow tract supporting this hypothesis. The exaggeration of early to mid LA to LV gradients using fluid-filled systems may be due to difficulty in obtaining the requisite damping of the left ventricular catheter. These findings suggest small early LA to LV gradients cannot be used alone to support the postulate of functional stenosis as the origin of the mid-diastolic component of the Austin Flint murmur.

Fortuin and Craige8 were able to correlate the mid-diastolic component of the Flint murmur with the initial closing movement of the anterior mitral leaflet. They noted the presystolic component to occur during the atriogenic closing motion or during the entire period of atrial systole and found this component would disappear when atrial systole failed to produce reopening. Our findings are compatible with their hypothesis that antegrade mitral flow is necessary for production of the Flint murmur. They postulated that this murmur is generated by an increase in mitral flow velocity due to two conditions: 1) incomplete left atrial emptying during early diastole, and 2) excessively rapid closure of the mitral valve due to two sources of left ventricular inflow. Although Madeira et al.11 have recently provided data demonstrating an increased rate of mitral valve closure in patients with hemodynamically significant aortic regurgitation, we were unable to confirm this finding.

However, the essence of the above hypothesis is that the mitral orifice is narrowed relative to the volume of blood transported. As seen in figure 4, the presystolic component of the Flint murmur occurs during the entire period of atrial systole; this includes the F to A portion of the mitral valve echocardiogram which represents an opening motion. Fortuin and Craige attributed this finding to incomplete reopening of the mitral valve. The concept may be extended to include the mid-diastolic Flint component; thus, incomplete valve opening rather than excessively rapid closure rate may be the essential requirement for producing increased mitral flow velocity. Incomplete opening may not be identifiable by the currently employed echocardiographic or pressure recording techniques. That a closing motion of the mitral valve is not an essential requirement for genesis of the Flint murmur is suggested by the earlier onset of the mid-diastolic component during the D to E phase of the mitral valve echocardiogram in some patients. While we were unable to exclude transmission of the aortic regurgitant murmur or catheter artifact as the cause of earlier onset, this finding is also present in Fortuin and Craige’s figure 4 which utilizes an external, rather than internal, phonocardiogram.

According to the theory of murmur production advanced by Rushmer,19 a critical flow velocity is required to produce turbulence. Aortic regurgitation could create turbulent flow in the region of the left ventricular inflow tract by 1) altering the pattern and/or rate of mitral valvular flow due to its effect on left ventricular volume or pressure, or 2) intersecting with the antegrade mitral stream. If the latter is true, then the critical antegrade mitral flow velocity required to produce a murmur may be diminished in the presence of the above aortic lesion. This thesis still assigns a primary role to mitral flow velocity in genesis of the Austin Flint murmur. Thus, most patients may have the hemodynamic bases for the Flint murmur, but critical energies or flow velocities are required for transmission to the chest wall. The demonstration of an intracardiac murmur having the external phonocardiographic characteristics of an Austin Flint murmur in a patient with mild to moderate aortic regurgitation, prolonged P–R interval, and no externally recordable Austin Flint is compatible with this hypothesis. Schaefer et al.20 have recently described two patients with prosthetic mitral valves, significant aortic regurgitation and Austin Flint murmurs. In one, the mid-diastolic component occurred in the presence of a completely open prosthetic valve and in the other, the presystolic component disappeared following surgical resolution of the aortic regurgitation despite unaltered poppet motion during this phase of the cardiac cycle. In one of our patients, a presystolic Flint component disappeared following cessation of the aortic regurgitant murmur even though antegrade mitral flow must have taken place immediately prior to left ventricular systole. While alternate explanations can be invoked to explain these observations, we believe the requirement for simultaneous aortic regurgitant flow in the genesis of the Austin Flint murmur merits further investigation.

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Electrocardiographic Manifestations of Concealed Junctional Ectopic Impulses

CHARLES FISCH, M.D., DOUGLAS P. ZIPES, M.D., AND PAUL L. McHENRY, M.D.

SUMMARY Thirteen episodes of concealed junctional ectopic impulses (JEI) in ten patients are described. In nine patients the JEI manifested as isolated automatic impulses and in one as a parasystolic junctional tachycardia. In the previous description of JEI as an expected phenomenon, the following phenomena were recorded: 1) marked, greater than 400 msec, and persistent prolongation of the P-R interval, 2) striking changes in the duration of the P-R with an occasional sequence of R-P and P-R intervals simulating "supernormal" A-V conduction, 3) unexpected variation of the junctional escape interval explained by a junctional parasystole with an impulse block, 4) postoperative compensatory pause, 5) concealed junctional discharge with reciprocation.

ATRIOVENTRICULAR (A-V) BLOCK due to concealed junctional ectopic impulses (JEI) was first described in 1947.1 Not until 1962 was the next case of A-V block due to JEI reported.2 To the best of our knowledge a total of 11 instances of spontaneous JEI concealed within the junctional tissue because of the outflow tracts and a concurrent block have been reported.3,4 In each instance the presence of JEI was suspected by its effect on the behavior of the subsequent impulse. The assumption that the unexpected and "unphysiological" behavior of A-V conduction was due to concealed JEI proposed by Langendorf and Mehman5 was confirmed in man by direct recording from the His bundle by Rosen, Rahimtoola and Gunnar,6 in the dog by Damato, Lau and Bobb7 and in the isolated rabbit A-V conduction system by Moore, Knoebel and Spear.8

The purpose of this communication is to present 13 episodes of concealed JEI recorded in ten patients. Manifestations, phenomena, not previously described include: 1) initiation of a marked, greater than 400 msec, prolongation of the P-R interval; 2) persistence of this prolongation; 3) wide variation of P-R duration in the same record with occasional R-P, P-R relationship suggesting "supernormality" of A-V conduction; 4) variation of junctional escape interval; 5) postoperative compensatory pause; and 6) reciprocation due to concealed junctional discharge, as opposed to the well recognized phenomenon of concealed reciprocation.

It is also the purpose of this communication to illustrate and emphasize that much information can be learned from a careful deductive analysis of the surface electrocardiogram and that, indeed, so many of our current electrophysiological concepts were arrived at by just such a process.9 This approach is especially important in clinical electrocardiography where the observations are truly "experiments" of nature, frequently not reproducible, and where invasive procedures for their documentation or analysis are not always justifiable, nor, as illustrated in this paper, necessary.10 In this regard, his bundle electrocardiography using standard techniques11 was performed in cases 4 and 8. In case 4, the invasive study confirmed that early premature JEI conducted abnormally (fig. 4A). During His bundle electrocardiography in case 8, only intermittent JEI which conducted normally or blocked totally (concealed) were recorded. Thus, the diagnosis of a concealed junctional parasystolic tachycardia still had to be

From the Krannert Institute of Cardiology, the Department of Medicine, Indiana University School of Medicine, Indianapolis, Indiana.

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