Left Atrial Rhythm

Experimental Production in Man

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Frank W. Kroetz, M.D., and James J. Leonard, M.D.

SUMMARY

Various criteria have been proposed for the diagnosis of left atrial rhythm (LAR) in previous experimental and electrocardiographic studies. Differentiation from A-V junctional rhythms remains ill-defined. In order to assess the left atrial (LA) P wave and to define LAR, the P wave and loop were studied by direct LA pacing in 11 patients undergoing diagnostic transeptal left heart catheterization. Experimental LAR produced changes in P-wave configuration and polarity. These changes were most obvious in lead V₁ and indicated reversal of the sequence and direction of activation with left atrial preceding right atrial depolarization. A typical "dome and dart" P wave occurred in two studies. P-wave configuration in leads I and V₆ was highly variable; frequent orientation of the LA P vector within the transitional zone of the horizontal electrical axis was thought to be responsible. The LA P loop showed reversal of the direction of inscription of the initial forces, indicating a change in the pattern of atrial depolarization. Stimulation of the appendage produced left-to-right spread of atrial depolarization, whereas activation of the main body was directed primarily anteriorly. Impulse formation in the right side of the posterior wall was usually directed from right to left. When the postero-inferior area was activated, the anterior P vector became directed superiorly, and resembled that of coronary sinus rhythm (CSR) in the frontal plane.

This study indicates that the P wave in lead V₁ is most important in the diagnosis of LAR; inversion of the P wave in lead V₆ is not essential. LAR can usually be differentiated from CSR, since in the horizontal plane, activation of the LA main body is more anteriorly directed than the spatial P vector of CSR.

Additional Indexing Words:
Arrhythmia Atrial activation Atrial vectorcardiogram
Ectopic atrial pacemaker Left atrial P wave Left atrial P loop
Left atrial pacing

Left atrial rhythm, an uncommon electrocardiographic diagnosis, has received limited experimental study in the past. In 1910 Lewis¹ investigated the relationship between P-wave morphology and atrial pacemaker origin in thoracotomized dogs. In eight of nine experiments in which the left atrial appendage or the region of the "inlet of the pulmonary veins" was electrically stimulated, the ectopic atrial complex in lead II began with a negative phase. In later studies on dogs, electrical,² or chemical stimulation of the left atrium produced an ectopic P wave that was usually inverted in lead I and positive in leads II and III. In man, the configuration of the surface P wave has been altered by mechanical stimulation of the left atrium during cardiac catheterization and thoracotomy.³ Somlyo and Grayzel⁴ found that the P wave in lead I was

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Table 1

<table>
<thead>
<tr>
<th>Case no. &amp; name</th>
<th>Age</th>
<th>Final diagnosis</th>
<th>RA a/v (m)</th>
<th>RV S/ED</th>
<th>LA a/v (m)</th>
<th>LV S/ED</th>
<th>Ao. gr., mm Hg</th>
<th>AVA (cm²)</th>
<th>Mi. gr., mm Hg</th>
<th>MVA (cm²)</th>
<th>LAE</th>
<th>Pacing site</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. B.H.</td>
<td>37F</td>
<td>AS, sev.; AI, mod.</td>
<td>12/6 (6)</td>
<td>50/5</td>
<td>30/39 (26)</td>
<td>226/33</td>
<td>92</td>
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<td>0</td>
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<tr>
<td>2. M.H.</td>
<td>52F</td>
<td>MS, sev.; AI, mild</td>
<td>4/1 (1)</td>
<td>88/6</td>
<td>32/27 (20)</td>
<td></td>
<td>Not entered</td>
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<tr>
<td>3. D.F.</td>
<td>22M</td>
<td>MS, sev.; AI, mild</td>
<td>9/5 (4)</td>
<td>56/8</td>
<td>31/33 (24)</td>
<td>126/9</td>
<td>32 (pb)</td>
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<td>0.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. J.F.</td>
<td>38M</td>
<td>MS, AI, MI, mild</td>
<td>2/- (0)</td>
<td>34/2</td>
<td>21/16</td>
<td>110/1</td>
<td></td>
<td>14</td>
<td>1.6</td>
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<tr>
<td>5. E.H.</td>
<td>29F</td>
<td>MS, mod sev.</td>
<td>9/6 (6)</td>
<td>36/8</td>
<td>24/32 (20)</td>
<td>95/6</td>
<td></td>
<td>8</td>
<td>C.O. unsatis.</td>
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</tr>
<tr>
<td>6. D.A.</td>
<td>25M</td>
<td>MI, mod. sev.; MS, mod.; AI, mild</td>
<td>2/1 (1)</td>
<td>47/2</td>
<td>27/30 (20)</td>
<td>104/7</td>
<td></td>
<td>10</td>
<td>1.4</td>
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<tr>
<td>7. J.T.</td>
<td>56M</td>
<td>AS, sev.; AI, min.</td>
<td>6/2 (2)</td>
<td>33/2</td>
<td>27/24 (16)</td>
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<td>90</td>
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<td></td>
</tr>
<tr>
<td>9. J.W.</td>
<td>36M</td>
<td>AS, mod.; AI, mod.</td>
<td>3/1 (0)</td>
<td>23/0</td>
<td>10/6 (6)</td>
<td>177/88</td>
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<td></td>
</tr>
<tr>
<td>10. T.M.</td>
<td>47M</td>
<td>MS, sev.</td>
<td>5/3 (3)</td>
<td>50/4</td>
<td>34/27 (26)</td>
<td>126/6</td>
<td></td>
<td>17</td>
<td>1.0</td>
<td></td>
<td></td>
<td>Appendage</td>
</tr>
<tr>
<td>11. V.A.</td>
<td>18F</td>
<td>MI, mod. sev.; MS, AI, mild</td>
<td>5/3 (2)</td>
<td>24/1</td>
<td>11/15 (11)</td>
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<td>5</td>
<td>2.8</td>
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<tr>
<td>12. M.J.</td>
<td>41F</td>
<td>MS, sev.</td>
<td>12/8 (7)</td>
<td>48/8</td>
<td>24/32 (19)</td>
<td>105/12</td>
<td></td>
<td>12</td>
<td>0.9</td>
<td></td>
<td></td>
<td>Appendage</td>
</tr>
</tbody>
</table>

Abbreviations: AS = aortic stenosis; AI = aortic insufficiency; MS = mitral stenosis; MI = mitral insufficiency; TI = tricuspid insufficiency; sev. = severe; mod. = moderate; min. = minimal; RA = right atrium; RV = right ventricle; LA = left atrium; LV = left ventricle; a/v = a wave/V wave; (m) = mean; S/ED = systolic/end-diastolic; Ao. gr. = aortic gradient; sm = mean systolic; Mi. gr. = mitral gradient; dm = mean diastolic; AVA = aortic valve area; MVA = mitral valve area; C.O. = cardiac output; unsatis. = unsatisfactory; LAE = left atrial enlargement; angio. = cineangiography; R = right; L = left; inf. = inferior; sup. = superior; post-sup. = posterosuperior; PV-LA junc. = pulmonary vein-left atrial junction.
usually negative, occasionally diphasic, and rarely iso-electric after left atrial stimulation. Prinzmetal and associates\textsuperscript{5} reported that stimulation of the appendage generally produced a positive P wave in leads I, II, III, and aVF, whereas ectopic foci in the caudal portion of the left atrium usually created an iso-electric P wave in lead I and a negative P wave in leads II, III, and aVF. In a recent study in humans, electrical stimulation of the left atrium produced highly variable changes in the configuration of the P wave, and no specific diagnostic criteria could be established.\textsuperscript{6}

Several clinical reports have attributed ectopic rhythms showing inverted P waves in lead I to a left atrial pacemaker.\textsuperscript{4,7-10} It was theorized that initial activation in the normally positioned left atrium would result in a left-to-right direction of the atrial depolarization wave, and therefore, P-wave inversion in lead I. Recently, on the basis of P-wave vectorial analysis, inversion of the P wave in lead V\textsubscript{6} has been offered as highly specific evidence for the diagnosis of left atrial rhythm.\textsuperscript{11} Among the 18 patients who demonstrated this finding, 16 exhibited negative P waves in leads II, III, and aVF, but only seven showed inverted P waves in lead I. A “dome and dart” P wave was present in lead V\textsubscript{1} in four cases.

Obviously, the criteria for the diagnosis of left atrial rhythm are varied and overlap with electrocardiographic patterns commonly as-

\begin{table}[h]
\centering
\caption{P-Wave Morphology Before and During Left Atrial Pacing}
\begin{tabular}{|c|c|c|c|c|c|c|c|c|c|}
\hline
\textbf{CASE NO.} & \textbf{& NAME} & \textbf{I} & \textbf{II} & \textbf{III} & \textbf{AVF} & \textbf{V\textsubscript{1}} & \textbf{V\textsubscript{6}} & \textbf{SITE OF PACING} \\
\hline
1. B.H. & & & & & & & & \textbf{RIGHT INF.} & \\
& & & & & & & & \textbf{PUL. V-A JUNC.} & \\
2. M.H. & & & & & & & & \textbf{RIGHT SUP.} & \\
& & & & & & & & \textbf{PUL. V-A JUNC.} & \\
& & & & & & & & \textbf{POST-SUP.} & \\
& & & & & & & & \textbf{PUL. V-A JUNC.} & \\
5. E.H. & & & & & & & & \textbf{APPENDAGE} & \\
7. J.T. & & & & & & & & & \\
10. T.M. & & & & & & & & & \\
11. V.A. & & & & & & & & & \\
\hline
\end{tabular}
\end{table}

\textsuperscript{*Case complicated by stimulus after potential during pacing; in lead V\textsubscript{6} of subject M.H., the stimulus artifact occurred during the inscription of a prominent U wave. \\
\textsuperscript{\#Same patient and control tracing as case 3. \\
\textit{Abbreviations:} PUL V-A JUNC. = pulmonary veno-atrial junction; for other abbreviations, see table 1. \\
\textit{Note:} P waves are traced representations of original recordings obtained at a paper speed of 25 mm/sec. (See text for discussion.)
cried to the A-V junctional arrhythmias. Experimental clarification is needed.

The present study employed electrical pacing of the left atrium. It was undertaken in an effort to assess the P-wave abnormalities occurring in left atrial rhythm, and to define further the electrocardiographic criteria for its diagnosis.

**Methods**

Eleven patients undergoing diagnostic transseptal left heart catheterization for rheumatic mitral or aortic valvular disease, or both, were studied. The relevant clinical and physiological data are summarized in table 1. Six of the patients were men; five were women. Ages varied from 18 to 56 years. All were in sinus rhythm, and all had left atrial enlargement by radiographic, electrocardiographic,

\[12-14\] or both, criteria. Six patients were receiving digitalis at the time of the study.

A 4F bipolar pacemaker electrode catheter,* with distal ring electrodes placed 1 cm apart, was introduced transseptally through a 9F Ross catheter,* and selected left atrial sites were directly and repetitively stimulated using a battery-powered cardiac pacemaker.† Three subjects were paced from the appendage and one from an upper central posterior location. In six studies the left

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† Westinghouse Electric Corp., Baltimore, Maryland.

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**Figure 1**

*During pacing at the right inferior pulmonary veno-atrial junction, a negative P wave occurred in leads II, III, aVp, and V_{1} to V_{6}. The initial component of the P wave in lead I became negative. In lead V_{1} (and V_{2}) a typical “dome and dart” P wave has developed. (See text for details.) T.L. = time lines.*

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superior, right superior, and right inferior pulmonary veno-atrial junctions were each used twice as pacing sites. An eleventh subject was paced from one right atrial and two left atrial sites. Placement of the catheter electrode at the veno-atrial junctions was verified under fluoroscopy by first advancing the catheter into the respective pulmonary vein and then slowly withdrawing it to the first position where electrical impulses continuously captured the left atrium. The catheter was kept in position manually. To maintain the position of the catheter tip accurately during pacing, its placement was monitored periodically by fluoroscopy, and the oscilloscope was observed closely for stability of P-wave contour. Left atrial pacing was performed with the minimum milliampere output and the slowest possible rate sufficient to capture and maintain the experimental rhythm. Pacing rates were frequently significantly different from rates at which control data were obtained because of changes in the sinus rate during the catheterization.

Scalar electrocardiograms were recorded in each subject before and during pacing on either a triple-channel, direct-writing electrocardiographic recorder* or a polybeam photographic recorder.† Records were obtained with standard and high sensitivities at paper speeds of 25 and 50 mm/sec. In five patients high gain P-loop vectorcardiograms using the cube system of electrode placement were recorded on the polybeam recorder before and during pacing. A triggering channel was utilized for the selective photographing of the P loop.

Results

Left atrial pacing could be initiated and maintained in all patients without difficulty. The catheter tip could be kept in position accurately at all pacing sites with the exception of the region of the left atroventricular ring, where all attempts at pacing were unsuccessful. Atrial tachyarrhythmias did not occur in any of the subjects during pacing, nor did aberrant ventricular conduction. Stimulus after potential complicated one case (M. J.) and essentially negated the results of that study. All patients immediately resumed sinus rhythm upon termination of pacing.

Pacing produced distinct changes in the configuration of the surface P wave in each subject. The various P-wave morphologies encountered in the study are grouped according to pacing site and illustrated in table 2. Only the leads previously reported to be of diagnostic significance (I, II, III, aVF, V1, and V6) are shown. The newly formed P waves were analyzed at each pacing site. P-loop vectorcardiograms were evaluated for spatial orientation and direction of inscription with respect to their controls, but were not analyzed for mean manifest axis, maximum vector, long axis, or magnitude.

Right Inferior Pulmonary Veno-atrial Junction

During stimulation at this site, the initial component, or the entire P wave in leads II, III, and aVF, became negative in all three subjects, whereas the P wave in leads I and V6 showed no consistent changes (table 2). Figure 1 demonstrates the classic pattern reported for left atrial rhythm. The entire P

M.H.  CONTROL P LOOPS

FRONTAL  HORIZONTAL  RIGHT SAGITTAL

LA PACE P LOOPS

RIGHT INFERIOR PULMONARY VEIN—LA JUNCTION

Figure 2

The control P loop (upper panel) is characteristic of biatrial enlargement. During pacing (lower panel) the loop became directed entirely anteriorly after a small, posteriorly directed initial deflection. In addition, in the horizontal and frontal planes, the direction of inscription (arrows) of the initial forces reversed to clockwise. The P loops shown in this and subsequent figures are traced representations of the original P loops.
During pacing from the right superior pulmonary veno-atrial junction, the P wave remained positive in leads I and V6, contrary to common opinion regarding left atrial rhythm. Note the change in P-wave configuration and polarity in lead V1. Positive P waves in leads V1 to V6 indicate that the mean horizontal P vector is directed principally anteriorly during left atrial stimulation. Paper speed is 25 mm/sec; standardization is normal.

wave in lead V6 and the initial component in lead I became negative during pacing. In lead V1 a typical “dome and dart” P wave developed. Typical “dome and dart” P waves were not recorded in the other subjects paced from the right inferior left atrium, but changes in the configuration and polarity of the P wave in lead V1 did occur (table 2). P loops were obtained in one of these subjects (M.H.) (fig. 2). The control loops showed features of biatrial enlargement. During pacing the loop became directed primarily anteriorly and the direction of inscription of the initial forces reversed to clockwise in two planes.

Right Superior Pulmonary Veno-atrial Junction

In the two subjects (E.H. and J.F.) paced at this site, the mean frontal P vectors remained between 0° and +90° and the P waves in leads I and V6 remained positive (table 2). A scalar electrocardiogram representative of this pacing site is illustrated in figure 3. The development of upright P waves in leads V1 through V5 indicated an anterior shift in the direction of the mean horizontal P vector during left atrial stimulation. P loops recorded in case 5 were consistent with left atrial enlargement. During pacing the direction of inscription of the P loop reversed in only the right sagittal plane. In the horizontal plane the entire loop became directed anteriorly with a maximum vector* of approximately +30°; this would explain the occur-

* A straight line drawn from the iso-electric point to the most distal part of the loop.

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Figure 4
The right panel shows an enlargement of a typical "dome and dart" P wave recorded during pacing from the central posterosuperior left atrium. NSR = normal sinus rhythm; LAR = left atrial rhythm.

Figure 5
The control P loops (upper panel) suggested combined atrial enlargement; in the horizontal loop shown, the iso-electric point momentarily shifted posteriorly. During pacing the entire loop became directed anteriorly, and the direction of inscription (arrows) of the initial forces reversed to clockwise and counterclockwise, respectively, in the horizontal and sagittal planes.

Central Posterosuperior Left Atrium
"Dome and dart" P waves were observed in the one subject (D. A.) paced from this location (fig. 4). The lead I and V_6 P waves remained upright during pacing (table 2). The control P loops suggested right and left atrial enlargement (fig. 5). During pacing the entire loop became directed anteriorly, and the direction of inscription of the initial P forces was reversed in two planes.

Left Superior Pulmonary Veno-atrial Junction
Three subjects were studied during left superior veno-atrial junction stimulation. The surface P waves were variable, but in general, there was a tendency for atrial activation to be directed from left to right during pacing at this site (table 2). Two patients (nos. 8 and 9) demonstrated inverted P waves in
P-wave morphology is shown in a subject stimulated at the left superior pulmonary veno-atrial junction. The change in the configuration and polarity of the lead V1 P wave indicates reversal of the sequence and direction of atrial activation during pacing. Note that the P wave in lead V6 remains slightly positive.

lead I, and in one (case 8), the initial component of the P wave in leads II, III, aVF, and V6 became slightly negative. All three patients exhibited P-wave alterations in the precordial leads, especially in lead V1, which indicated that atrial depolarization was directed primarily anteriorly during pacing. In atrial enlargement. During pacing, in the horizontal and right sagittal planes, the loop became directed primarily anteriorly; unlike the scalar electrocardiogram, the frontal and horizontal plane P loops indicate a rightward spread of atrial activation. The direction of inscription (arrows) of the initial forces of the P loop reversed to counterclockwise in all three planes during pacing.
case 7 (J. T.) the lead V₆ P wave remained slightly positive (fig. 6). P loops recorded in this subject were compatible with left atrial enlargement (fig. 7). During pacing the loop became directed primarily anteriorly and shifted to the right. The direction of inscription of the initial component of the P loop became reversed in all three planes during pacing. In this case the P loop more closely reflected the expected left-to-right direction of atrial depolarization from this site than did the scalar electrocardiogram.

**Left Atrial Appendage**

Three patients were paced from the left atrial appendage. Two (nos. 10 and 11, table 2) showed evidence of left-to-right atrial activation with P-wave inversion in leads I and V₆. Alterations in P wave form and polarity occurred in lead V₁ in both subjects, but unlike the other left atrial pacing sites, appendage stimulation did not produce a primarily anteriorly directed, mean horizontal P vector in either patient. In one patient (no. 10 [T. M.]), the initial half of the rightward directed atrial depolarization wave had a superior and slightly posterior vector orientation (fig. 8). During pacing in case 11, atrial activation was directed inferiorly, slightly posteriorly, and rightward. In the remaining subject (no. 12), left atrial depolarization was obscured by stimulus after potential and could not be evaluated.

**Scalar Electrocardiogram**

In the scalar electrocardiogram changes in the polarity and configuration of the P wave during pacing were most obvious in lead V₁;
significant changes occurred in this lead in the left atrial pacing studies of 10 of the 12 patients. The P wave in lead V6 was highly variable, becoming initially or entirely negative in four subjects and iso-electric in two, but remained positive in five. In leads II, III, and aVF the P wave became initially or entirely negative in five studies. Similar results were found for the lead I P wave.

**P-Loop Vectorcardiograms**

These were recorded in five subjects, and the configuration of the control and paced loops varied considerably from patient to patient. The control P loops all showed evidence of left atrial enlargement, and in three, superimposed right atrial enlargement was present. During left atrial pacing, changes occurred in the direction of inscription and spatial orientation of the P loops. When the main body (posterior wall) of the left atrium was stimulated, the P loop became directed primarily anteriorly in the horizontal and sagittal planes. The P loops consistently demonstrated reversal of the direction of inscription of the initial atrial forces during pacing.

**P-R Intervals and P-Wave Duration (Table 3)**

During pacing the P-R interval varied between 0.14 and 0.26 sec, and in nine of 10 studies, increased 0.02 to 0.09 sec from control values (mean = 0.05; \(P < 0.005\)). The duration of the P wave ranged from 0.11 to 0.19 sec, and in eight of 10 subjects had increased 0.02 to 0.07 sec (mean = 0.03; \(P < 0.005\)); in most subjects, this increase accounted for most of the augmentation in the P-R interval increase. Atrial pacing prolongs atrioventricular conduction time as the paced heart rate is increased. This mechanism may have played a role in the prolongation of the P-R interval in cases 1, 4, 6, and 7; P-R interval increases of similar magnitude, however, were observed with minimal increases in the paced heart rate (cases 2, 5, 8, and 10).

**Discussion**

In clinical electrocardiography the diagnosis of left atrial rhythm is rare. This may be partly due to the limited capacity of the left atrium to serve as a pacemaker of the heart. The full pacemaker potentiality of

**Table 3**

<table>
<thead>
<tr>
<th>Name &amp; case no.</th>
<th>Control (S.R.)</th>
<th>Left atrial pacing</th>
</tr>
</thead>
<tbody>
<tr>
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<td>P-R (sec)</td>
<td>P-wave duration (sec)</td>
</tr>
<tr>
<td>1. B.H.*</td>
<td>0.22</td>
<td>0.15</td>
</tr>
<tr>
<td>2. M.H.*</td>
<td>0.17</td>
<td>0.12</td>
</tr>
<tr>
<td>3. D.F.</td>
<td>0.16</td>
<td>0.12</td>
</tr>
<tr>
<td>4. J.F.*</td>
<td>0.13</td>
<td>0.12</td>
</tr>
<tr>
<td>5. E.H.</td>
<td>0.16</td>
<td>0.10</td>
</tr>
<tr>
<td>6. D.A.*</td>
<td>0.15</td>
<td>0.12</td>
</tr>
<tr>
<td>7. J.T.*</td>
<td>0.18</td>
<td>0.12</td>
</tr>
<tr>
<td>8. D.F.†</td>
<td>0.16</td>
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</tr>
<tr>
<td>9. J.W.</td>
<td>0.16</td>
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<tr>
<td>10. T.M.</td>
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</tr>
<tr>
<td>11. V.A.</td>
<td>0.14</td>
<td>0.12</td>
</tr>
<tr>
<td>12. M.J.*‡</td>
<td>0.15</td>
<td>0.08</td>
</tr>
</tbody>
</table>

*Receiving maintenance digitalis.
†SAME patient as case 3, different pacing site.
‡Receiving maintenance quinidine.
§P-R interval and P-wave duration complicated by stimulus after potential during pacing.

**Abbreviations:** S.R. = sinus rhythm; PHR = paced heart rate; W = Wenckebach cycles.

**Note** increases in the duration of the P wave (mean = 0.03 sec; \(P < 0.005\)) and the P-R interval (mean = 0.05 sec; \(P < 0.005\)) during left atrial pacing (cases 1 and 12 excluded). (See text for discussion.)
the left atrium remains to be established by intracellular microelectrode studies, but automatic cells are probably present in the left atrioventricular ring and at the pulmonary veno-atrial junctions.\textsuperscript{21, 22}

From previous experimental\textsuperscript{1-6} and clinical\textsuperscript{7-11} data it is apparent that the frequently modified diagnostic criteria proposed for left atrial rhythm require further experimental clarification in order to differentiate this rhythm from A-V junctional rhythms.\textsuperscript{22}

Atrial depolarization does not invariably spread in a left-to-right direction following left atrial stimulation. As suggested by Massumi and Tawakkol\textsuperscript{6} and demonstrated by

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure9}
\caption{This patient was paced from one right atrial and two left atrial sites as labeled. The left atrial rhythms can be differentiated from the right atrial rhythm in the precordial leads (see text). The two left atrial sites show changes in the lead V\textsubscript{1} P wave indicating reversal of the sequence of atrial activation with left atrial depolarization preceding right atrial depolarization. Note the variability of the P waves in leads I and V\textsubscript{6}. The left-right relationships in vector orientation were consistent with the trends established for these left atrial locations.}
\end{figure}
this study, foci originating posteriorly in the extreme right portion of the left atrium can produce an activation front that moves from right to left and results in upright P waves in leads I and V6. This concept of the spatial orientation of the left atrial P wave assumes that the atrial impulse propagates concentrically through the myocardium, in spite of increasing evidence to the contrary.22, 24–27

In this study, correlation of the mean spatial P vector with the location of the left atrial pacing site demonstrated certain trends. Stimulation of the appendage consistently produced a left-to-right direction of atrial depolarization. When the main body of the left atrium was stimulated, the direction of activation was primarily anterior. As the right side of the posterior wall of the left atrium was approached, particularly the upper segment, there was a tendency for the anterior depolarization wave to be directed from right to left. When the postero-inferior area was activated, the anterior P vector became directed superiorly. This latter pattern of left atrial rhythm resembles coro-nary sinus rhythm in the frontal plane; however, we find it to be more anteriorly directed than coronary sinus rhythm and may, as a result, be differentiated from it.

Although some investigators6, 28 have presented contradictory findings, most experimental evidence indicates that the P waves in leads II, III, and aVF are negative in coronary sinus rhythm.29, 30 The superiorly oriented P vector that results is usually directed slightly anteriorly, but may be directed posteriorly with left atrial enlargement.29, 30 In coronary sinus rhythm the precordial P waves are highly variable, may be positive, diphasic, or negative in lead V1, and are frequently negative in lead V6.6, 29, 31 These studies indicate that atrial activation is less anteriorly directed in coronary sinus rhythm than in posterior left atrial rhythms and show that inversion of the P wave in lead V6 is not specific for left atrial rhythm.

That left and right atrial rhythms can be differentiated was obvious by patient D. F., who was paced at a constant heart rate from three sites; that is, the medial postero-inferior right atrium and the right inferior and left superior pulmonary veno-atrial junctions (fig. 9). Atrial activation from the two left atrial sites was directed primarily anteriorly. Whereas both inferior right and left atrial sites showed a superiorly oriented P vector with negative P waves in leads II, III, and aVF, activation from the right atrial focus was directed posteriorly with negative P waves in leads V1 to V6. Identical horizontal lead changes (P-wave inversion in leads V1 to V6) have been attributed to an anterior left atrial pacemaker.32 We were unable to stimulate the anterior left atrium in the region of the left atrioventricular ring in any subject studied. In addition, the P waves in leads I and V6 show alterations in configuration and polarity during pacing consistent with horizontal transitional zone changes.

This evidence and previous studies30, 31 suggest that impulses originating from the lower interatrial septum and adjacent right and left atrial areas may show an overlap in electrocardiographic patterns because of the superior vector orientation within the lead I and V6 transitional zone. Although in different planes, both leads I and V6 have in common the transitional zone of the horizontal electrical axis; thus, a hypothetical projection of superiorly directed vectors from the areas in question through this transitional zone might result in positive, negative, or iso-electric P waves in leads I and V6.

Using a simultaneous esophageal lead, Masumi and Tawakkol6 have demonstrated that the normal activation sequence is reversed in experimental left atrial rhythm with left atrial preceding right atrial depolarization. Although simultaneous direct or semidirect left atrial leads were not recorded in our study, other evidence suggests that the initial component of the P wave following the stimulus artifact was produced by left atrial depolarization. In the scalar electrocardiogram changes in the polarity and configuration of the P wave during pacing were most obvious in lead V1. The morphology of the terminal component of the paced P wave in lead
V₁ frequently resembled the initial, and acknowledged, right atrial component of the control sinus P wave. The terminal negative or left atrial component of the sinus P wave in lead V₁ consistently reversed polarity, immediately followed the stimulus artifact, and preceded the terminal or right atrial component during pacing. When this early, now anteriorly directed left atrial depolarization front paralleled in orientation right atrial depolarization,¹⁰ a “dome and dart” P wave occurred in lead V₁ (fig. 4). Mirowski and co-workers¹⁰ initially observed “dome and dart” P waves in the chest leads facing the right atrium of certain patients with mirror-image dextrocardia and proven atrial inversion. In this group the P wave in lead I was positive, opposite to that normally expected with atrial inversion. Later they found “dome and dart” P waves in patients with normally positioned hearts. Left atrial rhythm was diagnosed in both instances. It was postulated that the smooth, low-voltage initial component, or “dome,” represents left atrial activation; the sharp, higher peaked terminal component, or “dart,” reflects right atrial activation. Our study supports the concept that “dome and dart” P waves can originate in the left atrium.

Analysis of the P loops showed that reversal of the direction of inscription of the initial forces was a consistent finding during pacing and usually occurred in two or more planes. Although this per se did not prove that the loops originated from left atrial foci, it did indicate a change in the pattern of atrial depolarization.

The extent to which the specialized atrial conducting pathways²²-²⁶ influenced the configuration of the left atrial P wave in this study can only be surmised. The frequent resemblance of the right atrial component (terminal component) of the paced P wave to its normal sinus counterpart in lead V₁ suggests that the propagated impulse may have entered preferential conducting pathways prior to depolarization of the right atrium. The exact function of the specialized pathways during normal and spontaneous ectopic atrial activation has not been determined.

The change in configuration and increase in duration of the P wave during pacing may have been partially modified by intra-atrial and interatrial conduction disturbances²⁹ due to the increase in size and shape and the structural alteration of the diseased left atrium.

Although it is recognized that the techniques used in this study are not as accurate as those used in open-chested animals where the electrodes are sutured into an exact location, the value of the observations is enhanced by the fact that the P wave has been studied in all planes in man.

The most common clinical manifestation of left atrial automaticity may prove to be atrial flutter. The occurrence of “dome and dart” P waves in atrial flutter has been emphasized recently.³³ Thus far, the “dome and dart” P wave has been associated exclusively with left atrial impulse formation. Although the occurrence of inverted P waves in leads I or V₆, or in both leads, should arouse suspicion of a left atrial pacemaker, their presence is neither specific nor essential for a diagnosis of left atrial rhythm.

It should be kept in mind that the data in this study were obtained from a small group of heterogeneous patients showing various degrees of left atrial enlargement. The P-wave and P-loop patterns of left atrial rhythm reported herein might be less obvious when this rhythm occurs spontaneously or is induced in patients with normal left atria. From a hypothetical viewpoint, however, spontaneous left atrial rhythm might be expected to occur more frequently in an enlarged, hemodynamically and structurally altered left atrium than in a normal left atrium.

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LEFT ATRIAL RHYTHM

References


Tercentenary of Human Blood Transfusion and Medico-legal Entanglements of Human Experimentation Three Centuries Ago

... Early in 1668 a hemiplegic woman was reported to have been cured by transfusion, and in March a mentally disturbed figure of the Paris streets was twice apparently restored to good sense by a transfusion. Falling back to his former state he was scheduled for a third transfusion which was accordingly tried, but it seemed to have been abandoned before any blood had been transferred from the donor, in this case a calf. This subject died the next day and precipitated a scandal that climaxed a growing controversy that stemmed as much from the extravagant claims of the proponents of the operation as from the unyielding conservatism of its opponents. The matter came to trial in the criminal courts of Paris, with charges against Denis by the widow, and countercharges against the widow by Denis. Imputations of murder were voiced against the widow, as well as thinly veiled suggestions that the whole affair had been instigated at the behest of the Faculty of Medicine of Paris to discredit transfusion. A trial was held and a sentence rendered that fully acquitted Denis of any culpability in the death, but at the same time prohibited any further transfusions in man.—Hebbel E. Hoff and Roger Guillemin: The Tercentenary of Transfusion in Man. Cardiov Res Center Bull 6: 55, 1967.
Left Atrial Rhythm: Experimental Production in Man

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