Effects on the Canine P Wave of Discrete Lesions in the Specialized Atrial Tracts

By Albert L. Waldo, Harry L. Bush, Jr., Henry Gelband, George L. Zorn, Jr., Kari J. Vitikainen, and Brian F. Hoffman

ABSTRACT

The role of the specialized atrial tracts in determining the polarity, morphology, and duration of normal and ectopic P waves in the canine heart was studied by producing discrete surgical lesions in selected portions of these tracts. Effects of these lesions were correlated with changes in the polarity, morphology, and duration of the P waves and with conduction time to selected atrial sites when the atria were paced from the sinoatrial (SA) node, the left atrial portion of Bachmann's bundle, the low interatrial septum near the atrioventricular (AV) node, and the posterior-inferior left atrium. A lesion in the anterior internodal tract where it leaves the head of the SA node prolonged conduction time between the SA and AV nodes and significantly increased P wave duration when the atria were paced from the SA node and low interatrial septum sites. A lesion in the branch of the anterior internodal tract running through Bachmann's bundle significantly changed P wave polarity, morphology, and duration when the atria were paced from the SA node, Bachmann's bundle, and low interatrial septum sites. A lesion in the posterior internodal tract failed to change P wave polarity, morphology, or duration. When the atria were paced from the posterior-inferior left atrium, the lesions had no significant effect on P-wave polarity, morphology, or duration. It is concluded that specialized atrial tracts play a functionally important role in the sequence of atrial activation during many normal and ectopic rhythms.

KEY WORDS sequence of atrial activation ectopic atrial rhythm posterior internodal tract Bachmann's bundle anterior internodal tract

The likelihood of functionally significant specialized atrial pathways was first emphasized by Eyster and Meek (1-3). In studies on the canine heart, they observed that during sinus rhythm the impulse reached the atrioventricular (AV) node before it reached the coronary sinus or the body of the right atrium (1). Further, they observed what was called atrioventricular dissociation after discrete lesions made in the vicinity of the sinus node which left many connections between sinus node and atrial muscle intact (2). They could not account for these observations solely on the basis of radial spread of excitation, and they concluded that specialized fibers were important in atrial and sinoventricular conduction (3). Lewis (4), however, in studies on the canine heart, found that the spread of excitation in the atria was radial or syncytial, and this is not compatible with the presence of specialized conduction fibers. Lewis's arguments prevailed despite the evidence of Eyster and Meek, the anatomical studies of Wenckebach (5, 6) and Thorel (7, 8) which demonstrated at least two internodal tracts, and despite the physiological studies of Bachmann (9), later confirmed by Rothberger and Scherf (10), which showed that a lesion in the internodal band (Bachmann's bundle)
prolonged conduction time to the left atrium and changed P-wave morphology during spontaneous sinus rhythm.

However, during the past decade, anatomical and electrophysiological evidence for the presence of specialized atrial tracts has been provided by several investigators. Anatomical studies by James (11-13) and by Merideth and Titus (14) have clearly demonstrated the presence of three specialized internodal tracts (anterior, middle, and posterior) as well as a branch of the anterior internodal tract which extends to the left atrium in Bachmann's bundle. Wagner et al. (15) and Childers et al. (16) demonstrated, by microelectrode techniques, the presence of specialized fibers in Bachmann's bundle, as did Hogan and Davis (17) in the posterior internodal tract. Also, Vassalle and Hoffman (18) and Holsinger et al. (19) demonstrated, by physiological techniques, the existence of specialized paths between the sinus (SA) and AV nodes. Although the presence of specialized atrial tracts seems established, their functional role has not yet been adequately defined or established.

Recently, we presented data which suggested that these specialized pathways play an important role in determining the polarity and morphology of the P wave, which results from normal and ectopic atrial excitation in man (20). In contrast to our results, Spach et al. (21), Durrer et al. (22), and Goodman et al. (23) concluded from their recent studies on atrial activation, as did Lewis (4) more than 50 years ago and as have others (24-27) more recently, that specialized atrial pathways do not influence the sequence of atrial activation. However, Spach et al. and Goodman et al. acknowledge that there are preferential pathways for atrial conduction.

Because of this disagreement, studies were designed to further examine the role of the specialized atrial tracts in determining the polarity, morphology, and duration of the P waves and with conduction time to selected atrial sites during normal and ectopic atrial rhythms.

Methods

Twenty healthy, adult mongrel dogs weighing 20–30 kg were studied. Each dog was anesthetized with sodium pentobarbital (30 mg/kg iv), intubated, and ventilated with a Harvard respirator using room air. The arterial blood pressure was monitored continually through an indwelling catheter in the femoral artery connected to a Statham strain-gauge transducer, and displayed on a DR-12 Electronics-for-Medicine switched-beam oscilloscope. A bilateral thoracotomy in the fourth intercostal space was performed, and the heart was secured in a pericardial cradle. Acrylic plaques (28, 29), each with five silver electrodes, were sutured with 5-0 cardiovascular silk to selected sites on the epicardial surface of both atria. These sites included the head of the sinus (SA) node, the midsulcus terminalis, the caudal right atrium, the right and left atrial appendages, the left atrial portion of Bachmann's bundle (BB), the posterior-inferior left atrium (PLA), and the region of the left atrium between the PLA and the base of the left atrial appendage (Fig. 1). Two stainless steel wire electrodes were inserted through the free wall of the right atrium into the low interatrial septum (LAS) near the AV node using the technique described by Scherlag (30). The heart was examined at the end of the experiment, and if these LAS electrodes were not in the proper position, or if there was hemorrhage in the vicinity of the electrodes, the study was discarded.

The P-wave polarity and morphology were determined during bipolar threshold stimulation of the SA, BB, LAS, and PLA sites (Fig. 1). These four sites were selected for several reasons: (1) P-wave polarity and morphology could be compared during anterograde and retrograde activation of the atria from both left and right atrial sites. (2) Each site is widely separated from the others (24–27) more recently, that specialized atrial pathways do not influence the sequence of atrial activation. However, Spach et al. and Goodman et al. acknowledge that there are preferential pathways for atrial conduction.

Because of this disagreement, studies were designed to further examine the role of the specialized atrial tracts in determining the polarity, morphology, and duration of normal and ectopic P waves of the canine heart by producing discrete surgical lesions in selected portions of the specialized atrial tracts. Effects of these lesions were then correlated with changes in the polarity, morphology, and duration of the P waves and with conduction time to selected atrial sites during normal and ectopic atrial rhythms.
Four views of the canine atria. Pacing sites are shown with an arrow. A: Location of the posterior-inferior left atrial (PLA) electrode site just posterior to the inferior vena cava (IVC), below the right inferior pulmonary vein (PV) and above the coronary sinus (CS). LV = left ventricle; RV = right ventricle; PV = pulmonary vein. B: Location of several electrode sites on the right atrium: SA = sinoatrial node; 1 = midsulcus terminalis; 2 = caudal right atrium; 3 = right atrial appendage. SVC = superior vena cava. C: Location of several electrode sites on the left atrium: BB = Bachmann’s bundle; 4 = left atrial appendage; 5 = region between posterior-inferior left atrium and the left atrial appendage. D: Low atrial septal (LAS) site near the AV node in the right atrium. The free wall of the right atrium has been cut out for visualization of the interatrial septum. RAA = right atrial appendage; CSO = coronary sinus ostium.

Pacing sites were measured directly from the oscilloscopic grid of a Tektronix 502 oscilloscope whose sweep was triggered from the pacing stimulus at a sweep speed of 1 mm/msec.

After control tracings and conduction times to selected sites were obtained, a discrete surgical lesion was made (Fig. 2) in the anterior internodal tract where it leaves the head of the sinus node, in the branch of the anterior internodal tract running through Bachmann’s bundle on either the the right or left atrial side of the interatrial septum, or in the posterior internodal tract just caudal to the electrode in the midsulcus terminals (Fig. 1). The lesions transected the atrial wall, and hemostasis was obtained by securing a purse-string suture around the lesion. The lesions measured 0.6–0.7 cm in length, i.e., slightly more than the width of a no.
LESIONS IN THE SPECIALIZED ATRIAL TRACTS

11 scalpel blade. Blood flow to the SA node was never completely interrupted, because at least one branch of the SA nodal artery was always left intact (31, 32). For each dog, the P waves and conduction times recorded prior to the first lesion served as their controls for the data obtained after subsequent experimental procedures. After a lesion was made in one of the tracts and the postlesion studies were performed, an additional lesion was made in another pathway, and studies were repeated.

Results
P-WAVE POLARITY, MORPHOLOGY, AND DURATION
When the atria were paced from the SA node region, the low interatrial septum near the AV node, or the left atrial portion of Bachmann's bundle, i.e., from the sites close to the specialized atrial pathways, the P waves in electrocardiographic leads II, III, and aVf, were always positive (Fig. 3). When the atria were paced from the posterior left atrium, a site distant from the specialized atrial pathways, the P waves in leads II, III, and aVf, were always deeply negative. The polarity and morphology of the P waves were recorded when the atria were paced from these four sites with the chest open and closed. When comparisons were made, the polarity of the P waves recorded with the chest open was identical to that recorded with the chest closed, and the morphology of the P waves differed to an insignificant degree. These results confirm previous observations (20, 21, 33).
Representative example from one experiment of the typical P wave recorded in ECG lead II when the atria were paced at threshold from the SA, LAS, BB, and PLA electrode sites. Recording speed 50 mm/sec. SA = sinoatrial node; LAS = low interatrial septum; BB = Bachmann's bundle; PLA = posterior-inferior left atrium.

When the atria were paced from the SA site, the mean duration of the P wave in lead II was 61 ± 7 msec (SD); from the LAS site, the mean duration was 57 ± 8 msec; and from the BB site, the mean duration was 57 ± 9 msec. There was no significant difference in the duration of the P waves recorded when the atria were paced from these three sites (P > 0.05). When the atria were paced from the PLA site, the mean duration of the P wave in lead II was 76 ± 10 msec. This value was significantly greater (P < 0.001) than those recorded when the atria were paced from the SA, LAS, and BB sites (Table 1).

Thus, in terms of polarity, morphology, and duration, the P waves were essentially the same when the atria were paced from the three sites in proximity to the specialized atrial pathways. When the atria were paced from the PLA site, which is remote from known specialized atrial pathways (11-14), the P waves were deeply negative in leads II, III, and aVF and also were of increased duration.

EFFECTS OF INTERRUPTION OF THE SPECIALIZED PATHWAYS

Pacing SA Electrode Site.—Figures 4 and 5 illustrate the P-wave polarity and morphology in lead II and the conduction times to selected atrial sites when the atria were paced through the SA electrodes. As illustrated in both Figures 4 and 5, conduction times to the left atrial portion of Bachmann's bundle and the midsulcus terminalis were the shortest of those measured, followed closely by conduction times to the caudal right atrium, the right atrial appendage, and the LAS electrode sites. Conduction time to the PLA electrode site was the longest when the SA site was paced. Conduction time to the BB site was always shorter than to the other left atrial sites. This could not be explained solely on the basis of distance from the stimulus site and supports the work of several investigators (15, 21, 23) who have observed a rapid velocity of

<table>
<thead>
<tr>
<th>Site paced</th>
<th>P-wave polarity</th>
<th>Mean P-wave duration (msec)</th>
<th>P value</th>
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<tr>
<td>SA</td>
<td>(+)</td>
<td>61 ± 7</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>LAS</td>
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<td>57 ± 8</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>BB</td>
<td>(+)</td>
<td>56 ± 9</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>PLA</td>
<td>(-)</td>
<td>76 ± 10</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

P values >0.05 indicate that duration of the P waves of the test population do not significantly differ from those of control population, whereas P values <0.05 indicate that duration of the P waves of the test population do significantly differ from the control population. The P waves recorded when the SA site was paced served as the control.

P-wave polarity was recorded from ECG leads II, III, and aVF. Mean P-wave duration was determined from ECG lead II and is given as mean ± SD.

SA = sinoatrial node; LAS = low interatrial septum; BB = Bachmann’s bundle; PLA = posterior-inferior left atrium.
Lesions in the Specialized Atrial Tracts

A: Control P wave recorded from lead II with the conduction time to several recording sites illustrated. The atria were paced from the SA electrode. B: P wave recorded from lead II after creation of a lesion in the anterior internodal tract where it leaves the head of the SA node. Changes in conduction time are shown. Conduction times to each recording site are given in Table 6. AT = anterior internodal tract as it leaves the head sinus node; S = stimulus artifact.

The P waves on this and all the remaining figures have been enlarged 10x for purposes of illustration.

Conduction in Bachmann's bundle, but not over the remainder of the left atrium.

When the anterior internodal tract was cut where it leaves the head of the sinus node, the P-wave duration increased significantly when compared with the control P wave (P < 0.05, Table 2). However, the polarity of the P wave remained the same (Fig. 4). Conduction times to the LAS site and all the left atrial sites increased by 12-20 msec in a representative experiment (Fig. 4). However, the morphology of the P wave remained about the same, probably because the relative sequence of atrial activation changed little compared with the control P wave, as evidenced by activation of the LAS and BB sites before or at the peak of the P wave and the activation...
TABLE 2

P-Wave Polarity and Duration before and after Lesions when SA Site Paced

<table>
<thead>
<tr>
<th>Lesion</th>
<th>P-wave polarity</th>
<th>Mean P-wave duration (msec)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>(+)</td>
<td>61 ± 7</td>
<td></td>
</tr>
<tr>
<td>AT</td>
<td>(+)</td>
<td>69 ± 7</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>BB(RA)</td>
<td>(+, -) or Isoelectric</td>
<td>78 ± 6</td>
<td>&lt;0.01</td>
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<tr>
<td>BB(LA)</td>
<td>(+, -)</td>
<td>76 ± 3</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>PT</td>
<td>(+)</td>
<td>62 ± 4</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Significance of statistical P values and recording procedures for P-wave polarity and mean duration are the same as in Table 1. Mean duration values are means ± SD.

AT = lesion in the anterior internodal tract where it leaves the head of the sinus node; BB(RA) = lesion in the right atrial portion of the branch of the anterior internodal tract which runs in Bachmann’s bundle; BB(LA) = lesion in the left atrial portion of the branch of the anterior internodal tract which runs in Bachmann’s bundle; PT = lesion in the posterior internodal tract; SA = sinoatrial node.

of the left atrial appendage, the left atrial region between PLA and the left atrial appendage, and PLA during a similar portion of the downslope of the P wave.

This study emphasizes the important role of the anterior internodal tract in preferentially carrying the wave of excitation to the low interatrial septum and AV node, thus confirming the observations of Eyster and Meek (1) and Holsinger et al. (19), and to the left atrium. However, while this lesion consistently increased conduction times to the LAS and left atrial sites, alternate pathways of conduction were readily available because the relative sequence of activation of the atrial sites and the polarity of the P waves were unchanged, and the morphology of the P waves was changed minimally.

When the lesion was created first in the left atrial portion of Bachmann’s bundle adjacent to the interatrial septum and the atria were paced from the sinus node electrode, the mean duration of the P wave increased significantly when compared with the control P wave (P < 0.02; Table 2). Representative changes in the P-wave polarity and morphology after the lesion are shown in Figure 5. The amplitude of the P wave was markedly decreased and the second half became slightly negative or isoelectric. Note in Figure 5 that the appearance of a slightly negative terminal portion of the P wave is associated with prolonged conduction times to Bachmann’s bundle, left atrial appendage, and the left atrial region between the PLA and the left atrial appendage. Also note that conduction times to the LAS, the right atrial, and the PLA sites were stable. This was observed whether the lesion was in the right or left atrial portion of Bachmann’s bundle or if both cuts were made sequentially. Thus, when a lesion was created in the right atrial portion of Bachmann’s bundle adjacent to the interatrial septum, P-wave polarity, morphology, and duration changed in a manner similar to that when the left atrial portion of Bachmann’s bundle was severed. If, after a lesion was made in the right atrial portion of Bachmann’s bundle, a second lesion was made in the branch of the anterior internodal tract running through the left atrial portion of Bachmann’s bundle, the P-wave polarity, morphology, and duration did not change significantly and conduction times to all electrode sites were essentially unchanged.

The stability of the conduction times from the sinus node pacing site to the LAS recording site indicates that the lesions created in Bachmann’s bundle involved only the branch of the anterior internodal tract running through Bachmann’s bundle and left the anterior internodal tract intact in its course to the AV nodal region. These findings also confirm the anatomical location of this specialized atrial pathway as described by James (12).

A lesion in the posterior internodal tract failed to change P-wave polarity, morphology, or duration and only increased conduction time to the caudal right atrium site.

Therefore, when the atria were paced from the sinus node electrodes, the anterior internodal tract appeared to play a dominant role in early activation of the LAS (and therefore the AV node) and the BB recording sites. If a lesion was created at the head of the SA node,
lesions in the specialized atrial tracts

Conduction time to the LAS and BB recording sites was prolonged; but the integrity of the anterior internodal tract at this site was not critical for maintaining normal P-wave polarity and morphology. However, the integrity of the branch of the anterior internodal tract running through Bachmann's bundle was critical for maintaining normal P-wave polarity and morphology. Thus, a conduction defect between the right and left atria in Bachmann's bundle was associated with abnormal P-wave polarity and morphology. It would seem that the major portion of the left atrium depends on the branch of the anterior internodal tract running through Bachmann's bundle for normal activation during sinus rhythm, thus confirming the early observations of Bachmann (9) and Rothberger and Scherf (10) and later observations of others (21, 23). Therefore, when the excitation of the right and left atria was no longer temporally integrated by conduction through the branch of the anterior internodal tract in Bachmann's bundle, abnormal P-wave polarity, morphology, and duration were recorded.

Pacing LAS Electrode Site.—When the atria were paced retrograde from the LAS site near the AV node, a positive P wave was always reported from leads II, III, and aV, (Figs. 3, 6, 7). The mean P-wave duration was 57 ± 8 msec (SD). The BB and SA sites were always activated relatively early during the inscription of the P wave (Fig. 6, 7). Activation of the BB site always preceded activation of the SA site, much as one would predict if retrograde activation of the atria from the LAS site proceeded up the anterior internodal tract (20). The PLA electrode site was activated late in the P wave in all experiments. Conduction time to the BB site was much shorter than to the other left atrial sites not located over specialized atrial pathways (left atrial appendage, region of the left atrium between PLA site and left atrial appendage, PLA).

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A: Control P wave recorded from ECG lead II when the atria were paced from the LAS site. Conduction time to the several recording sites is illustrated. B: P wave recorded from lead II after creation of a lesion in the anterior internodal tract where it leaves the head of the SA node. Changes in conduction time are shown. C: P wave recorded from ECG lead II after creation of an additional lesion in the left atrial (LA) portion of Bachmann's bundle (BB). Changes in conduction time are shown. Conduction times to each recording site are given in Table 6. AT = anterior internodal tract as it leaves the head of the sinus node; S = Stimulus artifact; LAS = low interatrial septum; SA = sinoatrial node.
After a lesion was made in the anterior internodal tract where it leaves the head of the sinus node, the mean duration of the P wave increased significantly when compared with the control P wave ($P < 0.05$; Table 3). However, as illustrated in a representative experiment, the P-wave polarity and morphology remained grossly unchanged except that the peak of the P wave was delayed (Fig. 6B). Conduction times to the SA node, mid-sulcus terminalis, and the right atrial appendage sites increased, while conduction times to the left atrial and caudal right atrium recording sites remained unchanged. When a second lesion was then created in the same heart in the left atrial portion of Bachmann's bundle (Fig. 6C), the P-wave polarity and morphology changed radically and the duration of the P wave increased significantly to 97 msec when compared with the control P wave ($P < 0.02$). As illustrated in the recording from lead II, the initial two-thirds of the P wave became almost isoelectric (note that it is very slightly positive in lead II) and the last one-third became frankly negative. This was associated with a marked increase in conduction times to Bachmann's bundle, left atrial appendage, and the region of the left atrium between the left atrial appendage and the PLA (20, 38, and 28 msec respectively). Conduction times to the right atrial recording sites and to the PLA site were not affected by this second lesion.

When a lesion was first made in the left atrial portion of Bachmann's bundle, conduction times from the LAS to the BB and left atrial appendage sites always increased significantly when compared to control conduction times. As illustrated in a representative experiment (Fig. 7), this was associated with a significant change in the polarity and morphology of the P wave (Fig. 7B, Table 3), which became biphasic (+, −) in leads II, III and aVf. Conduction time to the region of the left atrium between the atrial appendage and the PLA was increased in only half of the studies. Note that conduction time to several

![Diagram](image)

**TABLE 3**

<table>
<thead>
<tr>
<th>Lesion</th>
<th>P-wave polarity</th>
<th>Mean P-wave duration (msec)</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>(+)</td>
<td>57 ± 8</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>AT</td>
<td>(+)</td>
<td>67 ± 8</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>BB(RA)</td>
<td>(+, −)</td>
<td>66 ± 3</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>BB(LA)</td>
<td>(+, −)</td>
<td>55 ± 4</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

LAS = low interatrial septum. All other abbreviations are the same as in Table 2.
right atrial and PLA sites was unaffected by this lesion.

A lesion created in the posterior internodal tract failed to change P-wave polarity, morphology, duration, or conduction time to any recording sites (Table 3).

This series of experiments demonstrates that when the canine atria are paced from a site near the AV node (LAS), the integrity of the branch of the anterior internodal tract running through Bachmann’s bundle is critical for maintaining normal P-wave polarity and morphology; furthermore the anterior internodal tract provides a rapid pathway for retrograde conduction from the LAS to the SA node. However, it is clear that the sequence of activation of the left atrium determines the polarity and morphology of the P wave when the atria are depolarized in a retrograde fashion when the LAS site is paced. It is tempting to speculate that the negative portion of the P wave in leads II, III, and aVf, which was recorded following a lesion in the left atrial portion of Bachmann’s bundle, resulted from retrograde activation of a significant portion of the left atrium rather than from anterograde activation proceeding from Bachmann’s bundle (20).

Pacing BB Electrode Site.—When the atria were paced from the left atrial portion of Bachmann’s bundle near the base of the left atrial appendage (Fig. 3, 8, 9), a positive P wave was always recorded from leads II, III, and aVf. The mean P-wave duration was 56 ± 9 msec (sp). Note that as illustrated in two representative experiments (Fig. 8, 9), the LAS recording site was activated relatively early during the inscription of the P wave. The PLA site was the last to be activated, except for the right atrial appendage site on two occasions.

When the lesion was created first in the right atrial portion of Bachmann’s bundle (Fig. 8), the P wave became markedly biphasic, and the mean duration of the P wave increased significantly (Table 4). Conduction times to the left atrial recording sites were stable in this experiment but increased to the LAS, the SA, midicus terminalis and the

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TABLE 4
P-Wave Polarity and Duration before and after Lesions when BB Site Paced

<table>
<thead>
<tr>
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<td>&lt;0.02</td>
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<td>BB(LA)</td>
<td>(+, -)</td>
<td>70 ± 5</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>PT</td>
<td>(+)</td>
<td>63 ± 4</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

BB = Bachmann's bundle. All other abbreviations are the same as in Table 2.

right atrial appendage by 26, 20, 5, and 19 msec, respectively. Note that a terminal negative component of the P waves in leads II, III, and aVf is associated with these changes. A similar pattern was observed when the lesion was created in the left atrial portion of Bachmann's bundle between the pacing site and the anterior internodal tract where it leaves the sinus node (Fig. 9C).

When the lesion was first created in the anterior internodal tract where it leaves the head of the sinus node (Fig. 9), the P-wave polarity and morphology were unchanged, and the mean duration of the P wave did not increase significantly when compared with the control P waves (Table 4). As illustrated in Figure 9, conduction times to the SA node were prolonged without changes in conduction time to the other recording sites. Also of interest, though not readily explained, was the absence of any change in conduction time to the midsulcus terminalis. When a second lesion was created in the left atrial portion of Bachmann's bundle between the pacing electrodes and the interatrial septum (Fig. 9C), the mean duration of the P wave increased significantly and the P wave became flatter and biphasic (+, -). Conduction times to all the left atrial sites were unchanged or very minimally increased, while the conduction times to the LAS, SA, midsulcus terminalis, caudal right atrium, and right atrial appendage sites were prolonged by 20, 24, 27, and

are shown. Conduction times to each recording site are given in Table 6. S = Stimulus artifact.

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A: Control P wave recorded in ECG lead II when the atria were paced from the PLA site. Conduction times to the several recording sites are shown. B: P wave recorded in lead II after creation of a lesion in the anterior internodal tract where it leaves the head of the SA node (AT). A change in conduction time is shown. C: P wave recorded in lead II after creation of an additional lesion in the left atrial (LA) portion of Bachmann's bundle (BB). Changes in conduction time are shown. Conduction times to each recording site are given in Table 6. S = Stimulus artifact.

When a lesion was created in the posterior internodal tract, there was no change in P-wave polarity, morphology, or duration (Table 4), but there was a slight increase of conduction time to the caudal right atrium site.

Therefore, for left atrial rhythms originating near the specialized atrial tissue in the distal portion of Bachmann's bundle, the integrity of the branch of the anterior internodal tract running through Bachmann's bundle was critical for maintaining normal P-wave polarity, morphology, and duration and normal conduction time to the AV node. The severing of this interatrial pathway significantly disrupted the normal sequence of atrial activation and temporally dissociated the sequence of activation of the right and left atria. In comparison, a lesion in the anterior internodal tract where it leaves the head of the sinus node did not significantly alter P-wave polarity, morphology, and duration or conduction time to the AV node, because the latter lesion did not significantly alter the sequence of atrial activation.

Pacing PLA Electrode Site.—When the atria were paced from the PLA site, the P waves in leads II, III, and aVf, were uniformly negative (Fig. 3, 10). The mean duration of the P wave, 76 ± 10 msec (SD), was statistically longer than when the atria were paced from the three sites near specialized atrial conducting fibers (Table 1). Conduction times to the atrial recording sites for one representative study appear in Figure 10. Activation of the Bachmann's bundle and the low interatrial septum always occurred well after the peak of the P wave. Sinus node activation occurred quite late during the inscription of the P wave.

Single or sequential lesions through the anterior internodal tract where it leaves the head of the sinus node, the branch of the anterior internodal tract running through
TABLE 5
P-Wave Polarity and Duration before and after Lesions when PLA Site Paced

<table>
<thead>
<tr>
<th>Lesion</th>
<th>P-wave polarity</th>
<th>Mean P-wave duration (msec)</th>
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<td>BB(RA)</td>
<td>(-)</td>
<td>82 ± 10</td>
<td>&gt;0.05</td>
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<tr>
<td>BB(LA)</td>
<td>(-)</td>
<td>83 ± 9</td>
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<td>PT</td>
<td>(-)</td>
<td>84 ± 11</td>
<td>&gt;0.05</td>
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PLA = posterior-inferior left atrium. All other abbreviations are the same as in Table 2.

Bachmann's bundle, or the posterior internodal tract did not change the polarity of the P wave, nor did they significantly change the morphology (Fig. 10) or the mean duration of the P wave (Table 5), although conduction time to a few sites was increased somewhat (Fig. 10). Therefore, when the atria were paced from an area which is devoid of specialized atrial fibers, the specialized atrial tracts played no significant role in determining the sequence of atrial activation or the P-wave polarity, morphology, or duration.

**Discussion**

Several conclusions can clearly be drawn from these studies. First, if one accepts the premise that excitation of the atria spreads radially and at a uniform velocity (4, 24-27), then a small lesion anywhere in the atria should have no significant effect on P-wave polarity, morphology, or duration or on conduction time to parts of the atria distant from the point of activation. However, the fact that small, discrete lesions did significantly change P-wave polarity, morphology, and duration as well as selectively increase conduction time from the stimulus site to distant parts of the atria provides evidence that specialized conduction pathways were interrupted and that these pathways play an important role in the sequence of activation of the atria.

Second, of the specialized atrial tracts, the integrity of the branch of the anterior internodal tract running through Bachmann's bundle is most important in determining P-wave polarity and morphology when the atria are activated from the SA, LAS, and BB sites which significantly depend on the specialized atrial tracts for their normal sequence of atrial activation. Thus, when the atria are paced from the posterior-inferior left atrium, the integrity of this branch is not important for maintaining normal P-wave polarity and morphology, because activation of the atria from this site does not depend primarily on the integrity of the specialized atrial tracts (23). For similar reasons, the integrity of the anterior internodal tract or its branch in Bachmann's bundle, or both, is important in maintaining normal P-wave duration when the atria are activated from the SA, LAS, and BB, but not from the PLA sites.

Third, the anterior internodal tract provides the main pathway for rapid activation between the SA and AV nodes. This is evident from the studies in which the SA site and LAS site were paced. Interruption of the anterior internodal tract where it leaves the head of the SA node prolonged conduction time anterograde from the SA to the LAS and retrograde from the LAS to the SA sites. Eyster and Meek (34) were the first to demonstrate that lesions in the vicinity of the SA node on three sides, i.e., from the free wall of the right atrium and from what are now recognized as the anterior and posterior internodal tracts, but leaving intact the middle internodal tract. More recently, Holsinger et al. (19) demonstrated that a lesion in the posterior internodal tract failed to prolong conduction time to the AV node (which we confirmed in this study), but when a lesion was made in the anterior and middle internodal tracts simultaneously, conduction time to the AV node increased. Thus these previous studies are quite compatible with the conclusion drawn from our studies. It should be noted that this conclusion contradicts Goodman et al. (23) who concluded from their recent studies that the posterior internodal pathway was the main pathway for conduction between the SA and AV nodes. It is
LESIONS IN THE SPECIALIZED ATRIAL TRACTS

TABLE 6

Conduction Times (msec) Measured from the Stimulus Artifact to Each Recording Site for Figures 4-10

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Recording sites: BB = Bachmann's bundle; SA = sinoatrial node; MST = midendocardial terminalis; RAA = right atrial appendage; CRA = caudal right atrium; LAS = low interatrial septum; LAA = left atrial appendage; BLA = region of the left atrium between the posterior-inferior left atrium and the base of the left atrial appendage; PLA = posterior-inferior left atrium.

reasonable to extrapolate from our data to clinical situations. Recently, we (35) have demonstrated that patients with ostium primum atrial septal defects have prolonged conduction times from the SA to the AV node, thus explaining the first-degree heart block characteristic of this lesion. We have suggested that in these patients the lesion interrupts or distorts the anterior and middle internodal tracts, resulting in prolonged conduction time to the AV node. Also, Narula et al. (36) demonstrated with the catheter recording technique that patients may show prolonged "P-A times," i.e., prolonged SA node to AV node conduction times. It seems reasonable to suggest that in this latter example, fibrotic lesions or the like have interrupted conduction in a specialized tract, probably the anterior or both the anterior and middle internodal tracts, resulting in the increased P-A time.

Fourth, as we have previously suggested (20, 29), the polarity, morphology, and duration of the P wave are a poor indication of the site of origin of atrial activation. Clearly the P waves recorded when the atria were paced from the SA, BB, and LAS sites were of the same polarity and duration and of such similar morphology that they could not be used as an indicator of pacemaker locus. Further, it is clear that a discrete lesion in the atria could significantly change P-wave polarity, morphology, and duration while the pacemaker site remained unchanged. Extrapolating from this to the clinical situation, a pathologic lesion in the specialized atrial tracts could similarly distort P-wave polarity, morphology, and duration without the pace-
maker site having changed; one then would be greatly misled in suggesting that an atrial pacemaker site was not the SA node because the P wave in leads II, III and aVt was biphasic (+, −).

Finally, several points bear emphasis: (1) There is clear anatomic evidence for specialized atrial tracts (11-14). (2) Both during normal and ectopic atrial rhythm, rapid activation along pathways consistent with the anatomic location of the specialized tracts have been recorded (17, 18, 21, 23). (3) Discrete lesions at sites consistent with the anatomic location of these specialized tracts produced significant changes in P-wave polarity, morphology, and duration and in conduction time from the activated site to distant atrial recording sites. Therefore, it seems quite reasonable and, in fact, appropriate to recognize that the specialized atrial tracts play a functionally important role in the sequence of atrial activation during normal and many ectopic rhythms.

References
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Effects on the Canine P Wave of Discrete Lesions in the Specialized Atrial Tracts
Albert L. Waldo, Harry L. Bush, Jr., Henry Gelband, George L. Zorn, Jr., Kari J. Vitikainen and Brian F. Hoffman

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