Inferior Interatrial Pathway in the Dog

By Benjamin J. Scherlag, Billy K. Yeh, and Morton J. Robinson

ABSTRACT

In 20 anesthetized dogs, close bipolar electrograms consisting of two deflections were recorded along the ligament of Marshall in the posterior left atrium. At the inferior portion of the ligament the deflections were separated by an interval of 10-15 msec and at the level of the left superior pulmonary vein by an interval of 60 msec or more. Histological and electrophysiological studies indicated that both deflections were associated with striated cardiac muscle; the first deflection was designated as a left atrial muscle potential and the second as a left atrial tract potential. Although the left atrial muscle potential was coincident with the middle portion of the P wave, the left atrial tract potential occurred during some part of the P-R segment. Conduction to the left atrial tract potential was altered, delayed, or completely blocked during retrograde conduction in the atrium, rapid pacing of the atrium (300-500/min), or local cooling at the point where the ligament of Marshall joined the inferior left atrium near the origin of the coronary sinus. Crushing Bachmann’s bundle caused delay in conduction to the left atrial muscle but not to the left atrial tract. Left cardiac sympathetic nerve stimulation induced an atrial activation pattern consistent with pacemaker activity in the inferior left atrium, with inversion of the P wave in lead II. Pacing from the edge of the ligament of Marshall in the posterior left atrium produced the same change in P-wave morphology. These data indicate that the left atrial tract represents a terminal, insulated tract which is activated through an inferior interatrial pathway connecting the inferior right and left atria along the coronary sinus. The terminal end of the tract showed no reinsertion into atrial musculature.

KEY WORDS
bipolar electrograms  His bundle recordings
P waves  left atrial rhythms  ligament of Marshall  atrial tracts
cardiac sympathetic nerve stimulation  cardiac pacing  Bachmann’s bundle

Recently there has been a revival of interest in the patterns of atrial activation based primarily on the work of James (1), who described histologically distinct atrial tracts running from the sinoatrial (SA) node to the atrioventricular (AV) node. In 1850 Marshall (2) described the prenatal development of an area of the left atrium running between the superior and inferior left pulmonary veins, which became a ligamentous fold. This structure, now referred to as the ligament of Marshall, is a vestigial tissue encompassing portions of the embryonic sinus venosus and left cardinal vein (3). In the present study, we have recorded bipolar electrograms along the ligament of Marshall in the posterior left atrium consisting of two distinct atrial deflections. The data obtained during various physiological interventions and histological studies suggest an interatrial pathway connecting the posterior and the inferior portions of the right atrium in the area of the AV node with the inferior-posterior left atrium along the coronary sinus. In addition, the evidence suggests that the tract of atrial muscle running within the ligament of Marshall is the distal portion of this pathway and terminates "blindly," i.e., it does not reinsert into any portion of the atria. Evidence obtained from pacing along the edge of the ligament of Marshall and the immediately adjacent atrial
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musculature suggests a physiological basis for the similar morphology of P waves seen in AV nodal and some low right atrial rhythms and those obtained during stimulation of the posterior and inferior portions of the left atrium (4).

Methods

Twenty mongrel dogs, weighing 14–20 kg, were anesthetized with sodium pentobarbital (30 mg/kg, iv). Under controlled ventilation, a left thoracotomy was performed at the fourth intercostal space, the pericardium was incised, and the left atrium and base of the left ventricle were exposed. In some cases, the heart was exposed via a midsternotomy to more conveniently record from the epicardial surfaces of the right and left atria. Recordings were made using (1) bipolar plunge wire electrodes consisting of two Teflon-coated stainless steel wires (0.007 inches in diameter) passed through 22-gauge hypodermic needles and (2) an exploring bipolar electrode. The distance between the bipolar pairs of electrodes was approximately 2 mm. The technique of recording bipolar electrograms from the epicardial or endocardial surface of the heart using plunge wires has been previously described (5).

A left atrial map using number and letter coordinates was constructed to obtain consistent positioning of the exploring electrode on various portions of the appendage and body and on the posterior region of the left atrium in the area between the inferior and superior pulmonary veins. Such a map is shown in Figure 1 with typical recordings from various portions of the left atrium, including recordings of double deflections in the area of the ligament of Marshall, in 16 dogs. In 4 dogs these double deflections either were not recorded along the ligament or the area of the ligament in which such deflections could be recorded was limited to its inferior portion. The roving or probe electrode also allowed the exploration of the roof of the atrium along the interatrial band or Bachmann’s bundle and the inferior portions of the left atrium along the coronary sinus to the area of the coronary sinus ostium (AV nodal area). To establish a control sequence of atrial activation for the various experimental conditions described below, bipolar plunge wire electrodes were inserted into the right and left atria at the following points indicated in Figure 2: immediately anterior to the SA node (site 1), at the coronary sinus ostium (site 3), and on the inferior portions of the right and left atria along the coronary sinus (sites 6, 7). In addition, recording electrodes were placed at various points in the posterior left atrium along the edge of the ligament of Marshall (sites 8, 9) to record double potentials. In some cases, bipolar recordings were made at the beginning of Bachmann’s bundle near the SA node (site 2) and on the left atrium at a point where Bachmann’s bundle enters the underside of the left atrial appendage (site 5). A bipolar catheter, inserted via a femoral vein (6) or common carotid artery (7), was positioned at the His bundle area (site 4) for recording atrial, His bundle, and ventricular activity.

EXPERIMENTAL PROCEDURES

Effect of Atrial Pacing on Interatrial Conduction.—Electrical stimulation was applied through the bipolar electrode in the SA nodal area at rates up to 400–600/min. Atrial pacing was also performed through the roving electrode along the ligament of Marshall and just lateral to this structure in the posterior left atrium.

Selective Impairment of Atrial Conduction.—Measurements of interatrial conduction were made before and after lesions were produced in Bachmann’s bundle by crushing the interatrial band behind the aorta with a clamp. In other experiments local cooling of the posterior-inferior portion of the left atrium was performed with the use of specially constructed metal probes.

1Designed by S. M. Ross, Department of Pharmacology, College of Physicians and Surgeons, Columbia University, New York, N. Y.

2Designed by E. Fabré, Small Parts, Inc., Miami, Florida.

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Alcohol, cooled to $-10^\circ$ to $-20^\circ$C, was continuously passed through the metal probe whose tip diameter measured 3-4 mm. Application of the tip of the metal probe was made at the point where the ligament of Marshall merged into the left atrium (the posterior-inferior left atrium) to affect atrial conduction in the left atrial tract. Bachmann's bundle was intact during this procedure.

**Alteration of the Pacemaker Site.**—The effect of alteration of the site of pacemaker formation on the sequence of atrial activation was achieved by several means: (1) cooling or crushing the SA node to produce AV nodal rhythms, (2) pacing from several sites including the low atrium, His bundle, and ventricles, and (3) cardiac sympathetic nerve stimulation using silver wire electrodes attached to the undivided sympathetic nerve trunk that derives from the left stellate subclavian artery. Stimuli of 4-msec duration, a ganglion and passes anteriorly over the left frequency of 25 Hz, and an intensity of 4–10 v were applied to the nerve trunks.

**In Vitro Studies.**—In two cases, the heart was excised from two young mongrel dogs for recordings were made from the left atrial tract. In oxygenated Tyrode's solution with a millimolar content of NaCl 130, KCl 2.7, CaCl₂ 2.02, MgCl₂ 0.5, NaHCO₃ 24, Na₂HPO₄ 3.6, and dextrose 5.5, the inferior right and left atra were dissected starting at the coronary sinus ostium. The dissected portion included the epicardial surface of the coronary sinus in the inferior right and left atra, the posterior left atrium (containing the ligament of Marshall), and the adjacent left atrial myocardium. The tissue was mounted, with the epicardial surface exposed for exploration, in a Lucite chamber and perfused with Tyrode's solution bubbled with 95% O₂-5% CO₂ at 34°C. Two stimulating silver wires were applied on the epicardial surface of the coronary sinus near the location of the ostium or on the superior portion of the left atrial tract. Under the dissecting scope (40x magnification), the left atrial tract could be seen as a fine pink streak (less than 1 mm in diameter) running within the ligament of Marshall. Close bipolar wire electrodes held by micromanipulators were applied to the left atrial tract, the immediately adjacent atrial muscle (2 mm away), and the epicardial surface of the coronary sinus 2 cm distant (Fig. 11C).

left atrium (LA), (8) Inferior portion of the ligament of Marshall adjacent to the left inferior pulmonary vein (PV), and (9) Superior lip of the ligament adjacent to the left superior pulmonary vein. Other abbreviations not explained in A: Ao. = aorta and IVC = inferior vena cava (8).

**Conclusion.**—...
Histological Studies.—In several dogs with intact hearts (not subjected to cooling or crushing procedures), sections were excised, including the SA nodal, AV nodal, and His bundle regions, the inferior left atrium, and the posterior left atrium (ligament of Marshall). In some instances the ligament itself was dissected from the underlying tissue. Recordings from this area were made before and after excision of the ligament from the atrium. Postmortem sections of the tissue just beneath the ligament were also obtained. The tissues were fixed and buffered in formalin, subjected to routine histological preparation, and stained with hematoxylin and eosin, Gomori’s Trichrome, and the periodic Schiff’s reaction.

Results

Bipolar recordings along a deep fold in the

![FIGURE 3](image)

Recordings of the left atrial muscle (LAM) and left atrial tract (LAT) potentials during normal sinus rhythm. Traces from top: sinus node area (SA); coronary sinus ostium (CS); inferior junction of the right and left atria on the coronary sinus (AVG); posterior left atrial potentials (PLA), one inferior (LAM1, LAT1) and one superior (LAM2, LAT2), on the ligament of Marshall; His bundle electrogram (Hb) showing atrial (A), His bundle (H), and ventricular (V) activity; and a lead II ECG (L-2). Note that the left atrial tract potentials (arrows) occur during the P-R segment and that the tract potentials from the superior portion of the ligament occur later in time than the His bundle deflection. The interval between time lines equals 200 msec.

![FIGURE 4](image)

The effect of atrial pacing on the relationship of atrial activity and the left atrial potentials. Traces from top: stimulus delivered to the sinus node region (SA); inferior right and left atrial potentials on the coronary sinus (AVG); two recordings from the inferior (LAM1, LAT1) and superior (LAM2, LAT2) portions of the ligament of Marshall in the posterior left atrium (PLA); His bundle electrogram (Hb); and a lead II ECG (L-2). Atrial pacing at 257/min produced Wenckebach cycles. Note that left atrial tract (LAT) potentials, as well as other atrial (A) activity, separate from the His (H) and ventricular (V) activity and are completely dissociated during the blocked beat. The interval between time lines equals 200 msec. LAM = left atrial muscle, PI = pacer impulse.
Furthermore, recordings made along the posterior-inferior portion of the left atrium (coronary sinus) also showed compound electrograms which could be dissociated into distinct and separate activations under certain imposed conditions (see below).

Another unusual feature of these double deflections was the timing of the second deflection of the pair in relation to the P wave recorded on the surface electrocardiogram. Figure 3 shows bipolar electrograms recorded from the area of the SA node, the coronary sinus ostium, and the posterior junction of the right and left atria above the AV groove and two electrograms from the ligament of Marshall. Each of these close bipolar electrograms consisted of a left atrial muscle potential and a left atrial tract potential. These designations were based on histological evidence (see below). A His bundle electrogram showing atrial, His bundle, and ventricular activity and a lead II ECG were also recorded. Note that the left atrial tract potentials from both the inferior and the superior portions of the ligament occurred during the P-R segment.

During atrial pacing at 257/min (Fig. 4) a Wenckebach cycle was seen. The left atrial tract potentials, along with other atrial activity, separated from the His bundle and ventricular activity. In the blocked beat, atrial activity and ventricular activity were completely dissociated. Note that the left atrial muscle and left atrial tract potentials showed a constant relationship throughout.

This relationship was not inviolate and could easily be altered during His bundle or ventricular pacing with retrograde conduction or during AV nodal or low atrial rhythms. In Figure 5B, during ventricular pacing at the His bundle recording site, the left atrial muscle and tract potentials, from the inferior portion of the ligament have merged, whereas those from the superior portion are closer together as compared to sinus rhythm (Fig. 5A). Another means of altering the temporal relationship between the left atrial muscle and tract potentials was rapid atrial pacing. In Figure 6, during sinus rhythm, bipolar electrograms were recorded from Bachmann's bundle.
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A Control Sinus Rhythm (133/min)

B Atrial Pacing (420/min)

FIGURE 6
Disassociation of left atrial tract (LAT) potentials from atrial activity during atrial pacing at rapid rates. Traces from top: lead II ECG (L-2); electrogram from Bachmann's bundle close to the sinus node (Bb-SA); two electrograms from the posterior left atrium (PLA) on the ligament of Marshall, with double potentials from the inferior ligament (LAM_1, LAT_1) and from a more superior portion (LAM_2, LAT_2); and a His bundle electrogram (Hb) showing atrial (A), His bundle (H), and ventricular (V) activity. A: Sinus rhythm at a rate of 133/min. B: Atrial pacing (PI = pace impulse) from Bachmann's bundle area near the SA node at 420/min produced 2:1 AV block and 2:1 block in the left atrial tract. Note the appearance of the left atrial tract potentials from both inferior and superior portions of the ligament in alternate cardiac cycles. The interval between time lines equals 1 second.

near the SA node and from two sites on the ligament of Marshall in the posterior left atrium, each showing a left atrial muscle and a left atrial tract potential. A His bundle electrogram and a lead II ECG were also recorded. At an atrial pacing rate of 420/min, there was a 1:1 relationship between the pacing stimulus and the left atrial muscle potentials, but a 2:1 block occurred in the left atrial tract.
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A Smui Rhythm (180/min)

SA-Bb 26 \( \text{msec} \)
SA-AVG 40 \( \text{msec} \)
SA-LAM 35 \( \text{msec} \)
SA-LAT 66 \( \text{msec} \)

B Crush Bachmann's Bundle
SA-Bb 56 \( \text{msec} \)
SA-AVG 40 \( \text{msec} \)
SA-LAM 31 \( \text{msec} \)
SA-LAT 66 \( \text{msec} \)

FIGURE 7
The effect of crushing Bachmann's bundle on the sequence of atrial activation. Traces from top: lead II ECG (L-2); sinus node area electrogram (SA); electrogram from underside of left atrial appendage (LAP) where Bachmann's bundle (Bb) activates the left atrium; inferior left atrial electrogram above the AV groove (AVG); and a posterior left atrial (PLA) electrogram from the ligament of Marshall showing left atrial muscle (LAM) and left atrial tract (LAT) potentials. A: During sinus rhythm (180/min) the conduction time from the SA node electrogram to Bachmann's bundle (SA-Bb) = 26 msec, to the inferior left atrium above the AV groove (SA-AVG) = 40 msec, to the left atrial muscle (SA-LAM) = 25 msec, and to the left atrial tract (SA-LAT) = 66 msec. B: After crushing Bachmann's bundle with a clamp, the conduction times from the SA node to Bachmann's bundle and to left atrial muscle were prolonged to 56 msec and 31 msec, respectively. Conduction times from the SA node to the inferior left atrium above the AV groove and to the left atrial tract and heart rate were unchanged. Note the compound (slow and fast components) in the Bb, AVG, and LAM electrograms after crushing. The interval between time lines equals 1 second.

SELECTIVE IMPAIRMENT OF ATRIAL CONDUCTION

In Figure 7 the sequence of atrial activation, as indicated in recordings from selected atrial sites, was obtained using electrograms from the SA node area, the underside of the left atrial appendage where Bachmann's bundle enters the left atrium, the inferior left atrium above the AV groove, and the middle of the ligament of Marshall. After crushing the superior interatrial band (Bachmann's bundle), the conduction time from the SA node area to the inferior left atrium and the left atrial tract was the same, 40 msec and 66 msec, respectively. However, the conduction time from the SA node to Bachmann's bundle in the left atrium was prolonged from 26 msec to 56 msec, and from the SA node to the left atrial muscle it was prolonged from 25 msec to 31 msec. Note also the fractionation of the left atrial muscle potential and the prolongation of the P wave in the lead II ECG.

In another series of experiments, in which Bachmann's bundle was intact, a cold metal probe (-10° to -15°C) was applied to the posterior-inferior left atrium just below the beginning of the ligament of Marshall (Fig. 2B, site 8). In Figure 8 three standard ECG leads as well as bipolar electrograms from the area of Bachmann's bundle near the SA node and from the posterior left atrium showing left atrial muscle and tract potentials were recorded throughout. With the application of the cooling probe (tip diameter 3-4 mm), the conduction time from the SA node to the left atrial tract progressively increased from 105 msec to 154 msec with subsequent complete block of the left atrial tract potentials (B, C). With rewarming (C, arrow) the left atrial tract potential gradually resumed its original relationship to the left atrial muscle and SA node electrograms. Throughout the intervention the conduction time from the SA node to the left atrial muscle remained unchanged as did the heart rate and P-wave morphology.

INFERIOR-POSTERIOR LEFT ATRIAL SITE OF IMPULSE FORMATION

In Figure 9 the effect of pacing from the posterior left atrium, specifically, the edge of the ligament of Marshall, was compared with pacing of the adjacent atrial myocardium. In A bipolar electrograms from the area of the SA node and the posterior left atrium showing a left atrial muscle potential and a left atrial tract potential were simultaneously recorded with three standard ECG leads I, II, and AVR. The time from the onset of the SA node electrograms to the left atrial muscle potential was 15 msec, whereas the conduction time
The effect of localized cooling of the inferior portion of the ligament of Marshall on conduction in the left atrial tract. Traces from top: ECG leads I (L-1), II (L-2), and aVr; bipolar electrograms from Bachmann's bundle (Bb) near the SA node and a posterior left atrial recording (PLA) from the ligament of Marshall showing left atrial muscle (LAM) and left atrial tract (LAT) potentials. A: During sinus rhythm the conduction time from Bachmann's bundle to left atrial muscle (Bb-LAM) = 22 msec and to left atrial tract (Bb-LAT) = 105 msec. Application of a small cooling probe (−10°C to −15°C) where the ligament joins the posterior left atrium (arrow) produces a prolongation of the Bb-LAT interval (115 msec). B: Further prolongation of the Bb-LAT interval occurs until the left atrial tract potential is completely blocked. C: With rewarming (arrow) the original relationship of the left atrial tract deflection to Bachmann's bundle and left atrial muscle deflections returns. Note that the heart rate, P-wave morphology, and the Bb-LAM interval are unchanged throughout. The interval between time lines equals 1 second.

from the SA node to the left atrial tract was 80 msec. In B, pacing from the atrial myocardium adjacent to the ligament of Marshall (within 3 mm of the recording site as shown in A) showed a relatively unchanged P wave in leads I, II, and AVR. Note that time required for the pacing impulse to reach the node was 13–15 msec, approximately the same as the antegrade conduction time, between the SA node and the left atrial muscle. In C
The effect of pacing from the left atrial muscle and left atrial tract sites on P-wave morphology. Traces are the same as in figure 8, except a sinus node area electrogram (SA) was recorded rather than one from Bachmann's bundle. A: During sinus rhythm (112/min) conduction time from the SA node to left atrial muscle (SA-LAM) = 15 msec and to left atrial tract (SA-LAT) = 80 msec. B: Atrial pacing at 120/min, 3 mm from the edge of the ligament of Marshall, produces a P wave essentially unchanged from that seen in A and the retrograde time from the pacer impulse (PI) to SA node electrogram is 13–15 msec, equal to the antegrade conduction time from the SA node to left atrial muscle seen in A. C: Atrial pacing at 120/min, from the edge of the ligament, produces an inversion of P waves in lead II and aVR and the time between the pacer impulse and the SA node activity is 80–85 msec, equal to the antegrade conduction time from SA node to left atrial tract seen in A. Also note the latency of the stimulus to the onset of the P wave in all leads. The interval between time lines equals 1 second.

Another related observation is shown in Figure 10. During sinus rhythm at a rate of 160/min, electrograms were recorded from the SA node area, coronary sinus, inferior left atrium, and posterior left atrium (ligament of

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the stimulus was applied to the edge of the ligament of Marshall, which resulted in an inverted and broadened P wave as seen in leads II and AVR. Note also that the conduction time from the pacing impulse to the SA node was 80–85 msec, approximately the same as the antegrade conduction time from the SA node to the left atrial tract shown in A.
A Control - Normal Sinus Rhythm (160/min)

B Left Sympathetic Stimulation (20 Hz) - (165/min)

**FIGURE 10**
The effect of left cardiac sympathetic nerve stimulation on the site of impulse formation. Traces from top: bipolar electrograms from sinus node area (SA), coronary sinus ostium (CS), inferior left atrium above the AV groove (AVG), ligament of Marshall in the posterior left atrium (PLA), and a lead II ECG (L-2). A: During sinus rhythm (160/min) the sequence of atrial activation proceeds from the SA node area (earliest electrogram) to the left atrial tract (LAT) (latest deflection). B: During left cardiac sympathetic nerve stimulation the heart rate increases to 165/min with a shift in pacemaker, with the region of the inferior left atrium activated earliest. Note that the AVG electrogram (arrow) shows a fractionated deflection and the left atrial tract potential (arrow) precedes the left atrial muscle (LAM) potential. Also the P wave is inverted in lead II, but the P-R interval is essentially unchanged from that seen in A (90-100 msec).

Marshall) and a lead II ECG was also recorded (A). In this dog, cardiac sympathetic nerve stimulation produced an atrial activation pattern consistent with pacemaker activity arising in the inferior left atrium. An inverted P wave in lead II and a P-R interval approximately equal to that observed during sinus rhythm (90-100 msec, B) were recorded.
It should be noted that the heart rate was faster during the left atrial rhythm (165/min) than it was during sinus rhythm. Note that in B the left atrial tract potential precedes the left atrial muscle potential, and the electrogram from the inferior left atrium has dissociated into two distinct deflections. During normal sinus rhythm, the sequence of atrial activation indicated a progression from the SA node electrogram in the right atrium to the left atrial tract electrogram in the posterior left atrium. During left cardiac sympathetic nerve stimulation, the earliest activity was recorded in the inferior left atrium above the AV groove, near the posterior-inferior junction of the right and left atria, and proceeded retrograde to the coronary sinus ostium and the SA node area. In addition, during normal sinus rhythm the potentials in the inferior left atrium above the AV groove and in the left atrial muscle were activated almost simultaneously. However, the conduction time from the inferior left atrium above the AV groove to the left atrial tract was 47 msec. During left cardiac sympathetic nerve stimulation, the left atrial tract potential directly followed the onset of the potential in the inferior left atrium above the AV groove by 11 msec and the left atrial muscle potential was activated 21 msec later.

**IN VITRO STUDIES**

In two experiments, conduction was studied in vitro. A section of atrial tissue was dissected, including the epicardial surface of the coronary sinus and portions of the posterior left atrium surrounding and encompassing the ligament of Marshall (Fig. 11C). In Figure 11A stimulation at sites on the epicardial surface of the coronary sinus (equivalent position in vivo was the inferior right atrium) produced an activation sequence of bipolar electrograms at sites 1, 2,
and 3 as shown. The distance between site 1 and sites 2 and 3 was 20 mm, whereas the latter two sites were 2 mm apart. Site 2 was positioned on the left atrial tract, and site 3 was situated on adjacent atrial musculature, off the ligament. The results indicated that the impulse was conducted more rapidly from site 1 to site 2 (0.5 mm/sec) than from site 1 to site 3 (0.25 mm/sec). Stimulation above site 2 on the ligament, Figure 11B, altered the

**FIGURE 12**

Histological sections through the ligament of Marshall. Hematoxylin and eosin stain. A: Section through the inferior-posterior left atrium where the tract joins the left atrium and coronary sinus. The tract muscle (LAT) lies in this fibrofatty tissue just above the underlying left atrial musculature (LAM) and the vein of Marshall (V). The apposition of the transversely cut tract to the longitudinal cardiac muscle of the coronary sinus (CS) is apparent. N = nerve. B: Section through the ligament adjacent to the left superior pulmonary vein. Note that the atrial tract is reduced in diameter but still lies adjacent to the vein of Marshall. Many nerves (N) are seen within the ligament.
sequence of activation but not the timing between sites 2 and 1. However, site 3 was activated more than 135 msec after site 2, which was only 2 mm away.

**HISTOLOGICAL STUDIES**

Figure 12 shows a cross section through the inferior portion of the ligament of Marshall (site 8, Fig. 2B) showing that, within the fibrous tissues of this structure, bundles of atrial muscle are associated with a small vein. This tract, unlike other atrial tracts is distinctly segregated and insulated from the underlying left atrial myocardium and connects to the left atrial myocardium only at the level of the inferior pulmonary vein. Figure 12B shows a cross section through a more superior portion of the ligament of Marshall in which only the cut end of the left atrial tract is seen separated from the vein and surrounded by many nerves. All of these structures are embedded in the fibrofatty tissues of the ligament, and the isolation of the muscular tract from the underlying atrial myocardium is more evident.

In several experiments double recordings were made from the ligament of Marshall prior to (Fig. 13A) careful excision of the ligament from the underlying atrial tissue. After excision (Fig. 13B) only one potential, corresponding to the left atrial muscle deflection, was recorded from the same site. In a few instances crushing of a small area (2-3 mm²) at the point at which the ligament connects to the posterior-inferior left atrium produced the same result. The excised tissue was processed for histological study (see Methods). Examination of serial cross sections revealed a thin strand of cardiac muscle (Fig. 14) running within the fibrofatty tissue of the ligament associated with the vein of Marshall. No underlying atrial tissue was seen except where the ligament joined the atrial musculature at the level of the inferior pulmonary vein. It should be emphasized that no section of this area, which encompassed the ligament of Marshall, or of the coronary sinus contained pulmonary venous tissue.

**Discussion**

Marshall in 1850 (2), while studying the comparative embryology of various mammalian hearts, observed that the left anterior cardinal vein rapidly shrinks but does not entirely disappear. In adult hearts, he observed the persistence of this vein, which enters the coronary sinus, in close relationship to the “vestigial fold of the pericardium” (ligament of Marshall). Other investigators have indicated that the ligament of Marshall is a vestigial structure which incorporates portions of the left sinus horn and left common cardinal vein (8). Although Marshall indicated the relationship of the oblique vein (vein of Marshall) to the coronary sinus, he only alluded to the fact that the former is “embedded in the wall of the left auricle.” As for the ligament, no indication of its relationship to the atria was made other than its composition, i.e., fibrous connective tissue (2).
Subsequent electrophysiological studies and anatomic investigations indicated specific connections between the AV node and the left atrium (9, 10). However, these pathways were described as short, diffuse tracts running through the interatrial septum between the AV node and the left atrium. More definitive atrial tracts were delineated by the early electrophysiological studies of Bachmann (11), the more recent histological studies of James (1), and the electrophysiological studies of Wagner et al. (12). James has proposed that the previously described intra-atrial bundles of Bachmann, Wenckebach, and Thorel represent specialized atrial pathways connecting the sinus and the AV nodes (1). Two recent studies of intra-atrial conduction have indicated that large but distinct internodal areas of the atrium rather than localized tracts carry impulses from the sinus to the AV nodes (13, 14). On the basis of extensive endocardial mapping, Spach et al. (14) defined a special tract or pathway as one that is "effectively insulated electrically from the surrounding regular muscle."

The present study details several lines of evidence indicating the existence of functional atrial muscle within the ligament of Marshall,
which, in several respects, is unlike previously described atrial tracts. The histological sections in Figures 12 and 14 suggest that the atrial muscle within the ligament is insulated from the underlying atrial myocardium. The insulation of the left atrial tract was also indicated electrophysiologically on the basis of several observations. (1) The recorded left atrial tract potentials, at almost all positions along the ligament, were temporally coincident with the P-R segment rather than the P wave. In some cases the left atrial tract potentials appeared later in time than did the His bundle deflection (Figs. 3, 4). It should be pointed out that in the superior portion of the ligament the tract appeared to terminate without reinsertion into atrial muscle. This was indicated in Figure 8C in which localized cooling of the junction of the left atrium and the ligament produced block of the left atrial tract potential. With rewarming, the left atrial tract potential occurred 228 msec from the onset of activity in Bachmann's bundle near the sinus node. The reexcitation of the atrium would be expected if this potential reentered any portion of the right or left atrium. (2) The left atrial musculature directly adjacent to or underlying the ligament of Marshall was activated at approximately the midpoint of the P wave and activation proceeded from the superior to the inferior left atrium (Figs. 3, 4). On the other hand, the tract potentials were activated after the end of the P wave in the opposite direction. The pattern of activation in the area of the left atrium is not consistent with activation of a functional syncytium but is consonant to effectively insulated contiguous atrial tissues.

The hypothesis was entertained that the left atrial muscle potential and the left atrial tract potential were being normally activated through separate pathways: the left atrial muscle from the SA node via Bachmann's bundle along the interatrial roof and the left atrial tract from the SA node through the low right atrium and then along an inferior right-to-left interatrial pathway. In the critical evaluation of this postulation, two questions require comment. (1) Is the left atrial tract part of the atrium or is it extracardiac in nature? (2) Is it proper to refer to the proximal connections to the left atrial tract as an inferior interatrial pathway? In regard to the first question, the embryology of this region indicates that the coronary sinus, the ligament, and the vein of Marshall derive from closely associated anlage (left sinus horn and the left anterior cardinal veins) (2, 8). Histologically, serial sections indicated a continuous bundle of striated cardiac muscle (Figs. 12, 14) within the ligament of Marshall. In addition, at its inferior limit, the tract was continuous with atrial muscle of the posterior-inferior left atrium. Electrophysiologically, the tract was activated under most conditions with each cardiac cycle and maintained a fixed relationship with other atrial activation under most physiological conditions (Figs. 3-5). Recently, Ito et al. (15) showed that sinocaval tissues (junctional tissues of the pulmonary vein-atrial margin) demonstrated properties of slow conduction and action potentials which were characterized by low amplitude, slow or slurred upstroke, delay or block at high rates, and slight diastolic depolarization. Although the left atrial tract runs along the left pulmonary veins, it is composed of striated cardiac muscle (Fig. 14) and the underlying tissue is also composed of cardiac muscle (Fig. 12). Conduction velocity both in situ and in vitro is faster, 0.5-1.0 mm/msec, compared to ordinary atrial myocardium, 0.25 mm/msec. Action potentials from the left atrial tract indicate a rapid rate of rise, 300-450 v/sec, a well-developed plateau, and no evidence of diastolic depolarization (16). This evidence suggests that the left atrial tract is cardiac in nature, although it has somewhat different properties than ordinary cardiac muscle or sinocaval tissue.

In regard to the question of the validity of an inferior interatrial conduction pathway, this concept is not without precedent. Ito et al. (15) mapped atrial activation in the rabbit heart. In this species the left superior vena cava, which is the homologue of the ligament of Marshall, persists and was shown to be the latest area to be activated in the rabbit atrium.

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Activation occurred from the low right atrium across the inferior portion of the right and left atria into the left vena cava from the inferior to the superior aspect (15). The last portion of the left superior vena cava to be activated was affected 89 msec after SA node activation. In this same paper Ito et al. (15) give reference to a report that indicates that the rabbit and other mammals have striated muscle of the cardiac type in the superior vena cava but not in the inferior vena cava (17). Oishi, in 1967, described atrial activation in the isolated perfused canine heart (18). Activation was shown to proceed in two directions from the right to the left atrium: (1) from the SA node through Bachmann's bundle to the superior and the inferior left atrium and (2) from the superior to the inferior aspect of the right atrium along the crista terminalis and then laterally across the inferior right and left atria. The region located between the left inferior pulmonary vein and the AV ring was the last area activated. In our studies, bipolar electrode mapping of the atria (Figs. 1, 3, 10) and experimental interventions (Figs. 7, 8) confirmed and extended the findings of the Japanese investigators. During sinus rhythm mapping of the activation pattern of the inferior right and left atria along the coronary sinus and the posterior left atrium revealed a continuous sequence of atrial activation which proceeded from the middle portion of the P wave to the end of the P wave and into the P-R segment (Figs. 3, 4, 10). On the other hand, the superior-anterior portions of the atria showed no atrial activation occurring after the midpoint of the P wave. In addition, the compound nature of the potentials recorded from the coronary sinus and the inferior left atrium just above the AV groove were frequently observed, as well as the fractionation of these compound potentials into slow and fast components during several physiological interventions (Figs. 3-5, 10). Notably the compound electrogram showed alternation of slow and fast potentials at high driving rates (B. J. Scherlag, unpublished observation). Such alternation of configurations in a compound electrogram at high driving rates presented evidence for the existence of both slow- and rapid-conducting fibers in Bachmann's bundle (12). The findings in the present study suggest that at least the epicardial surface of the coronary sinus, which consists of atrial muscle, represents the proximal connection between the left atrial tract in the left atrium to the area of the coronary sinus ostium in the posterior-inferior right atrium. That there may be other connections between the area of the AV node and the left atrium has not been assessed in this report. Such tracts have been noted histologically (9, 10) but have not been studied electrophysiologically.

From an electrocardiographic point of view, the existence of an inferior interatrial pathway between the low right atrium (area of the AV node) and the posterior left atrium may aid in explaining the similarity of P waves in specific leads when activation arises in these areas. Two schools of thought have developed. One explains the P-wave morphology based on uniform activation from the site of stimulation or impulse formation (19-22). The other states that the P-wave configuration is in large measure due to the proximity of the pacing site or sites of impulse formation to various specialized atrial conduction pathways (23-25). In the present study atrial pacing at localized sites 3 mm apart in the posterior left atrium through close bipolar pacing electrodes indicated a significant difference in P-wave morphology. Pacing stimuli delivered 3-4 mm from the edge of the ligament of Marshall produced little change in the P-wave morphology as compared to that seen during normal sinus rhythm in leads I, II, and AVR (Fig. 9). Atrial pacing applied to the posterior left atrium at the edge of the ligament of Marshall at the same pacing rate produced a marked change in the P-wave morphology, i.e., inverted and broadened P waves in leads II and AVR. The same inversion of the P wave in leads II and AVR could be produced with pacing from the distal portion of the ligament adjacent to the left superior pulmonary vein or from the proximal portion of the ligament adjacent to the left inferior pulmonary vein.
The only difference observed was the duration of the P-R interval, which increased as the pacing site was moved from the proximal to the more distal portion of the left atrial tract (B. J. Scherlag, unpublished observations). Another experimental intervention, left cardiac sympathetic nerve stimulation (Fig. 10), produced an inverted P wave in lead II; however, there was little or no change in the P-R interval. The sequence of atrial activation was markedly different from that during sinus rhythm, with a probable site of impulse formation in the posterior-inferior left atrium. Moreover, there was a marked reversal of the relationship between the left atrial tract potential and the left atrial muscle potential in the posterior left atrial electrogram. The inverted P wave in lead II would seem to indicate an AV nodal rhythm; however, it should be noted that during nodal rhythm or retrograde atrial activation due to ventricular pacing (Fig. 5) the left atrial muscle and tract potentials tend to converge. The left atrial tract potential was never seen to precede the left atrial muscle potential under these circumstances. It would be difficult to justify an AV nodal rhythm with first degree heart block during sympathetic nerve stimulation.

Although the physiological role of this inferior interatrial tract remains to be definitively determined, the findings of the present study suggest the possibility that rapid ectopic foci in the posterior-inferior left atrium may masquerade as junctional tachycardias.

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References

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