The P Wave and P-R Interval

Effects of the Site of Origin of Atrial Depolarization

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SUMMARY

The atria of 37 patients were paced from selected sites during cardiac surgery. When the atria were paced from endocardial sites low in the right atrium, the P waves in ECG leads II, III, and aV_F were shown to be either negative, biphasic, or positive, depending on the site paced. When the endocardial sites were paced, the P-R intervals were, almost without exception, less than 0.12 sec. When those endocardial sites closest to the A-V junction were paced, the P-R intervals were always less than 0.12 sec. When the atria were paced, from the epicardial sites, the P-R intervals were always greater than 0.12 sec. Negative P waves in ECG leads II, III, and aV_F were recorded when the atria were paced from the postero-inferior left atrium and the caudal right atrium. The P-R interval did not always reflect the initial period of atrial activation because an isoelectric interval, generally of 0.01 to 0.025 sec, was frequently present between the onset of atrial stimulation and the first clear evidence of the P wave in the ECG. The implications of these results are discussed.

Additional Indexing Words:

Low atrial rhythm A-V junctional rhythm Specialized atrial conduction
Atrial pacing

THE ELECTROCARDIOGRAPHIC diagnosis of several atrial arrhythmias depends strongly on the polarity and morphology of the P wave and duration of the P-R or R-P interval. Such arrhythmias include A-V junctional or A-V nodal rhythms, coronary sinus rhythms, coronary nodal rhythms, and left atrial rhythms.[1-8] A knowledge of relationships between the site of origin of atrial depolarization and the resulting sequence of atrial activation is central to the understanding of these arrhythmias and the P waves associated with them. Scherf and Cohen have thoroughly reviewed this aspect of electrocardiographic interpretation.[1, 9]

Lewis[10] and others,[11-14] as a result of studies on the canine heart, accepted the premise that excitation of the atria spreads radially and at uniform velocity from its site of origin. Most interpretations of clinical electrocardiograms make the same assumption. In terms of this hypothesis, activation of the atria by an impulse originating at a site near the A-V node should result in negative P waves in leads II, III, and aV_F. However, reports from this laboratory[15, 16] and those of others[17-26] have provided evidence that this need not be true. Recently, Spach and associates[26] demonstrated that when the atria of the canine heart were paced from a site near the coronary sinus, retrograde activation of the atria was not radial. In studies on the canine heart,

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This work was supported in part by Research Grant HE 12738 from the National Heart and Lung Institute, U. S. Public Health Service. Work was carried out during Dr. Waldo's tenure of a John Polachek Medical Research Fellowship and Dr. Vitikainen's surgical traineeship from the National Institute of General Medical Sciences, National Institutes of Health.

Received March 30, 1970; revision accepted for publication June 22, 1970.

Table 1

Clinical and Therapeutic Information for 37 Patients Studied

Patient	Lesion*	Age (yr); sex	Preanesthetic medication†	Anesthesia		Other drugs†	Temperature (°C)	Preop P-R interval during spontaneous rhythm (sec)
				Induction†	Maintenance†			
1	ASD	15 M	S 100 mg A 0.3 mg M 25 mg	H SDC 10 mg	H	None	37	0.13
2	ASD, PS	8 F	S 60 mg MS 4 mg SC 0.3 mg	H N SDC 40 mg	H N	None	37	0.16
3	ASD	18 M	S 100 mg MS 4 mg SC 0.5 mg	P 100 mg SDC 60 mg	H N	None	37	0.15
4	Exploratory thoracotomy	11 M	S 75 mg SC 0.3 mg MS 8 mg	C SDC 80 mg	H N	None	37	0.17
5	VSD, inf. sten	9 M	MS 5 mg Pr 50 mg A 0.3 mg	H N SDC 60 mg	H N	None	34	0.17
6	PS	8 M	S 40 mg MS 2 mg SC 0.2 mg	H N A 0.1 mg E 5 mg	H N	None	37	0.13
7	VSD, PS	11 M	S 70 mg MS 4 mg SC 0.4 mg	H	H	None	34	0.14
8	Double outlet RV	9 M	S 40 mg MS 3 mg SC 0.2 mg	C H N	H N	None	34	0.15
9	ASD	53 F	CL 100 mg SC 0.4 mg	P 150 mg SDC 100 mg	H N E 5 mg	Q 100 mg TID	34	0.21
10	MS	35 F	None	P dose? SDC dose?	H, N M-dose? dTC 12 mg E 7 mg	None	34	0.16
11	ASD	12 F	S 65 mg MS 7 mg Ser 0.3 mg	P 125 mg SDC 100 mg	H N	None	37	0.16

12	MS, PDA	15 M	S 100 mg SC 0.4 mg	P 200 mg SDC 60 mg H	H N	None	35	0.14
13	ASD	17 F	S 100 mg A 0.5 mg	P 250 mg SDC 100 mg H	H	None	37	0.18
14	VSD, inf. sten.	10 F	SC 0.3 mg S 75 mg	H N	H N	None	34	0.16
15	ASD	12 F	S 80 mg MS 5 mg SC 0.5 mg	H N	H N	None	37	0.16
16	ASD	22 M	S 100 mg A 0.5 mg	P 455 mg SDC 140 mg	H N	None	37	0.19
17	VSD	16 M	S 100 mg MS 6 mg SC 0.5 mg	P 350 mg SDC 100 mg	N H	None	34	0.15
18	VSD	12 F	S 75 mg MS 4 mg A 0.4 mg	P 250 mg SDC 80 mg	H N	None	34	0.16
19	VSD	10½ M	S 75 mg MS 4 mg SC 0.4 mg	N C	H	None	34	0.17
20	VSD	6 F	S 45 mg MS 3 mg SC 0.25 mg	C	H	None	34	0.17
21	PS	5 M	S 75 mg SC 0.2 mg M 25 mg	H N	H N	None	37	0.17
22	ASD	12 F	S 100 mg MS 0.5 mg SC 0.5 mg	C SDC dose?	N H	None	35	0.16
23	VSD, PS	8½ F	S 50 mg MS 3 mg SC 0.3 mg	C SDC 20 mg	H N	None	36-37	0.13
24	PS	9 F	S 80 mg MS 4 mg SC 0.4 mg	C SDC 40 mg	H N	None	37	0.16
25	ASD	17 F	S 75 mg	P 200 mg SDC 60 mg	H N	None	37	0.16
26	AS	20 F	S 100 mg MS 5 mg SC 0.4 mg	P 175 mg SDC 50 mg	H N	None	36-37	0.16
27	ASD	15½ M	S 100 mg MS 5 mg	P 200 mg SDC 60 mg	H	None	37	0.18

Patient	Lesion*	Age (yr); sex	Preanesthetic medication†	Anesthesia		Other drugs†	Temperature (°C)	Preop P-R interval during spontaneous rhythm (sec)
				Induction†	Maintenance†			
28	PS	9 M	S 60 mg MS 3.5 mg	C H	H	None	37	0.16
29	VSD	8 F	S 60 mg MS 4 mg	C H	H	None	36-37	0.16
30	ASD	9 F	S 75 mg MS 5 mg	C H	H	None	36	0.18
31	MS	29 M	D dose?	P 250 mg SDC 100 mg	H	Dig. × 3 days	35	0.16
32	MS	66 F	None	P 175 mg SDC 80 mg A 0.2 mg	H	Q 200 mg q.i.d. Dig. 25 mg q.d.	35	0.20
33	VSD	19 M	S 100 mg	SDC 140 mg P 700 mg	H	None	34	0.13
34	ASD	41 F	S 100 mg	P 250 mg SDC dose?	H E 5 mg	None	37	0.19
35	ASD	12 M	S 60 mg MS 8 mg	SDC 60 mg N H	H	None	37	0.15
36	PS	16 F	S 100 mg MS 5 mg	P 250 mg SDC 100 mg	H N	None	37	0.16
37	PS, VSD	8 F	S 100 mg MS 2 mg A 0.3 mg	C H SDC 25 mg	H	None	36-37	0.16

*Abbreviations for lesions: AS = aortic stenosis; ASD = atrial septal defect; Inf. sten = infundibular stenosis; MS = mitral stenosis; PDA = patent ductus arteriosus; PS = pulmonary stenosis; RV = right ventricle; VSD = ventricular septal defect.

†Abbreviations for drugs: A = atropine; C = cyclopropane; CL = chlordiazepoxide; D = diazepam; Dig = digoxin; dTC = d-tubocuarine; E = ephedrine; H = halothane; M = meperidine; MS = morphine sulfate; N = N_2O; P = pentothal (thiopental); Pr = promethazine; Q = quinidine; S = secobarbital (Seconal); SC = scopolamine; SDC = succinylcholine; Ser = serpolan.

Moore and associates[15] found that activation of the atria resulting either from retrograde conduction of an impulse through the A-V node or from stimulation of the lower interatrial septum resulted in P waves which were predominantly positive in leads II, III, and aV_F. More recently, we have shown[16] that, in man, retrograde activation of the atria by an impulse initiated in the specialized conduction system distal to the N region of the A-V node can result in P waves which are biphasic (−, +) or positive (+) in leads II, III, and aV_F.

Because our results were in disagreement with the conclusions resulting from most clinical and experimental studies[10–14, 27–30] and because there have been few detailed studies on relationships between site of origin of an impulse in the human atria and the polarity and morphology of the P wave, we have conducted a systematic investigation of the changes in polarity and morphology of the P wave which occur when the atria of the human heart are paced from selected epicardial and endocardial sites.

Methods

All patients were studied during cardiac surgery. The experimental protocol varied somewhat for each patient and was determined by the nature and requirements of the surgical procedure. A summary of pertinent clinical data on the 37 patients studied is provided in table 1. The ages of the patients studied ranged from 5 to 66 years, the mean age being 19 years. The atria of 23 patients had no congenital defects. Fourteen patients with atrial septal defects of the secundum type are included because we assumed that a defect limited to the region of the fossa ovalis would not interfere with the sequence of atrial activation.[31, 32] In the preoperative ECGs, cardiac rhythm, polarity, and morphology of the P waves and P-R intervals were within normal limits for each patient. All but 12 patients received small amounts of atropine or scopolamine (table 1) at least 2½ hours prior to the study. P-R intervals recorded in the operating room prior to thoracotomy were comparable to those recorded at a similar heart rate prior to surgery.

An acrylic plaque containing five silver electrodes[16, 33] was sutured to the right atrial epicardium of each patient to provide a clear record of atrial activity at all times during the surgical procedure and to permit atrial pacing to suppress arrhythmias or to provide a supraventricular rhythm.[16, 34] One pair of electrodes in the plaque was used only to record a bipolar atrial electrogram; another pair was used either to record a second electrogram or to stimulate. The fifth electrode contact served as a spare. A probe, containing three silver electrodes, was used either for bipolar stimulation or to record bipolar electrograms from selected sites on the atria or ventricles. Use of such an electrode to locate the bundle of His and other parts of the specialized conducting system has been described previously.[35, 36] All leads in contact with the heart were isolated from ground and the recording instruments by isolation transformers. During periods of data collection, body temperature, measured from the retrocardiac portion of the esophagus or from the rectum, varied from patient to patient as determined by the requirements of the surgical procedure; the range was 34 to 37 C.

Patients were studied while in the supine position. Pacing was performed from sites on the atrial epicardium prior to atriotomy for cannulation of the cavae and initiation of cardiopulmonary bypass. Endocardial sites were paced during the period of cardiopulmonary bypass. The atriotomy extended from the tip of the right atrial appendage toward the inferior vena cava to a point 1 to 2 cm onto the lateral free wall of the right atrium. This incision avoided the sino-atrial (SA) node and the atrial internodal tracts.[31, 32] Furthermore, the atriotomy itself did not modify the polarity or morphology of the P waves in the standard and augmented ECG leads when the atria were paced from the SA nodal region.

The five epicardial pacing sites are shown in figure 1. The PLA site is that portion of the postero-inferior left atrium which lies just above the coronary sinus, below the right inferior pulmonary vein, and just posteromedial to the inferior vena cava. The CRA site is the portion of the caudal right atrium just anterolateral to the junction of the inferior vena cava with the right atrium. In some patients, the epicardial aspect of the coronary sinus just as it enters the right atrium also served as a pacing site (CS epi). The CST site is the caudal portion of the sulcus terminalis, and the MST site is its mid-portion. The SA site is near the head of the SA node.

The endocardial pacing sites are shown in figure 2. During the period when the endocardial sites were paced, vacuum suction from a dependent portion of the right atrial endocardium near the coronary sinus ostium permitted ready visualization of the endocardial pacing sites. Site 1 is the roof of the coronary sinus ostium. Site 2 lies on the dorsal lip of the coronary sinus (the eustachean ridge), and site 3 is just within the

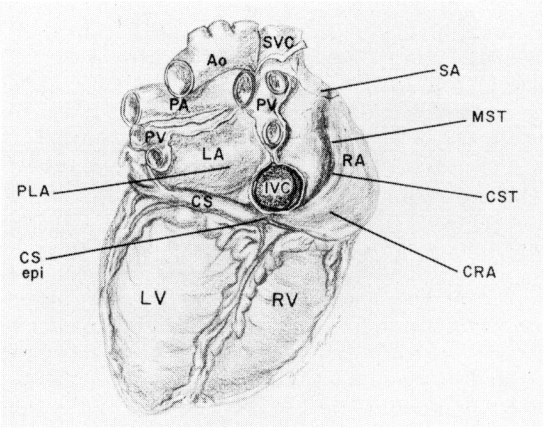

Figure 1

Postero-inferior aspect of the human heart showing the epicardial sites on the left and right atria from which the atria were paced. Abbreviations of sites: *PLA = postero-inferior left atrium: CRA = caudal right atrium; CST = caudal sulcus terminalis; MST = mid-sulcus terminalis; SA = sinoatrial node area; CS epi = epicardial aspect of the coronary sinus as it enters right atrial cavity.* Abbreviations of anatomy: *SVC = superior vena cava; Ao = aorta; PA = pulmonary artery; PV = pulmonary veins; LA = left atrium; RA = right atrium; CS = coronary sinus; IVC = inferior vena cava; LV = left ventricle; RV = right ventricle.*

coronary sinus ostium. Site 4 is on the ventral lip of the coronary sinus ostium, and site 5 is immediately medial to the ostium. Sites 6 and 7 are parallel to the atrioventricular groove and 4 to 5 mm medial to site 5; sites 8 and 9 are parallel to the atrioventricular groove and 4 to 5 mm medial to sites 6 and 7; and sites 10 and 11 are parallel to the atrioventricular groove and medial to sites 8 and 9. All epicardial and endocardial sites were not always paced in each patient.

The atria were paced at a rate slightly in excess of the spontaneous rate. Stimuli were provided by a Medtronic 1187 special digital threshold stimulator[37] and delivered through a stimulus isolator. Bipolar electrograms and simultaneous standard bipolar and augmented unipolar ECG leads were monitored on an eight channel Electronics for Medicine switched beam oscilloscope and recorded on photographic paper moving at speeds of 50 to 200 mm/sec. ECG recordings were calibrated at 2 cm/mv. ECG lead II was recorded for all patients. Only 18 patients, however, had all of the standard and augmented ECG leads recorded. Six patients had five leads recorded, 11 had three leads, two had two leads, and one patient had only one lead. Measurement of all intervals was made from the records with a vernier measuring device having an accuracy of ± 2 msec at the slowest recording speed used.

In many of these patients, bipolar wire electrodes were implanted in the atria at the end of the cardiac surgical procedure and brought out through the chest wall for use during the postoperative period as an aid in diagnosis and therapy.[38, 39] This enabled us to evaluate the effect of the open chest on P-wave polarity and morphology in 10 patients in whom the bipolar wire electrodes had been implanted at a site used to pace the atria during surgery. In these patients, on the first postoperative day, the atria were paced through the implanted electrodes and the P waves which resulted were compared to those recorded during surgery.

Results

Relationship Between the Site of Origin of the Impulse and P-Wave Polarity and Morphology

When the atria were paced from the epicardial and endocardial sites shown in figures 1 and 2, a consistent spectrum of changes in P-wave polarity and morphology resulted. P waves in leads II, III, and aV_F

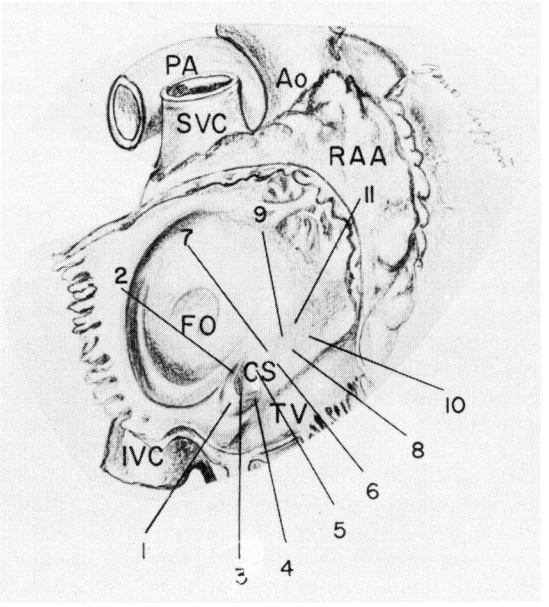

Figure 2

Right atrial endocardium of the human heart showing the endocardial sites (1 to 11) from which the atria were paced. Abbreviations: *FO = foramen ovale; CS = coronary sinus oriface; TV = posterior leaflet of the tricuspid valve; Ao = aorta; PA = pulmonary artery; RAA = right atrial appendage; SCV = superior vena cava; IVC = inferior vena cava.*

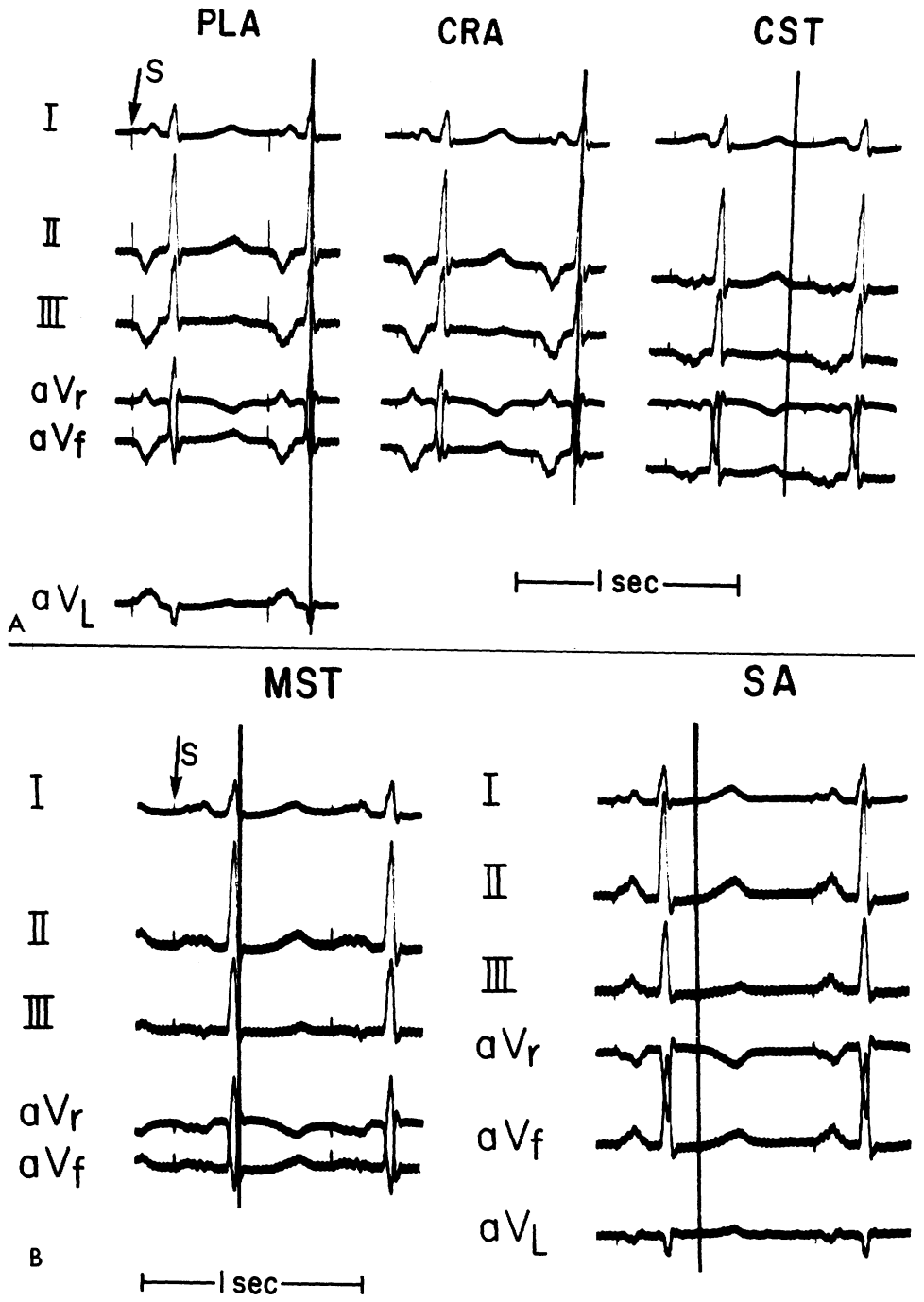

Figure 3

(A and B) P waves recorded from patient 31 when the atria were paced from the epicardial sites. A stimulus artifact (S) precedes each P wave. A time line appears in the record from each site. aVL was not recorded for this patient when sites CRA, CST and MST were paced. PLA = posterior left atrium; CRA = caudal right atrium; CST = caudal sulcus terminalis; MST = mid-sulcus terminalis; SA = sinoatrial nodal area. Paper speed is 50 mm/sec.

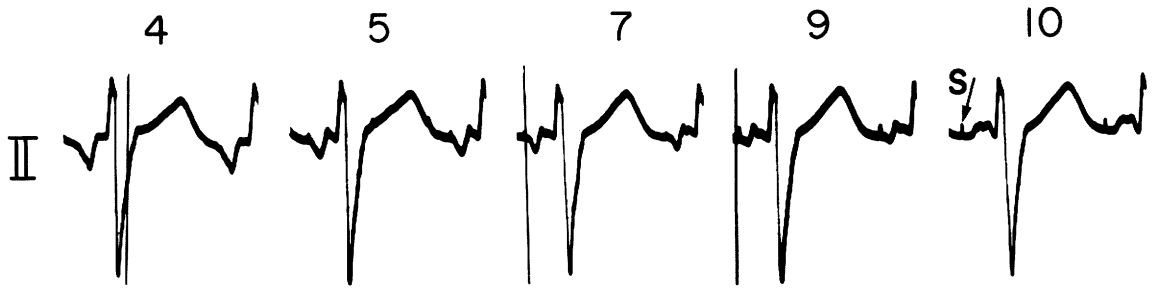

Figure 4

The P waves recorded in ECG lead II for patient 20, when the atria were paced from endocardial sites on either side of (sites 4 and 10) and from sites within this patient's endocardial transition zone (sites 5, 7, and 9). S represents the stimulus artifact. A time line appears in the records from sites 4, 7, and 9. Paper speed is 50 mm/sec.

Summary of P-wave Polarity for all Endocardial Pacing Sites

ECG leads	Polarity	Sites 1	2	3	4	5	6	7	8	9	10	11
II	+	0	0	0	0	0	0	12	27	**50**	**100**	**100**
	-+	8	32	15	6	39	40	**71**	**64**	43	0	0
	-	**92**	**68**	**85**	**94**	**61**	**60**	17	9	7	0	0
III	+	0	0	0	0	0	0	17	29	27	**100**	**100**
	-+	10	10	9	9	22	14	**58**	**71**	**64**	0	0
	-	**90**	**90**	**91**	**91**	**78**	**86**	25	0	9	0	0
aVf	+	0	0	0	0	0	0	20	33	30	**100**	**100**
	-+	11	9	8	11	30	14	**60**	**50**	**60**	0	0
	-	**89**	**91**	**92**	**89**	**70**	**86**	20	17	10	0	0

Figure 5

All numbers are expressed in per cent and indicate the percentage of time the P waves in ECG leads II, III, and aV_F were either positive (+), biphasic (− +), or negative (−) on pacing from right atrial endocardial sites 1 to 11. Shaded areas indicate an incidence of 50% or more.

always were negative when the atria were paced from the PLA and CRA (fig. 3A). When the atria were paced from the CST, the P waves in leads II, III, and aV_F were predominantly negative although they tended to be flat (fig. 3A). When the atria were paced from the MST, the P waves in leads II, III, and aV_F were predominantly positive although they tended to be flat (fig. 3B). When the atria were paced from the SA, the P

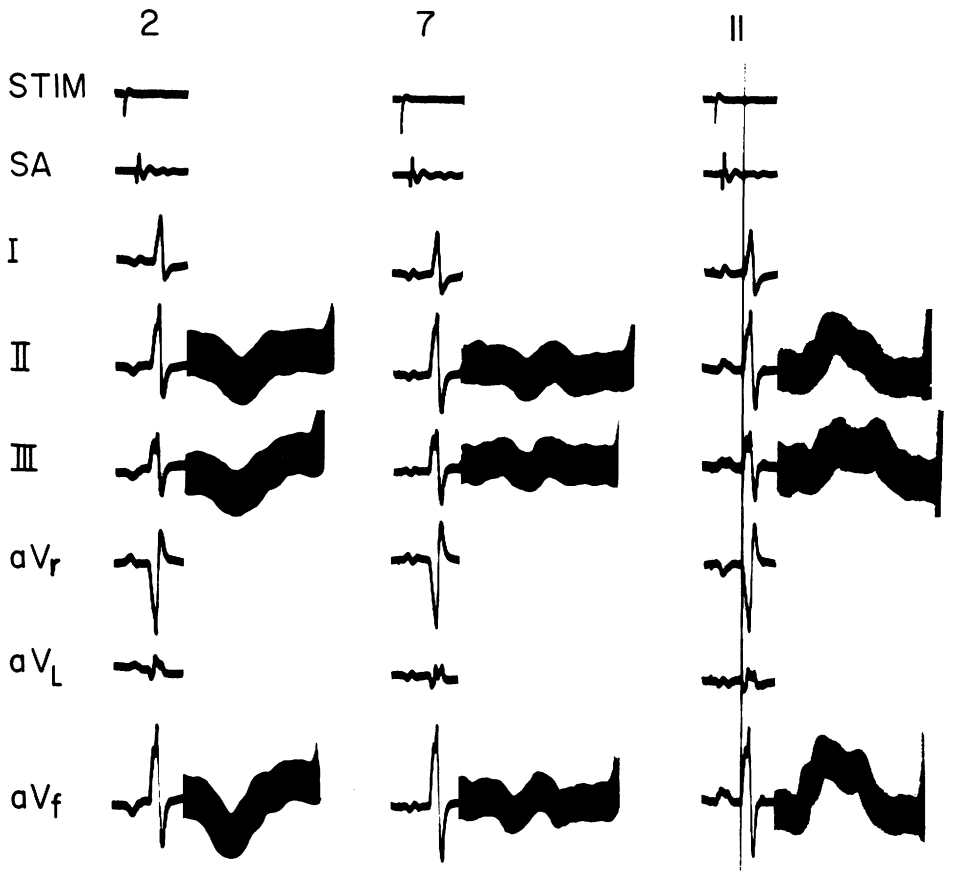

Figure 6

P waves recorded in all standard and augmented leads from patient 17 when the atria were paced from right atrial endocardial sites 2, 7, and 11. The P waves in leads II, III, and aV_F have been enlarged five times and appear beside the complete record. Stim identifies the stimulus artifact delivered through the pacing electrode. SA identifies the bipolar electrogram recorded from the region of the sinoatrial node. A time line appears in the record from site 11. Paper speed is 50 mm/sec.

waves in leads II, III, and aV_F always were positive (fig. 3B).

When the atria were paced from sites around the coronary sinus ostium (endocardial sites 1 to 4), the P waves in leads II, III, and aV_F almost always were negative. Medial to the coronary sinus ostium, there was a transition zone which generally extended from endocardial site 7 through site 9 and often included sites 5 and 6. Stimulation within this zone resulted in P waves in leads II, III, and aV_F which were biphasic (−,+). Stimulation at sites medial to the transition zone (endocardial sites 10 and 11) resulted in P waves in leads II, III, and aV_F which were frankly positive. Figure 4 shows for lead II the change in P-wave polarity and morphology when the atria of patient 20 were paced from sites on either side of and from within the transition zone. When the atria were paced from sites in the transition zone closest to the coronary sinus ostium (for example, site 5 in patient 20), the positive portion of the biphasic P wave in leads II, III, and aV_F was small and the major portion of the P wave was negative. When the atria were paced from sites within the transition zone but farther from (that is, medial to) the coronary sinus ostium (for example, sites 6 to 9 in patient 20), the positive portion of the biphasic P wave in

leads II, III, and aV_F became larger and the negative portion smaller. Medial to the transition zone (sites 10 and 11) the P wave was positive. The location and extent of this endocardial transition zone varied somewhat from patient to patient. Figure 5 summarizes in percentage form the P-wave polarity in leads II, III, and aV_F for all endocardial pacing sites. In relation to figure 5, it must again be noted that although lead II was recorded from each patient, leads III and aV_F were not always recorded simultaneously. For some patients lead II was recorded only with lead aV_F; for others, only with lead III, and for some, with neither lead III nor aV_F.

Figure 6 demonstrates the spectrum of P waves recorded from patient 17 in all the standard and augmented leads when the atria were paced from three of the endocardial sites. It again illustrates the negative P waves in leads II, III, and aV_F recorded when the atria were paced from sites around the coronary ostium (site 2), the biphasic P waves recorded in leads II, III, and aV_F when the atria were paced from within the endocardial transition zone (site 7), and the positive P waves recorded in the same leads when the atria were paced from sites medial to the transition zone (site 11). Careful examination of the P waves recorded when the atria were paced from endocardial site 7 suggests that the P waves in lead III and probably in lead aV_F are triphasic (+, −, +) while the P wave in lead II is biphasic (−, +). In three other patients, the polarity and morphology of the P waves recorded in lead III or aV_F or both leads were somewhat different from the P wave in lead II when the atria were paced from some but not all of the endocardial transition zone sites. However, the point of figure 6, as well as figures 4 and 5, is that the P waves in ECG leads II, III, and aV_F are not solely negative when the atria are activated from endocardial sites low in the right atrium. Depending on the site paced, P waves in these leads may be biphasic (−, +) or positive. Furthermore, it must be emphasized that the distance from endocardial site 5 to sites 10 and 11 was only 1.5 cm. Thus, the changes in P wave polarity and morphology which we recorded occurred when the atria were paced from endocardial sites separated only by short distances.

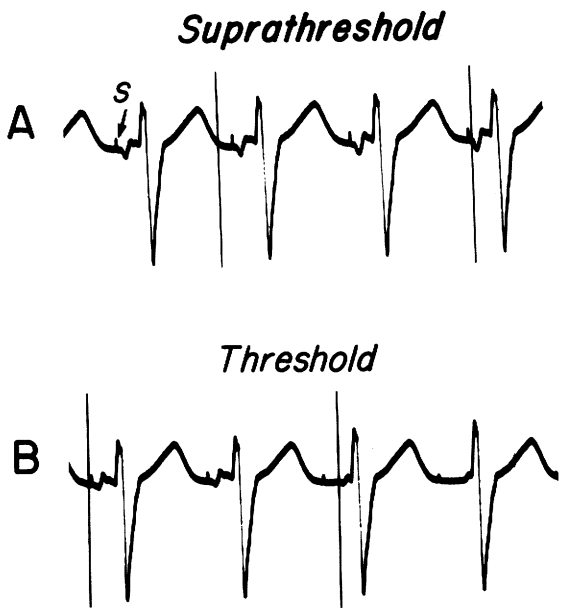

Figure 7

The P waves recorded in lead II from patient 20 when right atrial endocardial site 9 was paced. In panel A the stimuli were suprathreshold. During an interval of 34 beats, between the end of panel A and the beginning of panel B, the stimulus strength was lowered, becoming threshold for the first two beats in panel B and subthreshold for the third beat in panel B. Note that when the stimulus strength becomes subthreshold, atrial capture is lost. S indicates the stimulus artifact. Time lines are at intervals of 1 sec; paper speed is 50 mm/sec.

Stimulus strength also had an effect on the P wave at some endocardial sites. The polarity and morphology of the P wave differed for suprathreshold and threshold stimuli. Figure 7 illustrates for endocardial site 9 in patient 20 the change in P-wave polarity and morphology in lead II that occurred when the stimulus strength was decreased from suprathreshold to just threshold. Note that the biphasic (−, +) P wave has a prominent initial negative deflection during suprathreshold pacing, but when the stimulus was just threshold, the P wave, while still biphasic, is almost entirely positive.

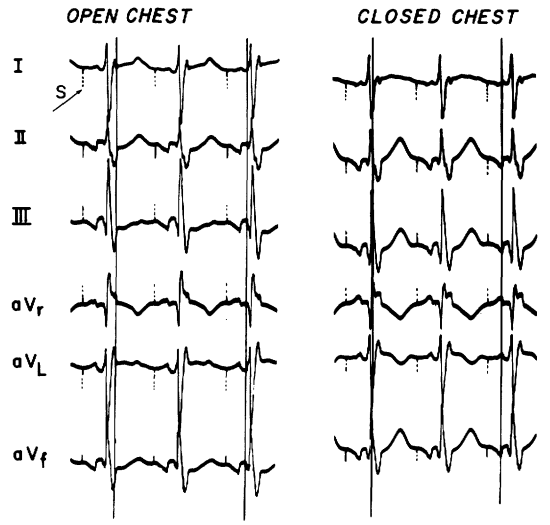

Figure 8

Standard and augmented ECG leads recorded from patient 37 when the atria were paced through the implanted wire electrodes from the same site (CRA) at the same rate in the operating room when the chest was open and during the first postoperative day. (The P-R intervals are 0.15 sec and the S-P intervals are 20 msec in each instance. See also the section on the P-R interval in "Results"). S represents the stimulus artifact. Time lines are at 1 sec intervals. Paper speed is 50 mm/sec.

Comparison of P-Wave Polarity and Morphology When the Chest Was Open and Closed

The same atrial site was paced in the operating room and during the immediate postoperative period in 10 patients and the recorded ECGs compared. In all instances, when such comparisons were made, the polarity of the P waves in leads II, III, and aV_F was identical, and the morphology of the P waves in leads II, III, and aV_F, although never identical, was similar. This confirms the observations of Moore and associates[15] and Spach and co-workers.[26] Figure 8 illustrates for patient 37 the polarity and morphology of the P waves in the standard and augmented ECG leads recorded when the atria were paced through the same wire electrodes at the same rate both in the operating room when the chest was open (left panel) and during the first postoperative day (right panel). Note that the P waves in leads II, III, and aV_F are identical in polarity and similar in morphology. We have therefore concluded that the open chest did not significantly affect our data.

The P-R Interval*

While this study was initially designed to examine P-wave polarity and morphology when the heart was paced from selected atrial sites, it soon became evident that the studies also provided important data regarding the P-R interval. As demonstrated in figure 3A and B and summarized in table 2, when the atria were paced from the selected epicardial sites, the P-R interval was always greater than 0.12 sec.† When the endocardial sites were paced,

*The P-R interval was measured from the earliest inscription of the P wave in any of the simultaneously recorded ECG leads to the earliest inscription of the QRS complex in any of the simultaneously recorded ECG leads.

†In two of seven patients in whom site CS epi was paced, intervals of 0.117 sec and 0.109 sec were recorded. The proximity of this site to the endocardial sites of the coronary sinus ostium (1 to 4) may explain the short P-R interval in these two instances (cf.). The range of P-R intervals for this site was 0.109 to 0.144 sec, the mean being 0.130 sec. The P waves in ECG leads II, III, and a V_F were always negative when this site was paced.

Table 2

Summary of P-R Intervals Recorded When Epicardial Atrial Sites Were Paced

Epicardial site	P-R intervals			
	% 0.12 sec or more	% less than 0.12 sec	Range (sec)	Mean (sec)
PLA	100	0	0.143 – 0.189	0.165
CRA	100	0	0.151 – 0.172	0.146
CST	100	0	0.168 – 0.213	0.185
MST	100	0	0.140 – 0.198	0.172
SA	100	0	0.131 – 0.208	0.159

Abbreviations: Defined in text and legend for figure 1.

Table 3

Summary of P-R Intervals Recorded When Endocardial Atrial Sites Were Paced

Endocardial site	P-R intervals			
	% 0.12 sec or more	% less than 0.12 sec	Range (sec)	Mean (sec)
1	9	91	0.098 – 0.125	0.101
2	9	91	0.092 – 0.120	0.108
3	17	83	0.089 – 0.127	0.098
4	0	100	0.077 – 0.113	0.090
5	0	100	0.080 – 0.119	0.090
6	0	100	0.073 – 0.112	0.098
7	0	100	0.089 – 0.119	0.104
8	0	100	0.083 – 0.113	0.100
9	12	88	0.098 – 0.124	0.108
10	14	86	0.088 – 0.125	0.104
11	25	75	0.094 – 0.126	0.107

the P-R intervals were, almost without exception, less than 0.12 sec (table 3). Pacing from endocardial sites closest to the A-V junction, sites 4 to 8, always resulted in P-R intervals of less than 0.12 sec. P-R intervals as short as 0.073 sec were recorded when pacing from site 6, and P-R intervals shorter than 0.099 sec were often recorded when pacing from all endocardial sites. The mean P-R interval for each endocardial pacing site was short and ranged from 0.090 to 0.108 sec. In the few instances in which the P-R intervals were greater than 0.12 sec, they did not exceed 0.127 sec (table 3).

These data clearly show that P-R intervals of less than 0.12 sec can result from initiation of impulses in the right atrium. In evaluating these data, it should be noted that several hours prior to surgery most patients received atropine or scopolamine (table 1), both of which can decrease atrioventricular conduction time.[40] However, in the doses administered, these drugs probably did not significantly affect the P-R intervals recorded when the atria were paced. Evidence for this is provided by the fact that the P-R intervals recorded in the operating room prior to thoracotomy were comparable to those recorded at a similar heart rate prior to surgery and prior to administration of preanesthetic medication. Also, the P-R intervals recorded from patients who had received neither of these drugs also were less than 0.12 sec when the atria were paced from all endocardial sites, except for a P-R interval of 0.12 sec, recorded in one patient when endocardial site 1 was paced.

In further consideration of factors tending to affect the P-R interval, it must be noted that many of the patients were studied at temperatures lower than 37 C (table 1). This would, if anything, prolong atrioventricular conduction time.[41] Also, pacing the heart at a rate slightly faster than the spontaneous rate would, if anything, prolong atrioventricular conduction time.[42] Thus, the P-R intervals recorded in these studies may, if anything, be slightly

Table 4

Summary of Data Pertaining to S-P Interval

Sites	% present	Range (msec)	Mean (msec)
	Epicardial		
PLA	56	0-16	7
CRA	100	12-32	23
CST	100	10-40	23
MST	100	12-40	22
SA	50	0-20	9
	Endocardial		
1	82	0-56	21
2	93	0-46	20
3	93	0-31	18
4	92	0-59	28
5	87	0-32	19
6	70	0-35	15
7	55	0-31	8
8	88	0-42	23
9	57	0-32	11
10	86	0-47	21
11	67	0-51	23

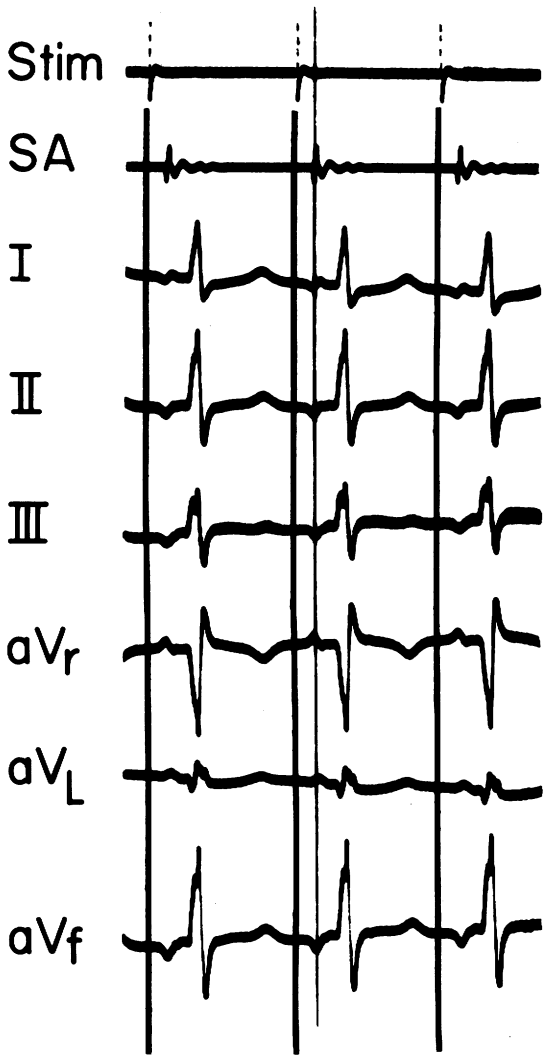

Figure 9

An S-P interval of 20 msec was recorded when pacing the atria from endocardial site 2 in patient 17. A line has been drawn through the stimulus artifact to demonstrate the 20-msec isoelectric interval between the stimulus artifact and the onset of the P wave. Stim identifies the stimulus artifact, and SA identifies the bipolar electrogram recorded from the region of the sino-atrial node. A time line appears through the P wave of the second beat on this record. Paper speed is 50 mm/sec.

prolonged because of the low temperature and a pacing rate faster than the spontaneous rate.

One important interval remains to be discussed. Frequently, there was an interval (S-P interval) between the time of stimulation of the atrium and the first clear evidence of the inscription of the P wave (table 4 and fig. 9). This interval, when present, was generally between 0.01 and 0.025 sec although it was once as long as 0.059 sec. Thus, in many instances, the beginning of the P wave does not necessarily coincide with the beginning of atrial activation. This observation clearly reflects on the utility of rigid criteria for the P-R interval in determining the site of origin of atrial activity.

Discussion

Our results suggest that it may be necessary to reevaluate the criteria employed for the diagnosis of certain arrhythmias (such as A-V nodal rhythm, coronary nodal rhythm, and coronary sinus rhythm) since the identification of these rhythms depends primarily on the polarity and morphology of the P wave and the duration of the P-R interval.

Upper Nodal Rhythm

In the absence of conduction defects, an electrocardiogram showing a P-R interval of less than 0.12 sec and negative P waves in leads II, III, and aV_F usually is thought to represent upper nodal rhythm with retrograde activation of the atria.[2-5] If these criteria were employed, the records we have obtained when the atria were paced from the vicinity of the coronary sinus ostium would be classified as upper nodal rhythm. Our data clearly show that negative P waves in leads II, III, and aV_F and P-R intervals of less than 0.12 sec and as short as 0.077 sec can result from impulses initiated in the right atrium.

Coronary Nodal Rhythm

This rhythm is characterized by positive P waves in leads I and II and a P-R interval between 0.10 and 0.02 sec.[6, 7] We have recorded short P-R intervals, positive P waves in lead II, and positive or biphasic (−, +) P waves in lead I when the atria were paced from endocardial sites 10 and 11 and sometimes from sites 7, 8, and 9. While we never have recorded P-R intervals less than 0.083 sec while pacing from these sites, our evidence suggests that some spontaneous arrhythmias identified as coronary nodal rhythm may

result from initiation of activity at one of these endocardial sites.

Coronary Sinus Rhythm

A P-R interval of 0.12 sec or more, in association with a negative P wave in leads II, III, and aV_F, usually is thought to represent either a rhythm originating in the coronary sinus[1, 3-5] or an A-V junctional rhythm with retrograde capture of the atria and markedly delayed conduction to the ventricles.[1-5] We have shown that if atrial activity is initiated at appropriate sites in left atrium (PLA) and right atrium (CRA), the electrocardiogram shows a P-R interval greater than 0.12 sec and negative P waves in leads II, III, and aV_F. To designate all such tracings as coronary sinus rhythm clearly is incorrect.

Our findings are in agreement with results obtained from studies on the canine heart when stimuli were applied to the same parts of the atria.[10, 43, 44] Also, others have paced the human heart through catheters placed within the coronary sinus and recorded negative P waves in leads II, III, and aV_F and P-R intervals of 0.12 sec or more.[45-50] It seems likely that in these studies the atria actually were activated from the PLA. As shown in figure 1, most of the coronary sinus lies adjacent to the left atrium, and, as shown in figure 2, only its ostium is within the right atrium. Because of these anatomic relationships, any catheter advanced into the coronary sinus necessarily lies adjacent to the PLA. It reasonably follows that, when pacing the atria through such a catheter, the impulse is initiated in the region of site PLA. Harris and associates[51] paced the PLA through a catheter inserted in the left atrium and recorded negative P waves in leads II, III, and aV_F and P-R intervals of more than 0.12 sec. Prinzmetal and co-workers,[29] also produced negative P waves in the same leads by mechanical stimulation of the human heart at right and left atrial sites comparable to PLA and CRA; their results differ from ours, however, in that they recorded P-R intervals shorter than 0.12 sec.

Lower Nodal Rhythm

We have previously described the polarity and morphology of the P wave during retrograde capture of the atria in some rhythms initiated at sites in or distal to the NH region of the A-V node.[16] In that study, we found retrograde P waves to be primarily biphasic in the standard and augmented leads. In leads II, III, and aV_F the retrograde P wave was most often largely negative with a late, small positive component, much as we have demonstrated in this study for stimulation of sites in the endocardial transition zone close to the coronary sinus ostium. Our impression, based on a review of a large series of ECGs demonstrating A-V junctional rhythm, confirms the observation of Puech[24] that the P waves in leads II, III, and aV_F are often negative but may, in fact, be biphasic (−, +). It should be noted that the positive component of this biphasic type of P wave may often be masked within the T wave, resulting in a P wave in II, III, and aV_F which appears as a completely negative deflection.[16] Also, the initial negative portion of the P wave in leads II, III, and aV_F may be masked within the preceding QRS complex during A-V junctional rhythm with retrograde atrial capture, resulting in a P wave which appears to be positive in II, III, and aV_F.[16]

A-V Junctional and Low Atrial Rhythms

We have previously defined an A-V junctional rhythm as a rhythm initiated by a pacemaker located somewhere between the NH region of the A-V node and the bifurcation of the bundle of His.[16] None of the results presented here appear to require modification of this definition. Also, records obtained by the use of catheter electrodes to record from the His bundle[52] have provided data which are in agreement with this definition and with the earlier conclusion of Hoffman and Cranefield[53] that automatic cells are found in the NH region of the A-V node and in the His bundle but not in the AN or N regions of the node. We suggest, therefore, that the term "A-V junctional rhythm" should

be employed to describe what formerly has been called lower nodal rhythm.

Our data show that the electrocardiographic patterns thought to be characteristic of upper nodal rhythm can result from activation of the atria in the vicinity of the coronary sinus ostium. This finding is consistent with the conclusions of Hoffman and Cranefield[53] that cells in the AN region of the A-V node do not show automaticity. Although Watanabe and Dreifus[54] concluded that impulses might be initiated in the AN region of the A-V node, their data consisted of several instances of primary negativity recorded from this region. However, as Erlanger[55] pointed out, the recording of primary negativity in a unipolar lead identifies not the pacemaker but only the part of the atrium first activated by a pacemaker. Primary negativity recorded by Watanabe and Dreifus from the vicinity of the AN region thus may result from automatic firing of a pacemaker located in the region of the coronary sinus. This interpretation was also suggested by others.[52]

It has been postulated that coronary nodal rhythm is initiated by a pacemaker in a region near the tail of the SA node.[6, 7] However, this explanation does not seem likely since our data consistently showed that when the atria were paced from an epicardial site near the tail of the SA node (MST), the P-R interval was always greater than 0.12 sec. If the term "coronary nodal rhythm" implies that the pacemaker is located in the vicinity of the coronary sinus,[6, 7] our data are in clear disagreement with this electrocardiographic interpretation. We never have recorded positive P waves in lead II when the atria were paced from this site.

Our findings also conflict with the usual criteria employed for the diagnosis of coronary sinus rhythm. We have found negative P waves in leads II, III, and aV_F and P-R intervals greater than 0.12 sec associated with activity initiated low in the right and left atria (CRA and PLA).

Our data, obtained by simulation of hearts in which there were no known conduction defects, suggest that it probably is reasonable to designate these three rhythms (upper nodal, coronary nodal, and coronary sinus) as low atrial rhythms, in keeping with the suggestion of Pick and Langendorf[56] until a precise identification of the mechanism responsible for each has been provided.

P-R Interval

Our findings also suggest that another assumption employed in electrocardiographic interpretations should be reevaluated. If there is no reason to think that atrioventricular transmission time has changed, it usually is assumed that differences in the P-R interval reflect changes in the site of origin of the atrial impulse or, in unusual circumstances, changes in conduction time throughout atrial muscle. Our findings indicate that differences in P-R interval need not provide an accurate indication of the time lapse between initiation of the impulse and its arrival at the A-V junction. As we have shown, and others have shown previously,[12, 15, 57] there may be an isoelectric interval between the time of activation of the atrium and the first clear evidence of a P wave in the body surface leads. Thus, the initial inscription of the P wave does not necessarily provide any indication of the time of initiation of the atrial impulse or the anatomic site of impulse initiation. Unless one assumes that the phenomena we have described would not occur when the atria are activated by intrinsic pacemakers, it seems reasonable to conclude that the beginning of the P wave provides, at best, only an imprecise index of the time at which a spontaneous impulse is initiated in the atrium. Furthermore, it seems likely that in the presence of inflammatory or degenerative disease, or perhaps after administration of certain drugs, the phenomena which we have described would assume greater importance. A short P-R interval, thus, may reflect in part the fact that the initial spread of activity from its site of origin does not generate a recognizable contribution to the electrocardiogram and that changes in P-R interval may result, not from change in the location of the pacemaker, but rather from changes in the time required for the impulse to activate a

sufficient segment of atrial muscle to cause a visible deflection in the ECG.

P-Wave Polarity

We have shown that the P wave in leads II, III, and aV_F is not always negative when the atria are activated from sites low in the right atrium. The P wave may be biphasic (−, +) or positive. It is difficult to explain these findings on the basis of the Lewis thesis[10] that spread of excitation in the atria is radial. If this were so, initiation of atrial activation at sites low in the right atrium should always result in negative P waves in leads II, III, and aV_F. Our results would be better explained if specialized atrial pathways or internodal tracts[31, 32] play a role in determining the sequence of atrial activation.

The likelihood of functionally significant specialized pathways in the atria was first emphasized by Eyster and Meek.[58–60] Many anatomic studies[31, 32, 61–65] have demonstrated at least three such tracts, the anterior (AINT), middle (MINT), and posterior (PINT) internodal tracts. Furthermore, James[31] demonstrated that a branch of the anterior tract extends to the left atrium in Bachmann's bundle (the interatrial band). Evidence for specialized fibers in Bachmann's bundle received physiologic support from the work of Wagner and associates[66] and Childers and co-workers.[67] Also, Vassalle and Hoffman[68] and Holsinger and associates[69] have demonstrated by physiologic technics the probable existence of specialized paths between the SA and A-V nodes.

It is reasonable to suppose that these pathways are important in determining the sequence of activation in the atria. They, therefore, must play an important role in determining the polarity and morphology of the P wave. This becomes even more likely when one considers the finding that changing the pacing site by only several millimeters in the endocardial transition zone usually results in significant change in P-wave polarity and morphology. The AINT, MINT, and PINT, as they approach and enter the A-V node, probably run close to most of the endocardial pacing sites.[31, 32] It is not difficult, then, to modify the hypothesis of Daniélopolu and Proca[19] and Brumlík[25] and suppose that an impulse can rapidly spread retrograde, probably through one or more of the internodal tracts, to Bachmann's bundle and the region of the SA node and from there can depolarize significant areas of both atria in a sequence similar to that which occurs during so-called normal sinus rhythm. The important point is that under these conditions a significant portion of the left atrium and some of the right atrium would be depolarized in a "normal" sequence. This is consistent with the data recently presented by Spach and associates[26] and would explain the positive P waves in leads II, III, and aV_F which are recorded when the atria are activated from some of the right atrial endocardial pacing sites.

No specialized pathways have been described in the PLA region[30, 31] which, when stimulated, results in negative P waves in leads II, III, and aV_F. Excitation of this region, therefore, could result in radial spread and truly retrograde activation. Negative P waves in leads II, III, and aV_F thus would be expected when this area is paced.

Data which would permit a complete explanation, in terms of the sequence of activation, for the negative P waves produced in leads II, III, and aV_F when the right atrium is paced are not yet available. Also, there is no ready explanation for the biphasic (−, +) P waves recorded in leads II, III, and aV_F when the atria are paced from sites within the transition zone on the right atrial endocardium. It is clear, however, that the closer the endocardial pacing sites are to the posterior left atrium, or conversely, the farther these sites are from the point where the AINT approaches the A-V node, the greater the negative portion and the smaller the positive portion of the P wave. When the atria are paced from the lips of the coronary sinus ostium, the P wave is almost always totally negative. This implies that if the impulse reaches the posterior left atrium quickly and begins to depolarize the left atrium in retrograde fashion before the activity reaches

Bachmann's bundle via the AINT, much of the P wave in leads II, III, and aV_F will be negative. The longer it takes for activity to reach the posterior left atrium relative to its arrival at Bachmann's bundle, the greater the positive portion of the P wave. We have previously demonstrated[70] in canine hearts that a negative P wave in leads II, III, and a aV_F is most consistently associated with early activation of the postero-inferor region of the left atrium (PLA). In this regard, it is worth suggesting that, when stimuli are applied to the caudal portion of the right atrial epicardium, the impulse spreads rapidly to the posterior left atrium, with subsequent retrograde radial activation of the left atrium. Furthermore, it is reasonable to assume that an impulse initiated at this site would not spread rapidly to Bachmann's bundle, and furthermore that it would depolarize the right atrium in a retrograde, as opposed to a "normal," fashion resulting in negative P waves in leads II, III, and aV_F.

Acknowledgment

The authors would like to express their appreciation to Dr. M. Irené Ferrer for her valuable comments and criticisms.

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