Determinants of Antegrade Echo Zone in the Wolff-Parkinson-White Syndrome

Edward L. C. Pritchett, M.D., John J. Gallagher, M.D., Melvin Scheinman, M.D., and William M. Smith, Ph.D.

SUMMARY Forty-five patients with the Wolff-Parkinson-White syndrome and reciprocating tachycardia which utilized the atrioventricular (A-V) node for antegrade conduction and an accessory pathway for ventriculo-atrial (retrograde) conduction were studied. The extrastimulus technique in the right atrium and coronary sinus at multiple pacing cycle lengths was used. Three types of "echo zones" were defined by the relationship of the antegrade effective refractory period (ERP) of the accessory pathway, the longest coupling interval accompanied by an atrial echo, the ERP of the A-V node, and the functional refractory period of the atrium. Eighteen of 45 patients (40%) had more than one type of echo zone demonstrated by changing either pacing site or pacing cycle length or both. Pacing site in-

traventricular conduction delay were found to have important effects on the longest coupling interval accompanied by an atrial echo in those echo zones in which the longest coupling interval accompanied by an atrial echo was less than the antegrade ERP of the accessory pathway.

The echo zone is a useful concept to explain the physiology of the common reciprocating tachycardia in patients with the Wolff-Parkinson-White syndrome. However, the echo zone is profoundly affected by pacing site and pacing cycle length. When these variables are changed, many patients can be shown to have more than one type of echo zone. Studies of the echo zone have limited value as therapeutic guides.

THE COMMON RE-ENTRANT TACHYCARDIA which occurs in patients with the Wolff-Parkinson-White syndrome is an exceptional model with which to study re-entrant rhythms. Using programmed electrical stimulation, Durrer et al. showed that premature atrial beats which were not conducted with ventricular pre-excitation but were conducted to the ventricles with normal QRS morphology initiated an atrial echo and reciprocating tachycardia. The coupling intervals used during a refractory period determination could then be divided into a "zone of ventricular pre-excitation" and an "echo zone." Subsequent electrode catheter studies, epicardial mapping studies, and surgical correction of the Wolff-Parkinson-White syndrome have established that this common re-entrant rhythm in these patients uses the A-V node for antegrade conduction and an accessory pathway for ventriculo-atrial (retrograde) conduction. The refractory periods of these conduction pathways constitute the limits of the echo zone, and are altered as pacing cycle length and (sometimes) as pacing site change. The purpose of this study was, therefore, to characterize the determinants of the antegrade echo zone in patients with the Wolff-Parkinson-White syndrome and to examine whether the antegrade echo zone was changed in any way by changes in pacing site or cycle length. Since changes in the antegrade echo zone have been proposed as a potential guide to drug therapy, the variations accompanying change of pacing site have important clinical implications.

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Methods

All patients presented here were among those referred to the Duke University Medical Center for evaluation of arrhythmias associated with the Wolff-Parkinson-White syndrome. Every patient had ventricular pre-excitation due to an accessory atrioventricular pathway and in every patient a reciprocating tachycardia was induced in which atrioventricular (antegrade) conduction passed through the A-V node and ventriculo-atrial (retrograde) conduction passed along the accessory pathway. The technique of study of these patients has been described in detail. As part of this study, refractory period determinations were routinely done using two atrial pacing sites. In these determinations every eighth paced beat was followed by an induced premature beat at progressively shorter coupling intervals until atrial refractoriness was reached. When the coupling interval of the extrastimulus approached a critical value (antegrade ERP of the accessory pathway, upper limit of the echo zone, atrial refractoriness, etc.), 10 msec decrements were used. Refractory periods determined in recently studied patients were measured using an interactive computer program. This program has been shown to allow blinded observers to make measurements which are reproducible within 2 msec. For most patients complete refractory period determinations were done at two or three cycle lengths using catheters in both the right atrium and the coronary sinus. In some patients the length of the study, the instability of the clinical condition, technical problems associated with catheter placement, or the frequent occurrence of atrial fibrillation precluded complete determinations. This report describes results in 45 patients who had complete antegrade refractory period determinations using equal cycle lengths and two pacing sites or one pacing site at two cycle lengths different by at least 100 msec. Almost all patients had observations made using at least two cycle lengths at both pacing sites.

Definitions

S₁, A₁, H₁, and V₁ refer to the stimulus, atrial electrogram, His Bundle electrogram, and ventricular electro-
gram of the basic drive beat. S₀, A₀, H₀, and V₀ refer to the stimulus, atrial electrogram, His Bundle electrogram, and ventricular electrogram of the induced extra beat. CSA and CSV refer to the atrial and ventricular electrograms recorded on a catheter in the coronary sinus. HBEA and HBEV refer to the atrial and ventricular electrograms recorded on the catheter recording the bundle of His electrogram. RA refers to the atrial electrogram recorded in the right atrium.

A₁₋ₐ₁ refers to the atrial electrogram of the first atrial echo following an induced premature beat.

Effective refractory period (ERP) of the A-V node: The longest A₁₋ₐ₂ interval not followed by an H₄ nor conducted with pre-excitation.

ERP of the atrium: The shortest A₁₋ₐ₂ interval attainable at a specified site.

FRP of the atrium: The shortest A₁₋ₐ₂ interval not eliciting an A₂.

Antegrade ERP of the accessory pathway: Longest A₁₋ₐ₂ interval not conducted with ventricular pre-excitation. This measurement is made on a catheter positioned near the accessory pathway.

Antegrade echo zone: All coupling intervals (A₁₋ₐ₂) in which A₂ is conducted to the ventricle without pre-excitation, returns to the atrium using the accessory pathway, and initiates an atrial echo (A₂₋ₐ₁).

Upper limits of the echo zone: The longest coupling interval (A₁₋ₐ₂) in which A₂ is conducted to the ventricle without pre-excitation, returns to the atrium using the accessory pathway, and initiates an atrial echo (A₂₋ₐ₁).

Lower limit of the echo zone: Either the FRP of the atrium or ERP of the A-V node, whichever is longer. All echo zones described here were continuous, i.e., every coupling interval (A₁₋ₐ₂) between the upper and lower limit of the echo zone could initiate an atrial echo.

Range of the echo zone: The difference between the upper limit of the echo zone and the lower limit of the echo zone.

Echo time: (A₂₋ₐ₁) is the interval between the atrial electrograms of the induced premature beat (A₁) and the first atrial echo (A₂₋ₐ₁) recorded at a specified site during stimulation at a specified site. The minimum echo time is the shortest A₁₋ₐ₁ recorded on a catheter positioned near the accessory pathway.

Results

Types of Antegrade Echo Zones

Three types of antegrade echo zones were described by the relationship of antegrade ERP of the accessory pathway, the upper limit of the echo zone, the ERP of the A-V node, the FRP of the atrium, and the minimum echo time determined at each pacing site and each cycle length. Twenty-seven of the 45 patients had the same type of antegrade echo zone at all cycle lengths and both pacing sites.

Type I Antegrade Echo Zone (No Echo)

If no echoes were induced during antegrade refractory period determinations because the FRP of the atrium exceeded the antegrade ERP of the accessory pathway (fig. 1A) or the ERP of the A-V node was equal to or greater

![FIGURE 1. Type of echo zones. FRP = functional refractory period; ERP = effective refractory period; LIM EZ = upper limit of the echo zone. In Type I no echoes occur. Type II is the "classic" Wolff-Parkinson-White echo zone. In Type III the zone of pre-Excitation is separated from the echo zone by a range of coupling intervals in which V₄ has normal QRS morphology but no echoes occur. The physiology of the Type III echo zone resembles the physiology of the echo zone in patients with accessory pathways which lack antegrade conduction ("concealed Wolff-Parkinson-White syndrome").]
than the antegrade ERP of the accessory pathway (fig. 1B), then the echo zone was described as Type I. (The setting defined by this Type I relationship was not a true "echo" zone because no echoes were observed at any coupling interval. However, the term "echo zone" was used in this setting because of its relationship to other echo zones.) Ten patients had Type I antegrade echo zones at all cycle lengths and both pacing sites.

Type II Antegrade Echo Zone (Classic Antegrade Echo Zone)

If every coupling interval which was conducted to the ventricles exclusively using the A-V node initiated an echo, then the antegrade echo zone was described as Type II (fig. 1C). In Type II antegrade echo zones the upper limit of the echo zone was the ERP of the accessory pathway, and all shorter coupling intervals initiated atrial echoes. Type II antegrade echo zones fit the classic description of the antegrade echo zone of Durrer et al. Figure 1C shows that there was an inverse relationship between coupling interval and echo time in these patients. Fourteen patients had this type of antegrade echo zone using all cycle lengths and both pacing sites.

Type III Antegrade Echo Zones

This type of antegrade echo zone resembled Type II antegrade echo zones because coupling intervals shorter than the ERP of the accessory pathway were conducted to the ventricles using the A-V node and bundle of His. In Type II antegrade echo zones, all such beats with normal A-V conduction initiated atrial echoes, but in Type III antegrade echo zones, relatively long coupling intervals which were close to the ERP of the accessory pathway failed to initiate an echo. Therefore the zone of pre-excitation was not adjacent to the echo zone but was separated from it. The coupling intervals which separated these two zones were conducted with normal QRS morphology but failed to initiate atrial echoes (fig. 1D). The failure of these premature beats to initiate echoes persisted at progressively shorter coupling intervals until A-V node delay was sufficiently long to allow recovery of all return limbs of the re-entry circuit. When adequate delay was achieved, progressively shorter coupling intervals initiated echoes with progressively longer echo times. Therefore the inverse relationship between coupling interval and echo time observed in Type II antegrade echo zones occurred also in Type III. The minimum echo time, which was achieved near the upper limit of the echo zone, was equal (± 20 msec) to the ERP of the atrium measured during stimulation with a catheter near the accessory pathway.

In Type III echo zones, events which allowed more time to elapse before the return limb of the circuit was entered shifted the upper limit of the echo zone to longer coupling intervals. Thus if V₃ was conducted with bundle branch block in the ventricle containing the insertion of the accessory pathway, longer coupling intervals initiated echoes. For example, figure 2 shows records from a patient with an

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Right atrial pacing at CL = 600 msec; S₁–S₂ = 340 msec. Panel A) V₄ had normal QRS morphology. The H₁–H₄ interval was 395 msec. No echo followed. Panel B) The A₁–A₂ interval and H₁–H₂ interval are the same as panel A. V₃ had left bundle branch block morphology. The additional intraventricular delay allowed atrial excitability to recover and reciprocating tachycardia was initiated. V₁ = ECG lead V₁. RV = right ventricular electrogram; LLRA = low lateral right atrial electrogram; HBE = His bundle electrogram; Prox CS = proximal coronary sinus electrogram.
TABLE 1. Echo Zone Measurements in Three Patients with Type III Echo Zones

<table>
<thead>
<tr>
<th>Pt</th>
<th>AP</th>
<th>Site</th>
<th>CL</th>
<th>ERP-Atr†</th>
<th>Min echo time</th>
<th>ERP, AP</th>
<th>Echo zone, upper limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Left</td>
<td>CS</td>
<td>600</td>
<td>264</td>
<td>271 &gt;600</td>
<td>315</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>RA</td>
<td>600</td>
<td>—</td>
<td>261 &gt;600</td>
<td>298</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>CS†</td>
<td>400</td>
<td>260</td>
<td>268 &gt;400</td>
<td>279</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Left</td>
<td>CS</td>
<td>600</td>
<td>255</td>
<td>265 &gt;600</td>
<td>355</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>RA</td>
<td>600</td>
<td>—</td>
<td>250 &gt;600</td>
<td>315</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Right</td>
<td>CS</td>
<td>800</td>
<td>302</td>
<td>288</td>
<td>436</td>
<td>374</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RA</td>
<td>800</td>
<td>311</td>
<td>429</td>
<td>399</td>
<td></td>
</tr>
</tbody>
</table>

*Based on an interactive computer system of measurement.† Measured at pacing site closest to the accessory pathway.

No observations were made using right atrial pacing at cycle length 400.

Abbreviations: AP = accessory pathway; Atr = atrium; ERP = effective refractory period; RA = right atrium; CL = cycle length.

accessory pathway located in the left A-V groove during right atrial refractory period determination. In panel A, the A1-A2 interval was 340 msec and the QRS morphology of V2 was normal. The H1-H2 interval was 395 msec and no echo occurred. In panel B, the A1-A2 interval and H1-H2 interval were the same as in panel A, but the QRS morphology of V2 was left bundle branch block. The additional delay in the ventricular limb allowed atrial excitability to recover and reciprocating tachycardia was induced. Similar effects were produced by the change in transatrial conduction time accompanying change in stimulation site. In patients who had Type III echo zones using both pacing sites, the upper limit of the echo zone was shifted to a longer coupling interval by pacing at the site closer to the accessory pathway. Table 1 contains measurements of the echo zone determined in three patients who had Type III echo zones using both pacing sites.

Effect of Changing Pacing Site and Cycle Length on Antegrade Echo Zone Type

Eighteen of the 45 patients had more than one type of antegrade echo zone demonstrated as pacing site or cycle length was changed. Figure 3 shows data from patient 6, in whom an accessory pathway was located in the left A-V groove. With right atrial pacing, the FRP of the atrium exceeded the FRP of the atrium during antegrade sinus pacing. With left atrial pacing, the FRP of the atrium exceeded the FRP of the atrium during antegrade sinus pacing.

TABLE 2. Echo Zone Type Changes Accompanying Change of Pacing Site and Cycle Length

<table>
<thead>
<tr>
<th>Pt</th>
<th>CL</th>
<th>Pacing site</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Accessory Pathways Located in the Right or Left A-V Groove</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>500</td>
<td>II</td>
</tr>
<tr>
<td>2</td>
<td>400</td>
<td>II</td>
</tr>
<tr>
<td>3</td>
<td>600</td>
<td>II</td>
</tr>
<tr>
<td>4</td>
<td>700</td>
<td>II</td>
</tr>
<tr>
<td>5</td>
<td>800</td>
<td>II</td>
</tr>
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<td>600</td>
<td>II</td>
</tr>
<tr>
<td>7</td>
<td>500</td>
<td>II</td>
</tr>
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<td>400</td>
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<td>300</td>
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<td>16</td>
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<td>II</td>
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<tr>
<td>17</td>
<td>300</td>
<td>II</td>
</tr>
<tr>
<td>18</td>
<td>200</td>
<td>II</td>
</tr>
</tbody>
</table>

Abbreviations: CL = cycle length; RA = right atrium; CS = coronary sinus.

Figure 3. Effect of pacing site and cycle length on the relationship of the FRP of the atrium and the ERP of the accessory pathway in patient 6. The FRP of the atrium exceeded the ERP of the accessory pathway at all cycle lengths using right atrial pacing. During coronary sinus pacing, the ERP of the accessory pathway exceeded the FRP of the atrium at cycle lengths 700 msec and 500 msec but not at cycle length 300 msec. These changes combined to produce Type II echo zones at cycle length 700 msec and 500 msec during coronary sinus pacing and Type I echo zones at all other combinations of pacing site and cycle length.
ceed the ERP of the accessory pathway at all cycle lengths, and therefore right atrial pacing produced only Type I echo zones. With coronary sinus pacing the ERP of the accessory pathway exceeded the FRP of the atrium at cycle lengths 700 msec and 500 msec, but not at a cycle length of 300 msec. In this patient all coupling intervals with which V2 had normal QRS morphology initiated echoes at cycle lengths 700 msec and 500 msec. These cycle lengths, therefore, produced Type II echo zones while a Type I echo zone was produced by cycle length 300 msec. The echo zone type observed at each cycle length and each pacing site for these 18 patients is given in table 2. Patients in whom the accessory pathway was located in the right or left A-V groove are described in section A. Patients in whom the accessory pathway was located in the septum are described in section B. The most common changes observed are those changes which were demonstrated in figure 3 by patient 6.

There were two patients in whom an antegrade echo zone was demonstrated at one combination of pacing site and cycle length which could not be classified into any of the three types. In these two determinations the ERP of the accessory pathway exceeded the upper limit of the echo zone, but the minimum echo time was much greater than the FRP of the atrium.

Discussion

The use of the extrastimulus technique and intracardiac recordings to study patients with the Wolf-Parkinson-White syndrome has helped in understanding the physiology of this syndrome and in selection of patients for surgical treatment.11 If these techniques are also to have utility in the selection of appropriate pharmacologic treatment of the common reciprocating tachycardia, then there must be some reproducible method of depicting the effect of drug interventions in serial studies. The "echo zone" is theoretically useful because an echo zone has a clearly measurable upper limit, lower limit, and range.12 However, this study indicates some very real practical limitations to this concept.

Table 3 shows the factors influencing the lower and upper limit of the echo zone which were operative in any one refractory period determination using one cycle length and one pacing site in this study. The range of the echo zone is equal to the difference between its upper limit and its lower limit. The relationship of upper and lower limits of the echo zone to the ERP of the accessory pathway defined three types of echo zones in this study. Previous studies of A-V conduction have shown that the FRP of the atrium, the ERP of the A-V node, and the antegrade ERP of the accessory pathway may all change as pacing cycle length changes. Further, all except the antegrade ERP

| TABLE 3. Factors Influencing the Upper and Lower Limits of the Antegrade Echo Zone at One Pacing Site and One Pacing Cycle Length |
|---|---|
| 1. Lower limit |
| ERP of the atrium |
| FRP of the A-V node |
| 2. Upper limit |
| ERP of the accessory pathway |
| Minimum echo time* |
| Intraventricular conduction |
| (concealed antegrade conduction into the accessory pathway) |

*Minimum echo time was equal to the FRP of the atrium (± 20 msec) in antegrade echo zone Type III in this study.

of the accessory pathway may change as pacing site changes.8,11 These studies have not, however, been addressed to the effect which these changes may have on the antegrade echo zone. This study shows that the relationship of these variables also changes with pacing site or cycle length in such a way as to cause the echo zone type to change in at least 40% (18/45) of patients studied.

In addition to changing echo zone type, changing pacing site may shift the upper limit of the echo zone in patients with a type III echo zone. In these cases the zone of preexcitation is separated from the zone of normal conduction by a range of coupling intervals in which premature beats are conducted with normal QRS morphology but do not initiate reciprocating tachycardia. This phenomenon appears to be derived from the important role of recovery of atrial excitability. In this study of these patients, the minimum echo time was equal (±20 msec) to the FRP of the atrium measured using the extrastimulus technique with a catheter positioned near the accessory pathway. This equality persisted despite change of pacing site and caused the shift of the upper limit of the echo zone to a shorter coupling interval during pacing with a catheter remote from the accessory pathway. Figure 4 depicts this phenomenon schematically. Pacing at point P, the time elapsed between the arrival of the atrial extrastimulus A and the first atrial echo A in the atrial end of the accessory pathway (P, A) is a + N + V (a = transatrial conduction time from stimulation site to the A-V node; N = A-V node conduction time; V represents the sum of conduction times of the His bundle, ventricle, and retrograde conduction by the accessory pathway). When stimulation is given at point P, the circuit time to point P is given by the expression b + N + V - c (where b is transatrial conduction time between the stimulation site and the accessory pathway; N and V are the same as in panel A). If the minimum circuit time required to produce an echo is the same regardless of pacing site, then additional delays must

![Figure 4. Effect of pacing site change on upper limit of echo zone in patients with Type III echo zone at both pacing sites. This is a schematic presentation of a heart with an accessory pathway in the left A-V groove. Pacing at P shifts the upper limit of the echo zone to shorter coupling intervals because additional A-V node delay (N) is required to compensate for transatrial conduction time (c). CS = coronary sinus; RA = right atrium.](image-url)
be achieved to compensate for the transatrial conduction time (c) incurred by pacing at point P₂. Incurred additional delay in the A-V node (N) by using shorter coupling intervals shifts the upper limit of the echo zone. A similar phenomenon has been previously reported in patients in whom accessory pathways in the left A-V groove conducted in the retrograde direction only. In contrast, Wellens et al. attributed this finding to greater concealed antegrade conduction into the accessory pathway while pacing at the site near the accessory pathway. They dismissed the role of atrial refractoriness probably because echo times were compared to the ERP of the atrium rather than the FRP of the atrium.

Since A-V node delay is an important factor in the initiation of an atrial echo using an accessory pathway, autonomic variation, change of pacing site, and drugs which alter A-V node refractoriness will have important further effects on echo zones. Denes et al. have presented preliminary results showing that propranolol increases the upper limit of the echo zone in those patients with accessory pathways which have exclusive retrograde conduction. The echo zone in patients with accessory pathways with exclusive retrograde conduction resembles the Type III echo zone described here.

Rosen reported one patient with an echo zone which resembled the Type III antegrade echo zone of this present study. He cited the presence of this type of antegrade echo zone as evidence that re-entry occurred within the A-V node. In contrast, Wellens and Durrer reported that the longest coupling interval accompanied by an echo was shorter than the antegrade ERP of the accessory pathway in six of 45 patients in whom re-entry utilized the accessory pathway for retrograde conduction by other criteria. Our results corroborate those of Wellens and Durrer.

Use of Serial Echo Zone Measurements To Select Drug Therapy

Since the demonstration that premature atrial depolarizations which were blocked in the accessory pathway could initiate reciprocating tachycardia, researchers have hoped that serial studies after drug administration would provide a useful guide to selection of drug therapy. The antegrade ERP of the accessory pathway and the range of the echo zone are easily measurable and could be applied as therapeutic guides. Wellens and Durrer, for example, studied the effect of intravenous ouabain in six patients. Four of the six patients had a reciprocating tachycardia, and using the criteria from this present study, all had Type II echo zones. Since ouabain decreased the antegrade ERP of the accessory pathway and increased the ERP of the A-V node, the range of the antegrade echo zone was invariably decreased. Ouabain and other digitalis compounds, it was inferred, were likely to be useful therapeutic agents in these four patients.

If one attempts to apply the same principles to quinidine and procainamide, which both tend to increase the antegrade ERP of the accessory pathway, the results are considerably more confusing. Mandel et al. and Wellens and Durrer showed that tachycardia could still be initiated by premature atrial depolarizations despite complete antegrade block in the accessory pathway. Moreover, in the study of Wellens and Durrer, the antegrade echo zone range was unchanged in some patients because the antegrade ERP of the accessory pathway and the lower limit of the echo zone both increased by similar increments. Mandel made similar observations, and additionally showed that some patients could have tachycardia induced by premature atrial depolarizations only after procainamide administration when the antegrade ERP of the accessory pathway had been increased more than the FRP of the atrium or the ERP of the A-V node. Sellers et al. studied patients at multiple cycle lengths after quinidine and procainamide administration and found that the echo zone range could be either increased, decreased, or unchanged by the drugs. Moreover, the echo zone range was increased at some cycle lengths and decreased at others in the same patient.

All these results are explainable by understanding the effect of these drugs on the upper and lower limits of the antegrade echo zone obtained with each refractory period determination. Since these individual limits are known to vary as pacing cycle length varies, it is not surprising that a drug can have many different effects on the range of the antegrade echo zone depending on that drug's relative effect on the measured upper and lower limits. Since the changes in these limits as pacing cycle length changes are not necessarily uniform, the variable effects of the drugs are expected.

This study has shown that the antegrade ERP of the accessory pathway is only one of several determinants of the antegrade echo zone. Moreover the antegrade echo zone has been shown to vary greatly in any single patient and is profoundly affected by pacing site, cycle length, and intraventricular conduction delay. Because of this variability, and because the antegrade echo zone contains no information about other spontaneous electrophysiologic events which initiate reciprocating tachycardia use of the antegrade echo zone to predict drug therapy has serious limitations. While the cycle length of the tachycardia induced by atrial extrastimuli may be shown to change during drug administration, demonstration of complete ventriculo-atrial conduction block by the simpler technique of ventricular pacing alone has more potential value. However, selection of the ideal therapeutic regimen — which is the minimum amount of medication producing the desired therapeutic effect — continues to be based on empirical decisions despite the important advances made using intracardiac electrophysiologic techniques.

Acknowledgment

The authors wish to express their gratitude to the many cardiology fellows who participated in these studies; to Laura Cook, R.N., and to Donald Kopp, M.D., the staff of the Electrophysiology Laboratory; to Jackie Kasell, electronics consultant; to Don Powell and David Huggett who prepared the illustrations; and to Bonnie Farmer and Ann Clayton who prepared the manuscript.

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RISK OF A-V BLOCK DURING SURGERY/Pastore et al. 677


The Risk of Advanced Heart Block in Surgical Patients with Right Bundle Branch Block and Left Axis Deviation

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SUMMARY The risk of advanced atrioventricular block during anesthesia was studied prospectively in 44 patients with right bundle branch block and left axis deviation who underwent a total of 52 operations over a 14 month period. All patients had continuous electrocardiographic monitoring throughout anesthesia induction, operation, and surgical recovery. Of the 52 operative procedures, 24 were done under general anesthesia, 11 under spinal, and 17 under local. The preoperative cardiac rhythms were atrial fibrillation in two patients, atrial tachycardia with block in one patient, atrial flutter in one patient, and sinus rhythm in the remaining patients. Temporary pacemakers were inserted preoperatively in six patients, usually because of PR interval prolongation on the preoperative electrocardiogram. There was only one episode of transient complete heart block in 51 of the 52 operative procedures. In two of the six patients with temporary pacemakers, significant pacer-related ventricular irritability occurred. This study indicates that temporary pacemaker insertion is rarely required in patients with chronic right bundle branch block and left axis deviation who require noncardiac surgery.

ALTHOUGH PATIENTS who develop complete heart block commonly have preceding complete right bundle branch block and left axis deviation on the electrocardiogram, the incidence of progression to complete heart block in prospective studies of patients with this form of bifascicular block appears to be less than 10%. Cardiologists at our hospital are frequently consulted on the question of standby cardiac pacemaker placement when patients with this electrocardiographic abnormality require surgery. The assumption has been that the stress of anesthetic induction and surgery might predispose patients with complete right bundle branch block and left axis deviation to advanced heart block.

We have recently followed prospectively through surgery a number of such patients in an attempt to determine the intraoperative risk of their developing advanced atrioventriculo-
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