

Ventriculo-atrial Conduction in Man

By BRUCE N. GOLDREYER, M.D., AND J. THOMAS BIGGER, JR., M.D.

SUMMARY

Transvenous electrodes were placed in the right atrium and ventricle of 50 patients with heart disease and arrhythmias. The ventricles were paced at several rates, each in excess of the sino-atrial rate. An atrial electrogram and surface electrocardiogram were recorded simultaneously in each patient; utilizing strict criteria, the incidence with which 1:1 ventriculo-atrial conduction (VAC) could be elicited in these patients was assessed. These patients were divided into two groups—26 patients with normal A-V conduction comprised group 1, and 24 patients with prolonged A-V conduction, group 2. VAC was demonstrated in 89% of group 1 patients but in only 8% of the group 2 patients. The difference in the incidence of 1:1 VAC between the two groups was statistically significant ($P < 0.01$). The presence or absence of 1:1 VAC could not be related to type of heart disease, medication, presence of acute myocardial infarction, or presence of intraventricular conduction defect.

Altering the paced ventricular rate or introducing stimulated ventricular premature depolarization in group 1 patients could produce any degree of VAC impairment. During such maneuvers, ventricular reciprocal beats (ventricular echoes) and ventricular reciprocal rhythms were observed.

Additional Indexing Words:

A-V block

Cardiac electrograms

Complete heart block

Ventricular echoes

Ventricular pacing

Reciprocal rhythm

THE bidirectional conduction capacity of the A-V conduction system has been demonstrated in many classes of animals.¹⁻⁵ Using specialized technics for P-wave recognition, V-A conduction has occasionally been demonstrated in patients with spontaneous ventricular premature contractions,⁶ spontaneous⁷ and catheter-induced ventricular tachycardia,^{8,9} complete A-V block during ventricular pacing,¹⁰⁻¹² normal A-V conduction, and severe sinus bradycardia, when ventricular pacing was employed.¹³

From the Departments of Medicine and Pharmacology, College of Physicians and Surgeons, Columbia University, New York, New York, and the Medical Service, The Presbyterian Hospital in the City of New York.

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Although these reports are sufficient to indicate that ventriculo-atrial conduction (VAC) may occur under a variety of circumstances, they do not fully indicate an incidence with which VAC might be expected under normal or abnormal physiologic conditions. It is difficult to interpret the meaning of such disparate reports as those of Samet and associates¹⁴ who found ventriculo-atrial conduction in 10 of 15 patients with complete A-V block, and Kistin and Landowne,⁶ who noted it in only 15 of 33 patients studied, none of whom had high-grade A-V block.

This report represents an attempt to assess the capacity of the human A-V conduction system (AVCS) to sustain 1:1 ventriculo-atrial conduction (1:1 VAC), by selecting controlled experimental conditions designed to maximize its occurrence and facilitate its recognition.

Patients were divided into two groups, one with normal (group 1), the other with prolonged A-V conduction time (group 2), so

that the relation between A-V conduction and the capacity for 1:1 VAC could be analyzed. A-V and V-A conduction times and refractory periods were compared, and during these studies single and multiple ventricular reciprocal beats were observed.

Methods

Fifty-six consecutive patients with a variety of cardiovascular diseases were admitted to our Cardiac Intensive Care Unit with arrhythmias and were referred to us for the placement of intracardiac electrodes required for the evaluation or therapy of these arrhythmias. In 50 patients the capacity for VAC was assessed. Six patients were not studied because extreme ventricular irritability was judged a contraindication to a period of fixed-rate ventricular pacing.

The initial placement of intracardiac electrodes was accomplished without moving the patient from his bed in the Unit. Using sterile technique and local procaine anesthesia, the left basilic vein was isolated and used for transvenous placement of a unipolar platinum-tipped electrode* in the outflow tract of the right ventricle. The unipolar electrogram obtained from the probe was used to identify its position. An electrode was inserted subcutaneously above the left clavicle as the anode of the pacing circuit.

A second identical electrode probe was then advanced through the same vein until the probe electrogram demonstrated large amplitude, predominantly negative P waves indicating a position high in the right atrium. The basilic vein was tightly ligated around the two probe wires to assure both hemostasis and stability of electrode position.

In 18 patients, fluoroscopy verified probe positions within the right ventricular outflow tract and at the junction of the superior vena cava and right atrium in close approximation to the sinoatrial node.

In several patients, the His bundle electrogram was identified. A catheter of our design, bearing 10 electrodes, was advanced via the saphenous vein under fluoroscopic control, until the catheter tip crossed the A-V valve and one set of electrodes bracketed the His bundle. The His bundle potential recorded from these electrodes was a sharp biphasic deflection lying within the P-R segment of the standard electrocardiogram.

In all patients, either the plug-in pacemaker module of a wall-mounted monitoring system or an external battery-powered pacemaker was used

to deliver cathodal stimuli 1.8 msec in duration and 3 to 5 ma in amplitude to the right ventricle at a variety of constant rates. The atrial electrogram and a surface electrocardiographic lead were simultaneously monitored on a switched-beam oscilloscope and recorded on an FM magnetic tape recorder. Taped records were later transferred to photographic paper and analyzed.

Ventricular pacing rates 10 to 15% in excess of the existing sinus rate were employed. This minimized competitive utilization of the A-V conduction system (AVCS) by impulses of sinus and ventricular origin and simplified the recognition of V-A conduction by providing continuous 1:1 VAC.

Criteria established for the conclusive demonstration of 1:1 VAC during ventricular pacing were: (1) identical ventricular and atrial cycle lengths (CL) at each of several paced rates exceeding the sinus rate; (2) a constant V-A conduction time at each rate; and (3) a pause prior to the resumption of sinus rhythm when ventricular pacing was abruptly interrupted. Examples illustrating fulfillment of these criteria are presented in figures 1 and 2.

A-V conduction time measured during sinus rhythm (the P-R interval) was compared to the V-A conduction time during established 1:1 VAC at comparable rates. In six patients with A-V dissociation or junctional tissue rhythms with continuous atrial capture, the P-R interval was *not* a measure of A-V conduction time. One-to-one A-V conduction was produced with atrial pacing, and A-V conduction time was measured. The capacity for 1:1 VAC was then assessed as previously described.

The patients were divided into two groups based on A-V conduction time.

Group 1

Group 1 was composed of 26 patients with normal A-V conduction times ($P-R < 200$ msec). Clinical indications for electrode placement included the following: severe sinus bradycardia frequently accompanied by A-V dissociation (sick sinus syndrome¹⁵) in 15 patients; marked left axis deviation and right bundle-branch block complicating an acute anteroapical myocardial infarction in five patients; and junctional tissue rhythms complicating acute inferior myocardial infarction in two patients.

Group 2

Group 2 was composed of 24 patients with A-V conduction impairment. Four patients had incomplete A-V block ($P-R$ interval > 200 msec); eight patients had intermittent A-V conduction, such as Wenckebach periods or 2:1 A-V block; 12 had

*Davis & Geck Division of the American Cyanamid Company, Pearl River, N. Y.

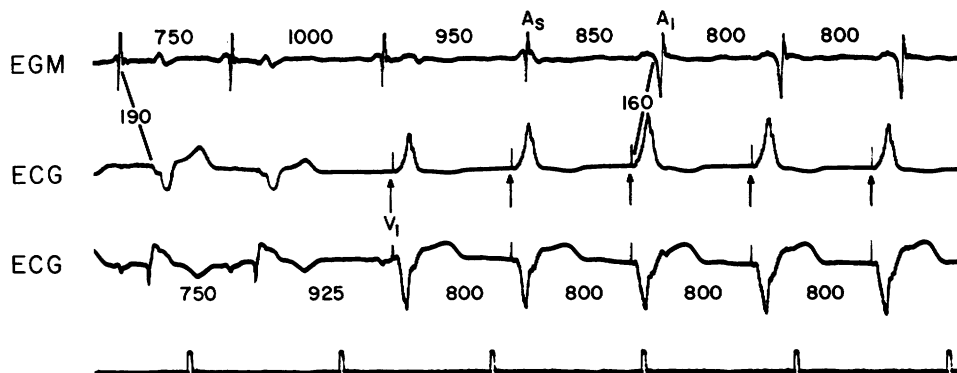


Figure 1

Criteria for 1:1 VAC at the initiation of ventricular pacing. Tracings from top to bottom are: atrial electrogram (EGM), two surface electrocardiographic leads (ECG), and time markers at 1-sec intervals. Time intervals in msec between successive depolarizations are indicated below the lower ECG.

This patient had marked sinus arrhythmia. Note the large variation in the first three atrial cycles (750, 1,000, 950 msec) which were of sinus origin. The last atrial depolarization of sinus origin is labeled A_s . A-V conduction time during sinus rhythm is indicated on the first diagonal (190 msec). After the second conducted sinus beat, ventricular pacing is begun. Stimuli are indicated by arrows, and the first ventricular response by V_1 . The ventricle is thereafter driven at a constant rate of 75/min (CL, 800 msec). Competition with sinus rhythm prevents VAC until the third paced beat. The first atrial depolarization resulting from VAC is labeled A_1 . From this point, atrial and ventricular cycle lengths are precisely equal; V-A conduction time is constant (160 msec). Atrial depolarizations are not seen on either surface ECG lead during 1:1 VAC.

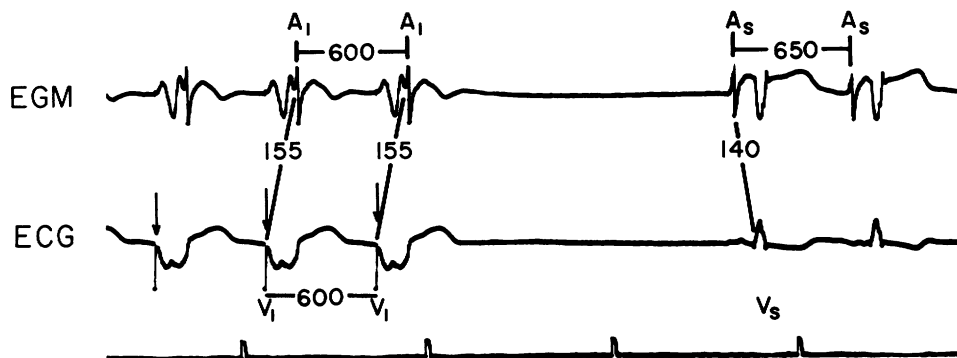


Figure 2

Criterion for 1:1 VAC at the termination of ventricular pacing. Recorded from top to bottom are unipolar atrial electrogram (EGM), surface electrocardiogram (ECG), and time markers at 1-sec intervals. To the left, during ventricular pacing at a rate of 100/min (CL, 600 msec), stimuli are indicated by arrows and ventricular responses by V_1 . One-to-one VAC was assumed because the atrial and ventricular cycle lengths were identical, and V-A conduction time was constant (155 msec). Atrial depolarizations resulting from VAC are labeled A_1 . After the third paced beat, electrical stimuli are abruptly terminated. A 1.8-sec pause results before the first atrial depolarization of sinus origin occurs (A_s); A-V conduction time was normal (A_s - V_s , 140 msec). The long pause, after ventricular pacing was discontinued and before sinus rhythm resumed, confirmed prior 1:1 VAC.

complete A-V block. In all patients, intracardiac electrodes were placed prophylactically, and therapeutic ventricular pacing was instituted (in the demand mode) if the ventricular rate fell below 60 beats/min.

In the patients with complete A-V block, the total duration of A-V block prior to the day of study varied. Six patients had complete A-V block documented for periods of from 2 mo to 16 years. In four patients, complete A-V block followed the acute myocardial infarction which led to the hospital admission, and in two patients the duration of heart block prior to hospitalization was unknown.

Twenty-three of the group 1 patients (no A-V block) demonstrated 1:1 VAC. In 11 of these, the A-V and V-A refractory periods were measured using an extra stimulus method.¹⁶ To measure the V-A refractory periods, rectangular cathodal stimuli, 1.8 msec in duration, provided by a series of wave form and pulse generators, were used to pace the ventricles at a rate in excess of the sinus rate. An electronic counting device allowed a premature test stimulus, one and one-half times threshold, to be delivered during every 10th drive cycle. The next drive stimulus was omitted to prevent rapidly successive stimuli from entering the ventricle. The test stimulus was made progressively more premature by 5 to 10-msec decrements.

Although this stimulation sequence was not specifically designed to elicit ventricular echoes, the pause following the test stimuli provided the opportunity to observe such reentrant activity when it occurred.

Patient medications were prescribed by their physicians and not altered for the purposes of this study. The features of the study protocol which

departed from routine clinical procedure were (1) the insertion of a second intracardiac electrode and (2) a period of ventricular pacing in those patients for whom only atrial pacing would ordinarily have been employed. No complications arose from either the use of two flexible intracardiac electrodes or the brief period of ventricular pacing required to assess the capacity of VAC.

Results

The Capacity for V-A Conduction

Group 1 consisted of 26 patients with normal A-V conduction times. Group 2 consisted of 24 patients with all degrees of A-V block and may be divided into three subgroups according to the degree of A-V block present at the time of study (table 1). The two groups were not significantly different ($P > 0.05$) in terms of sex, type of heart disease, presence of congestive heart failure, or presence of acute myocardial infarction. There was no significant difference in terms of serum potassium, sodium, or urea nitrogen (Student's *t*-test between means). Neither was the incidence of electrocardiographic features such as marked left axis deviation or intraventricular conduction defects significantly different.

The age of group 2 patients was higher (71 ± 8 years) than that of group 1 patients (59 ± 15 years). Group 2 patients had a higher sinus rate (82 ± 13) than group 1 (73 ± 15), but this difference in sinus rate may largely be ascribed to the fact that in many of the patients with normal A-V conduction, sinus bradycardia was the indication for which cardiac electrodes had been placed.

Table 1

*Group 2 (Abnormal A-V Conduction Time, P-R > 200 msec): A-V Conduction Impairment**

	1:1 V-A conduction	No V-A conduction
Prolonged A-V conduction	1	3
Intermittent A-V conduction†	0	8
No A-V conduction	1	11
Total	2	22

*Two of these patients demonstrated 1:1 VAC — one had only a prolonged P-R interval; the other showed no A-V conduction.

†Seven patients with A-V Wenckebach cycles or 2:1 A-V block and one patient with intermittent Mobitz II complete A-V block.

Table 2

*Groups Divided According to A-V Conduction Time**

	Group 1 P-R < 200	Group 2 P-R > 200
1:1 V-A conduction	23	2
No V-A conduction	3	22
Total	26	24

*The observed difference in incidence is statistically significant ($P < 0.01$).

The capacity of the A-V conduction system to sustain 1:1 VAC was assessed in every patient. Table 2 shows that 1:1 VAC was produced in 89% of the patients with normal A-V conduction times. Only 8% of the patients with any degree of A-V conduction impairment demonstrated 1:1 VAC under these same conditions (two of 24 in group 2). One of the two patients in whom 1:1 VAC was found merely had a prolonged P-R interval (P-R, 260 msec); in the second, complete A-V block was present. A higher incidence of VAC was not found in patients with lesser degrees of A-V block. The difference in the incidence of 1:1 VAC between group 1 and group 2 patients was significant when analyzed by the χ^2 method ($P < 0.01$).

V-A Compared to A-V Conduction in Patients with Bidirectional Conduction Capacity

In group 1 patients who demonstrated 1:1 VAC, V-A conduction time was measured and compared to A-V conduction time at a similar rate. V-A conduction time was significantly longer than A-V conduction in all but two of these patients (fig. 3).

In 11 group 1 patients antegrade and retrograde functional and effective refractory periods were measured and compared at the same drive rates. These will be reported separately, but the V-A effective refractory period* was longer than the A-V effective refractory period at similar drive cycle lengths.¹⁶ The fact that, at any given rate, V-A conduction was longer than A-V conduction, and the V-A effective refractory period was longer than the A-V effective refractory period, probably contributed to the ease with which varying patterns of retrograde conduction impairment could be produced. Any and all degrees of V-A block could be produced by increasing the ventricular pacing rate or introducing ventricular test stimuli during the relative V-A refractory period (fig. 4).

*The V-A effective refractory period is defined as the shortest interval between drive and test ventricular responses at which the test response conducts to the atrium.

At rates lower than those at which V-A block occurred, group 1 patients showed conduction in both directions through the AVCS, providing a setting for ventricular reentry.

Single ventricular echoes were observed in three of 11 patients (27%) during the measurement of the V-A refractory periods (fig. 5). In one patient, brief episodes of ventricular reciprocating rhythm occurred (fig. 6).

Another patient had an unusually long interval during which the introduction of a premature ventricular stimulus would result in ventricular echoes. The relationship between V-A and A-V conduction times during ventricular echoes (fig. 7) supports the contention by Kistin¹⁷ that antegrade and retrograde conduction times are reciprocally related during ventricular reentry.

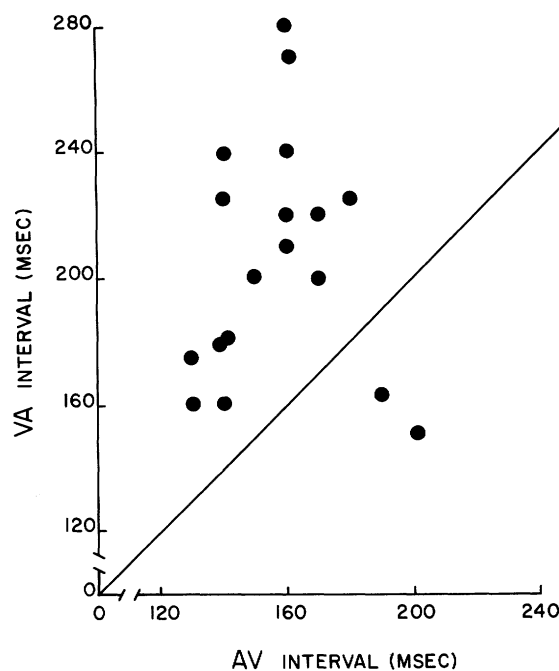


Figure 3

VA compared to AV conduction time. In group 1 patients (normal AV conduction) the VA interval, or conduction time, in msec, is plotted on the ordinate as a function of the AV interval in msec on the abscissa. If VA and AV conduction times were identical, points would fall on the diagonal line. In all but two patients with normal AV conduction, VA conduction was longer than AV conduction.

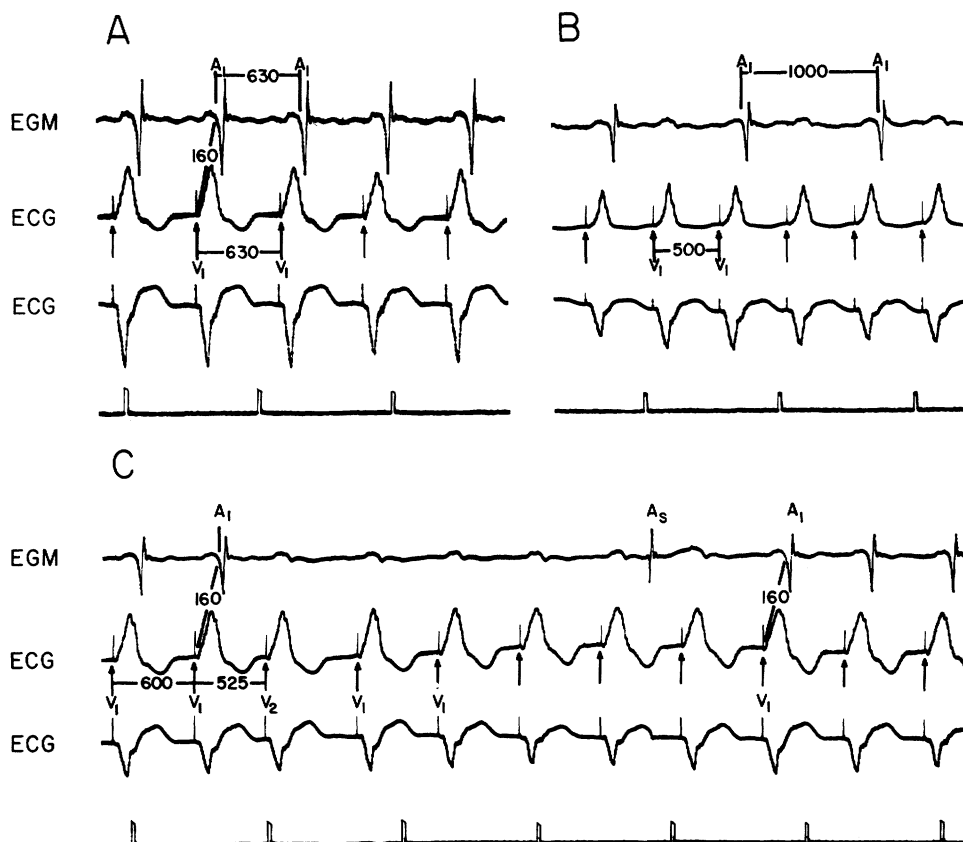


Figure 4

Retrograde block produced in a patient with 1:1 VAC by increasing the ventricular paced rate and by stimulating the ventricle prematurely. In each panel the recordings shown from top to bottom are atrial electrogram (EGM), two surface electrocardiograms (ECG), and time markers at 1-sec intervals. In each panel, ventricular stimuli are indicated by arrows. Ventricular responses to drive stimuli are labeled V_1 ; conducted atrial responses are labeled A_1 .

In panel A, ventricular pacing at a rate of 95/min (CL, 630 msec) results in 1:1 VAC. V-A conduction time (V_1 - A_1) is 160 msec.

In panel B, the ventricular pacing rate is increased to 120/min (CL, 500 msec). At this rate, the interval between atrial depolarizations (1000 msec) is exactly twice that between ventricular responses; this represents 2:1 VAC.

In panel C, the ventricles are paced at a rate of 100/min (CL, 600 msec). At this rate, 1:1 VAC is again present and V-A conduction time is 160 msec. A premature stimulus is introduced into the pacing sequence and the ventricular response V_2 results (V_1 - V_2 , 525 msec). V_2 fails to conduct to the atrium since it falls within the V-A effective refractory period. Similarly, five subsequent ventricular paced beats fail to conduct to the atrium (repetitive concealed conduction). After 3.2 sec, the sino-atrial nodal escapes and depolarizes the atrium (A_s). Although this sinus impulse fails to propagate to the ventricles, it blocks the next retrograde depolarization low in the A-V conduction system allowing subsequent driven ventricular responses to conduct to the atrium, and 1:1 VAC resumes.

In patients with normal antegrade conduction (group 1), we demonstrated that (1) the A-V conduction system has a bidirectional conduction capacity; (2) V-A conduction is

usually longer than A-V conduction; (3) V-A refractoriness exceeds A-V refractoriness; and (4) ventricular reentrant beats may be produced with relative ease.

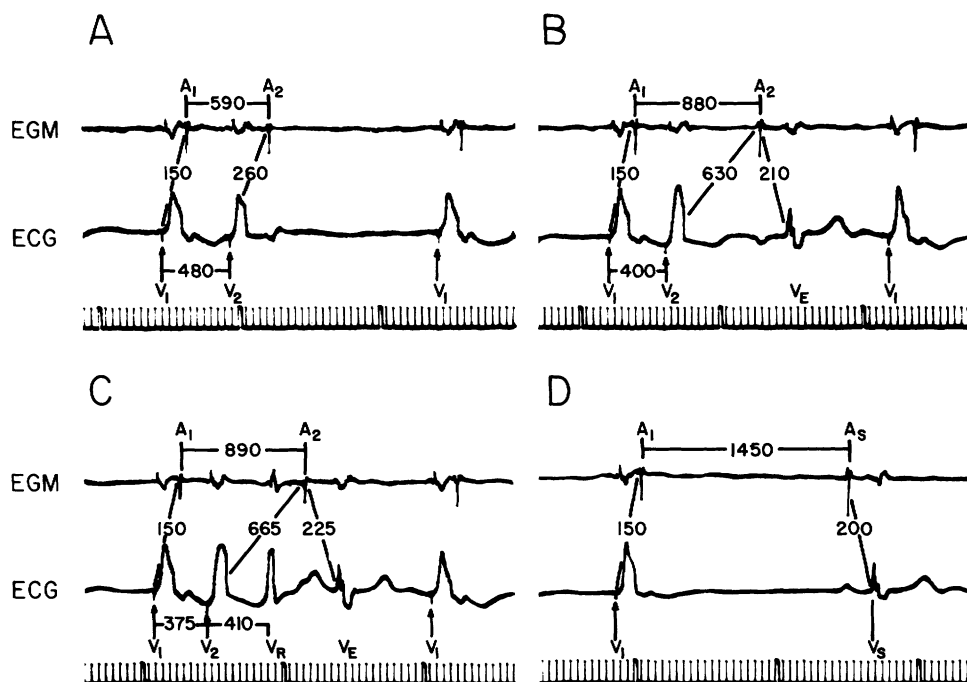


Figure 5

Ventricular echoes elicited during measurement of the ventriculo-atrial refractory periods by the extra stimulus method. In each panel recorded from top to bottom are atrial electrogram (EGM), surface electrocardiogram (ECG), and time markers at 50-msec intervals. Stimuli are indicated by arrows; the intervals in msec between consecutive atrial and ventricular depolarizations are indicated on the horizontal lines. V-A and A-V conduction times in msec are indicated on diagonal lines connecting conducted atrial and ventricular depolarizations. The ventricles are being paced at a rate of 60/min (CL, 1000 msec). Ventricular responses to the driven stimuli are labeled V_1 . Atrial depolarizations conducted from V_1 are labeled A_1 . V-A conduction time (V_1 - A_1) is 150 msec. During each 10th drive cycle, a premature ventricular stimulus elicits the ventricular response, V_2 . Atrial depolarizations conducted from V_2 are labeled A_2 . The following ventricular drive stimulus is omitted to avoid introducing rapidly successive stimuli. In panels A to C, test cycles are shown as V_2 is made progressively more premature.

In panel A, V_2 , occurring 480 msec after V_1 (V_1 - V_2 , 480 msec) enters the AVCS during its relative refractory period, V-A conduction delay results (V_2 - A_2 , 260 msec).

In panel B, V_1 - V_2 is shortened to 400 msec. V-A conduction time is prolonged markedly (V_2 - A_2 , 630 msec), and a ventricular echo, V_E , results.

In panel C, V_2 is more premature (V_1 - V_2 , 375 msec) and V-A conduction delay increases further (V_2 - A_2 , 665 msec). At this V_1 - V_2 interval, a reentrant beat, V_R , was consistently produced 410 msec following V_2 . This reentrant beat probably utilized the lower portion (intraventricular) of the AVCS, since the ventricular echo, V_E , still occurred. Note the prolongation of antegrade conduction during the ventricular echo. This is probably due to the simultaneously occurring ventricular reentrant beat leaving the AVCS slightly more refractory to antegrade conduction.

In panel D, V_2 is omitted and the ventricular stimulator turned off. Sino-atrial escape (A_s) occurs after 1.45 sec. The prolonged sinus escape time demonstrates that the A_s shown in the prior panels do not result from sino-atrial depolarization of the atrium.

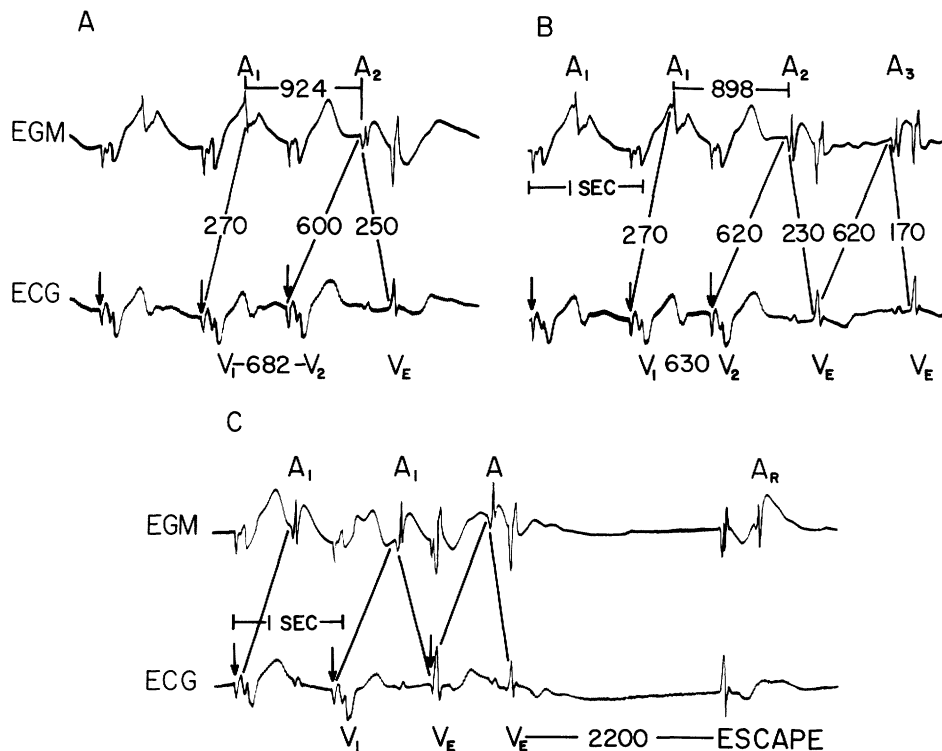


Figure 6

Ventricular reciprocal rhythm during ventricular pacing in a patient with 1:1 VAC. In each panel atrial electrogram (EGM) and surface electrocardiogram (ECG) are shown, and the ventricle is paced at a CL of 800 msec. Stimuli are indicated by arrows. Ventricular responses to drive stimuli are labeled V_1 . During each 10th drive cycle, a premature ventricular stimulus elicits V_2 . Intervals in msec between consecutive atrial and ventricular depolarizations are indicated on the diagonal lines connecting atrial and ventricular depolarizations. During 1:1 VAC, V-A conduction time (V_1 - A_1) is 270 msec.

In panel A, a premature ventricular response (V_1 - V_2 , 682 msec) enters the AVCS during its relative refractory period. Retrograde conduction delay (V_2 - A_2 , 600 msec) is sufficient to allow the impulse to return to the ventricle (ventricular echo, V_E).

In panel B, V_2 is more premature (V_1 - V_2 , 630 msec), retrograde conduction delay is more pronounced (V_2 - A_2 , 620 msec), and two echo cycles of ventricular reciprocal rhythm result.

Panel C demonstrates that these rhythms do not result from sinus interference. During a period of increasing retrograde conduction delay (retrograde Wenckebach phenomenon) the stimulator is turned off. As in panel B, two ventricular echoes result (V_E). Then, 2,200 msec elapse before an escape beat occurs. In this patient, the sino-atrial node was so depressed that the escape beat originates in the His bundle. This beat also conducts retrograde to the atrium (A_R).

Discussion

Ventriculo-atrial conduction is difficult to detect in the standard electrocardiogram because the high amplitude QRS complexes obscure the retrograde P waves. Esophageal recordings⁶ or intra-atrial electrograms¹⁸ provide accurate means for P-wave detection.

Using these technics many attempts have been made to determine the frequency with which VAC occurs. In the first of these, Kistin and Landowne⁶ demonstrated VAC in 15 of 33 patients with frequent ventricular premature contractions. This incidence (45%), however, probably did not reflect all those individ-

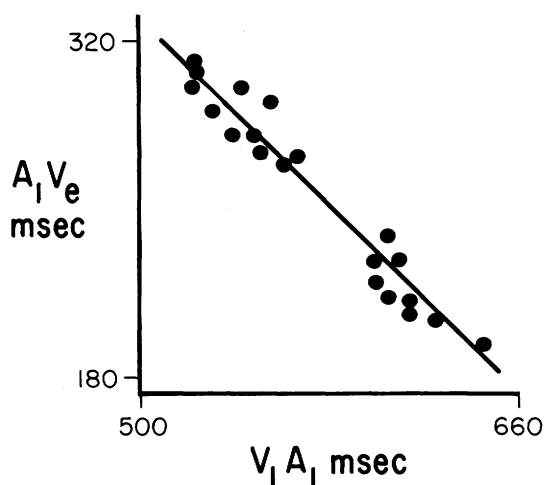


Figure 7

During measurement of the V-A refractory periods, 20 ventricular test responses resulted in ventricular echoes in one patient. The graph shows the antegrade conduction time (A_1V_e) of the echo beats plotted as a function of the retrograde conduction time (V_1A_1) of the ventricular test responses which initiated ventricular reentry. The correlation coefficient calculated for these points is -0.94 . The reciprocal relationship between antegrade and retrograde conduction time during ventricular reentry is significant ($P < 0.01$); as retrograde conduction time increases, antegrade conduction time decreases.

uals with the capacity for VAC. As these investigators recognized, the relative prematurity of any ventricular premature contraction is a factor in determining whether VAC will occur. After a propagated sinus beat, the A-V conducting system is first absolutely and then relatively refractory to retrograde depolarization¹⁹ (fig. 8).

When the ventricular rate exceeds the sinoatrial rate for several beats, competitive utilization of the AVCS should be eliminated and the chances for VAC enhanced. Kistin analyzed the incidence of VAC during brief episodes of ventricular tachycardia⁷ and Masumi and associates⁸ did likewise during rapid (125 to 170) ventricular pacing. Their respective incidences of 48 and 27% probably still fail to reflect the number of patients with a capacity for VAC, because the rapid ventricular rates cause VAC to fail in those cases in

which the ventricular cycle length is shorter than the V-A effective refractory period.

Ventricular pacing at rates only slightly in excess of the sinus rate should maximize the incidence of VAC. Lister's group²⁰ reported that pacing the ventricles at rates faster than the spontaneous atrial rate resulted in 1:1 VAC in six of seven patients during open heart surgery. Samet and co-workers,¹⁸ using atrially synchronized ventricular pacing, found that, at an optimal atrioventricular relationship, retrograde P waves could be detected in 29 of 31 patients (94%).

The assumption that separate pathways are involved in V-A as opposed to A-V conduction^{3, 17, 19, 21} may have obscured the relationship between A-V and V-A conduction. V-A conduction was assessed without specific consideration of whether A-V conduction was normal. In Kistin and Landowne's original report,⁶ each patient with VAC had a P-R interval of less than 0.20 sec, but A-V conduction times of patients without VAC are not given. Similar considerations apply to other studies.^{7, 8}

In the present study, therefore, the capacity for VAC rather than its incidence was related to A-V conduction. For this purpose the ventricles were paced at rates only slightly in excess of the sinus rate, and the patients were grouped on the basis of A-V conduction time. We observed a large discrepancy in the capacity for 1:1 VAC between groups of patients with (group 2) and without (group 1) A-V conduction impairment.

Further evidence that A-V and V-A conduction are functionally related is provided by a comparison of antegrade and retrograde conduction times. The illustrations of most investigators^{13, 18, 20} show that V-A conduction is generally longer than A-V conduction although these conduction times are not specifically given. In all but two of our 23 group 1 patients with 1:1 VAC, V-A conduction time is significantly longer than A-V conduction time (fig. 3).

A comparison of effective refractory periods (A-V and V-A) in 11 of our patients with 1:1

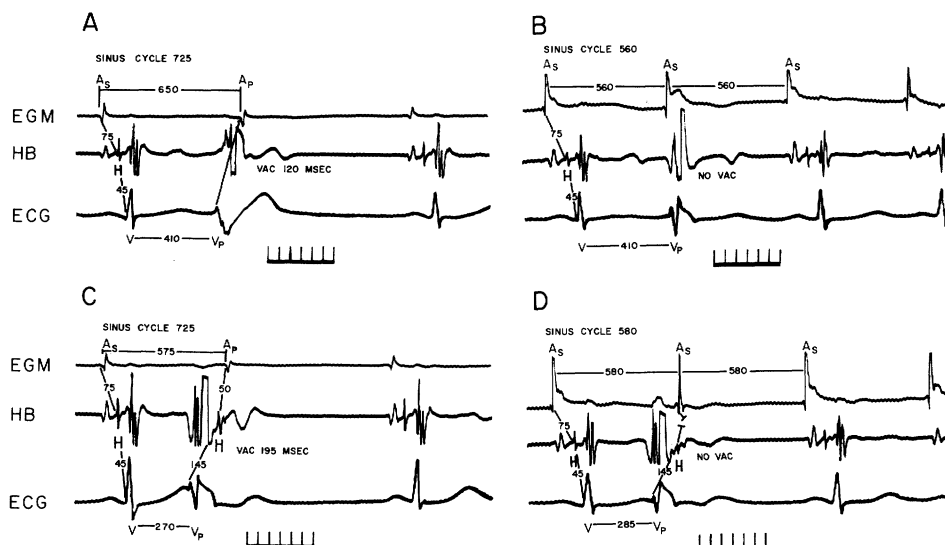


Figure 8

Four panels selected from the records of a single patient demonstrate that VAC of spontaneous ventricular premature systoles (V_p) is a function both of the prematurity of the V_p and the length of the sinus cycle. In each panel recorded from top to bottom are atrial electrogram (EGM), His bundle electrogram (HB), and surface electrocardiogram (ECG). The atrial cycle length (CL) during sinus rhythm is given above, and time markers at 50-msec intervals are given below each panel. The first beat in each panel is of sinus origin; atrial depolarization (A_s), His bundle depolarization (H), and ventricular depolarization (V) are labeled. A-V conduction time (120 msec) is composed of an A_s -H interval (75 msec) and an H-V interval (45 msec). These two intervals represent consecutive conduction delay across the A-V node, and within the ventricular specialized conducting system. A-V conduction time remains constant at the two sinus rates depicted.

In Panel A, at the slower sinus rate (CL, 725 msec) a spontaneous ventricular systole (V_p) occurs 410 msec after the ventricular depolarization of sinus origin (V - V_p , 410 msec). V-A conduction results in the premature atrial depolarization, A_p . V-A conduction time is 120 msec. During VAC, with this short V-A conduction time, depolarization of the His bundle is obscured by the ventricular depolarization.

In panel B, a similarly timed V_p (V - V_p , 410 msec) occurring when the sinus rate is faster (CL, 560 msec) fails to result in VAC. The atrial CL is uninterrupted by this V_p since the sino-atrial node activates the atrium prior to the expected time of arrival of the impulse proceeding retrograde through the AVCS. Impulses of atrial and ventricular origin collide within the AVCS and conduction is blocked in both directions.

In panel C, at the slower sinus rate (CL, 725 msec) a very early V_p (V - V_p , 270 msec) again results in VAC. The prolonged V-A conduction time (195 msec) results from V_p occurring earlier in the V-A relative refractory period. This retrograde conduction delay is subdivided by the HB electrogram. The major conduction delay occurs within the ventricular specialized conducting system (V_p -H, 145 msec); retrograde conduction across the A-V node is relatively rapid (H- A_p , 50 msec).

In panel D, although V_p is similarly premature to the one demonstrated in panel C (V - V_p , 285 msec), VAC fails because of the faster sinus rate (CL, 580 msec). The same 145-msec conduction delay is encountered before the impulse arrives at the His bundle, but at this sinus cycle length, the atrium is depolarized by the sino-atrial node prior to the expected arrival time of the retrograde impulse.

V-A conduction showed that the A-V conduction system is more refractory to retrograde depolarization. Two facts help to explain why

1:1 VAC is rare in the presence of impaired A-V conduction: (1) In hearts with normal A-V conduction retrograde conduction times are

longer than antegrade conduction times; and (2) at any given heart rate the AVCS is more refractory to retrograde than antegrade depolarization.

Isolated cases of V-A conduction in the presence of A-V block were reported by Wolferth and McMillan,²² and the subject was critically reviewed in 1944 by Winternitz and Langendorf.²³ A few recent case reports of V-A conduction in the presence of complete A-V block may be found.^{12, 24-26} Utilizing the intra-atrial electrogram, Samet's group,¹⁴ however, found VAC in 10 of 15 patients with complete A-V block.

Variations between different studies in the incidence of VAC when A-V block is present may be caused by sampling small numbers of patients in a highly varied population; however, the present study represents a moderately large consecutive patient sample in whom the capacity for 1:1 VAC was specifically assessed. Only two of 24 patients with any degree of A-V block and only one of 12 patients with complete A-V block demonstrated VAC. Our results seem to indicate that although VAC may occur in patients with impaired (or even absent) A-V conduction, it is uncommon.

A clear distinction also exists between A-V block and either interference dissociation or A-V junctional rhythm with constant retrograde depolarization of the atrium. In our six patients with these conditions, atrial pacing at rates faster than any endogenous pacemaker resulted in normal A-V conduction times. In addition, all then demonstrated 1:1 VAC. The AVCS exhibited normal bidirectional conduction capabilities despite the ectopic A-V junctional pacemaker.

Ventricular reciprocal beats (ventricular echoes) result when a beat originating in the ventricle traverses the entire AVCS in a retrograde direction and then conducts (antegrade) to the chamber of its origin. The phenomenon requires a sufficient prolongation of V-A conduction following the initiating beat, to allow subsequent A-V conduction.^{27, 28} Cases of reciprocal rhythm initiated by ventricular premature contractions have been

reported.^{21, 29, 30} With the recent employment of ventricular pacemakers in the treatment of bradycardias resulting from mechanisms other than A-V block, two recent reports of reciprocal beating have appeared.^{13, 31}

The introduction of early ventricular test responses during measurement of the V-A refractory periods in 11 of our patients with 1:1 VAC provided an ideal opportunity for the observation of ventricular echoes. Test responses early in the V-A relative refractory period produced the prolonged V-A conduction necessary to produce ventricular echoes (figs. 5 and 6) in three of these 11 patients.

Scherf³ observed that "the PR interval of the return extrasystole became shorter as the length of the preceding RP interval became longer" and suggested that "at least part of the pathway was used twice by the stimulus." Although other authors have argued that reentrant activity results from two independent pathways through the A-V conduction system, observations on one of our patients supports Scherf's contention. In this patient ventricular echoes were elicited over a wide range of test intervals. In figure 7, antegrade conduction of the reentrant beat (A_1-V_E) is plotted as a function of the retrograde conduction time V_1-A_1 of the beat initiating ventricular reentry. The relationship demonstrated is linear and reciprocal ($n=11$, $r=0.915$, $P<0.001$). This observation agrees with Scherf's hypothesis that antegrade and retrograde pathways are not totally independent.

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