CASE REPORTS

Ventricular Paired Pacing to Control Rapid Ventricular Heart Rate Following Open Heart Surgery


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SUMMARY An unusual case is presented in which an atrioventricular (A-V) junctional tachycardia at a rate of 285 beats/min developed in the immediate postoperative period following surgical repair (a Mustard procedure) of transposition of the great vessels in a four-month-old infant. With that heart rate the systolic blood pressure became 35-40 mm Hg and urinary output ceased. Ventricular paired pacing was employed successfully to halve the mechanically effective ventricular rate. This resulted in a clinically effective blood pressure and return of normal urinary output. The clinical course of the A-V junctional tachycardia, plus its response to several interventions, suggested that the mechanism of the A-V junctional tachycardia was automatic rather than re-entrant. The study demonstrates that ventricular paired pacing for the control of cardiac arrhythmias is a useful clinical technique in selected cases.

VENTRICULAR PAIRED PACING and ventricular coupled pacing enjoyed a vogue about one decade ago. As summarized previously, their primary application was in the study of postextrasyostolic potentiation of ventricular contraction which followed the introduction of extrastyostosis. The clinical application of these techniques in an effort to improve cardiac output in patients with severe heart failure proved largely disappointing. However, although little emphasized at that time, the application of this technique in the treatment of tachycardia, particularly tachycardia not amenable to the more usual modes of therapy, clearly was recognized. Over the years there have been scattered case reports of the use of this technique to treat recalcitrant tachycardias, although it has been applied to a notable degree (133 cases) in eastern Europe.

We have had the opportunity to utilize the technique of ventricular paired pacing to treat an atrioventricular (A-V) junctional tachycardia which became manifest at a rate of 285 beats/min in a four-month-old child following open-heart surgical repair of transposition of the great vessels. This case is unique for several reasons. First, the very rapid A-V junctional rhythm most likely was an automatic tachycardia. Second, it emphasizes that ventricular paired pacing may be useful in the treatment of selected cardiac arrhythmias, particularly in children. Further, it suggests this technique may be useful in improving cardiac output in selected patients who manifest inadequate cardiac output following surgical repair of various congenital heart defects.

Case Report

The patient was a four-month-old, 5.6 kg male admitted to the Columbia-Presbyterian Medical Center on October 12, 1971, because of cyanosis which had increased since birth. On admission, the patient's hemoglobin was 19.7 g/100 ml. Cardiac catheterization demonstrated D-transposition of the great vessels with an intact ventricular septum and normal left ventricular pressure. A small patent ductus arteriosus was demonstrated by angiography. Inadequate arterial-venous mixing, as evidenced by an arterial oxygen saturation of about 40-50%, was provided by the ductus arteriosus and a small interatrial communication which could not be enlarged by balloon septostomy. Therefore, it was elected to correct the cardiac lesion during open-heart surgery by interatrially redirecting pulmonary and systemic venous return to the physiologically appropriate ventricle (the Mustard procedure). A normal sinus rhythm was documented prior to surgery.

Operative Course

A Mustard operation was carried out in the usual manner. During the period of cardiopulmonary bypass and prior to performing the atrial septectomy, the course of the Hisbundle was electrophysiologically delineated using techniques previously described. However, during the placement of the interatrial baffle, technical problems made the placement
of sutures in the region of the A-V node and proximal His bundle unavoidable. Upon termination of cardiopulmonary bypass, the electrocardiogram demonstrated A-V dissociation with a high degree of A-V block. The rate of the A-V junctional rhythm was regular at 140 beats/min and the atrial rate was 80 beats/min. The rate of the A-V junctional tachycardia gradually increased such that at the end of the surgical procedure, the ventricular rate was 180 beats/min with intermittent retrograde conduction to the atria. Atrial pacing at rates in excess of the ventricular rate was unsuccessful in producing antegrade A-V conduction or in influencing the A-V junctional tachycardia in any way. Prior to closure of the chest incision, two Teflon-coated stainless steel wire electrodes were implanted in the right atrial epicardium and one in the right ventricular epicardium and the distal ends brought out through the anterior chest wall for postoperative diagnostic and therapeutic use.\textsuperscript{22}

**Postoperative Course**

By the time the patient arrived in the open-heart recovery room, the A-V junctional tachycardia had increased to a rate of 220 beats/min with retrograde Wenckebach-type conduction to the atria (fig. 1A). Sixty minutes later, the rate had increased to 240 beats/min and 90 minutes later, the rate had increased to 285 beats/min with 2:1 retrograde conduction to the atria (fig. 1B). The morphology of the QRS complex in the ECG leads was the same at these rapid rates when compared with that during the previous slower rates. Of note, the beat-to-beat cycle length of the arrhythmia at these several rates varied somewhat (figs. 1A and 1B).

After the ventricular rate became 285 beats/min, the blood pressure gradually fell to a systolic pressure of 35–40 mm Hg and there was a marked decrease and finally complete cessation of urinary output. Over the next three hours, various interventions were ineffective in influencing either the A-V junctional tachycardia, systemic blood pressure, or urinary output. These therapeutic measures included: intravenous infusion of 7 μg of isoproterenol over 7 minutes; several trials of rapid atrial pacing at rates greater than 300 beats/min; intravenous digitalization; intravenous administration of lidocaine (5 mg) and procaine amide (15 mg); intravenous administration of mannitol (6 cc) and furosemide (6 mg). The laboratory data reflecting the patient's acid-base status at this time were: blood pH 7.4; Pco, 26 mm Hg; Po, 122 mm Hg; bicarbonate 22 mEq/L; O2 saturation 98%; base excess –2. The serum potassium was slightly elevated at 6.4 mEq/L. Six hours following surgery, the patient's condition appeared desperate. Therefore, to effectively suppress the A-V junctional tachycardia and to improve cardiac output, ventricular paired pacing was initiated. Using a Medictronic 5837 R wave-coupled pacemaker, the basic driving stimulus was delivered at an interval (S\textsubscript{1}-S\textsubscript{2}) of 400 msec. The premature ventricular stimulus (S\textsubscript{p}) was introduced initially at an interval (S\textsubscript{1}-S\textsubscript{2}) of 200 msec after the basic (S\textsubscript{1}) driving stimulus, i.e., midway through the S\textsubscript{1}-S\textsubscript{2} interval. This permitted ventricular capture at a paced rate of 300 beats/min. Abrupt termination of rapid ventricular pacing at this rate (300 beats/min) failed to interrupt or change the spontaneous A-V junctional tachycardia. Therefore, ventricular paired pacing at an S\textsubscript{1}-S\textsubscript{p} interval of 400 msec with an S\textsubscript{2} of 200 msec was resumed. The S\textsubscript{1}-S\textsubscript{2} interval was then decreased. Initially, at an interval between 170–180 msec, the S\textsubscript{2} interval was short enough that the S\textsubscript{2} stimulus produced an electrical response without an effective mechanical response, thereby halving the effective mechanical heart rate (fig. 2). When the S\textsubscript{1}-S\textsubscript{2} interval was decreased below 170 msec, the ventricular paired pacing became ineffective because the A-V junctional tachycardia escaped. However, ventricular paired pacing at an S\textsubscript{1}-S\textsubscript{2} interval of 400 msec and an S\textsubscript{2} interval between 170–180 msec proved incompletely effective, as the A-V junctional tachycardia became manifest periodically. Empirically, we discovered that ventricular paired pacing at an S\textsubscript{1}-S\textsubscript{2} interval of 385 msec and an S\textsubscript{2} interval of 180 msec (fig. 3) completely suppressed the underlying arrhythmia. In retrospect, effective ventricular paired pacing at the latter intervals can be understood readily. First, the S\textsubscript{1}-S\textsubscript{2} interval of 180 msec permitted halving of the mechanically effective heart rate. Second, both the S\textsubscript{1}-S\textsubscript{2} interval of 180 msec and the resulting S\textsubscript{2} interval of 205 msec were short enough to suppress the A-V junctional tachycardia whose spontaneous cycle length ranged from 205–228 msec. The escape of the underlying A-V junctional tachycardia at the initial ventricular paired pacing intervals (S\textsubscript{1}-S\textsubscript{2} = 400 msec, S\textsubscript{2} = 170–180 msec) was interpreted to mean that reset of the A-V junctional focus at the 170–180 msec S\textsubscript{1}-S\textsubscript{2} interval was accomplished, but during the requisite 230–220 msec S\textsubscript{2} interval which followed, the focus periodically was able to escape. These data are consistent with the notion that the spontaneous arrhythmia was an automatic A-V junctional tachycardia.

Immediately following initiation of stable ventricular pacing, the patient's hemodynamic status improved. The mean aortic pressure rose to 60–65 mm Hg and urine output resumed. Ventricular paired pacing was continued throughout the night, as each time it was stopped, the rapid

![Figure 1](http://circ.ahajournals.org/Download/177/1/E177_177_1_F1A.jpg)

**FIGURE 1.** Panels A and B each demonstrate in the top trace an ECG lead and in the bottom trace the recorded atrial electrogram. Time lines are at 1 second intervals. A = atrial electrogram. Paper recording speed = 50 mm/sec.
A-V junctional tachycardia returned at the same ventricular rate (285 beats/min). Fourteen and one-half hours after its onset, when the ventricular paired pacing was stopped, the spontaneous A-V junctional tachycardia had slowed to a rate of 240 beats/min (fig. 4). At this ventricular rate, the patient was hemodynamically stable. At this time, in an effort to further treat and better understand the arrhythmia, lidocaine was administered in three separate 5 mg intravenous doses over a total period of 30 minutes. During this period, the rate of A-V junctional tachycardia slowed to 200 beats/min (fig. 4). Also, the escape interval of A-V junctional rhythm after ventricular paired pacing was abruptly terminated, increased by 53 msec when compared with that immediately before the lidocaine administration (fig. 4). With the A-V junctional rhythm now at a rate of 200 beats/min, the patient’s clinical status was quite acceptable and it was elected to terminate ventricular paired pacing. Intravenous lidocaine then was continuously administered at a rate of 20 \( \mu \)g/kg/min.

The A-V junctional tachycardia continued to slow gradually. Twenty-four hours following surgery, the rate of the A-V junctional rhythm had decreased to 180 beats/min, and for the first time, there was 1:1 retrograde conduction to the atria. By the morning of the second postoperative day, the rate had decreased to 150 beats/min (fig. 5A). Atrial pacing at a rate of 160 beats/min was instituted and 1:1 A-V conduction with a long P-R interval was obtained (fig. 5B). At this time, the lidocaine infusion was terminated. By the third postoperative day, the patient’s spontaneous rhythm was primarily an A-V junctional rhythm which alternated with short periods of a conducted atrial rhythm. By the fifth postoperative day, the spontaneous rhythm was still either A-V junctional or atrial, the rates varying between 110 and 120 beats/min. By the ninth postoperative day, the patient was in a stable atrial rhythm. On the 15th postoperative day, having had a clinically successful postoperative course, the patient was discharged in a regular sinus rhythm (fig. 6).

Discussion

1. Ventricular Paired Pacing

There is little doubt that the successful use of ventricular paired pacing to control the very rapid A-V junctional tachycardia in this patient was life-saving. While the need for ventricular paired pacing to control various cardiac arrhythmias is not very frequent, clearly it is a useful and

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**Figure 2** The top panel demonstrates the recorded ECG during ventricular paired pacing at an \( S_1-S_1 \) interval of 400 msec, and an \( S_1-S_2 \) interval of 170 msec. The bottom trace records the blood pressure from a catheter in the femoral artery. Although these records were monitored simultaneously on an Electronics-for-Medicine Model IR-2 bedside oscilloscope, they were not recorded simultaneously, but rather were recorded sequentially. Note in the bottom record that there is only one pressure pulse for every two QRS complexes. Paper recording speed for both traces = 50 mm/sec.

**Figure 3** Top trace is the recorded ECG. The bottom trace is the recorded atrial electrogram during ventricular paired pacing at an \( S_1-S_1 \) interval of 385 msec and an \( S_1-S_2 \) interval of 180 msec. Time lines are at 1 second intervals. Paper recording speed = 100 mm/sec. \( S \) = stimulus artifact, \( A \) = atrial electrogram.
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**FIGURE 4** The top trace illustrates the ECG recorded immediately prior to administration of lidocaine. When the ventricular paired pacing was abruptly terminated, the escape interval of the A-V junctional rhythm measured from the last stimulus artifact to the initiation of the QRS complex of the first A-V junctional beat was 336 msec. Note that the spontaneous A-V junctional rhythm is now 240 beats/min. The bottom trace illustrates the ECG recorded immediately following the administration of the second 5 mg dose of lidocaine. Now, when the ventricular paired pacing was abruptly terminated, the escape interval of the A-V junctional rhythm was 389 msec. Note that the A-V junctional tachycardia has slowed to 200 beats/min. Paper recording speed = 100 mm/sec. Time lines are at 1 second intervals.

In any discussion of ventricular paired pacing, it always is appropriate to emphasize that utilization of this technique includes as a major hazard the accidental precipitation of ventricular fibrillation. Therefore, it is worth noticing that methods have been outlined which, if followed properly, reduce the likelihood of precipitating ventricular fibrillation virtually to zero. The following is a summary of these methods: 1) Probably most important, the duration and strength of the delivered stimuli must not be excessive and are best limited to a duration of 2 msec or less and a stimulus strength that does not exceed twice the diastolic threshold. 2) The electrode(s) utilized to deliver the stimuli should be in sustained firm contact with the myocardium. 3) As with any form of cardiac pacing, care must be taken to prevent inadvertent grounding of the heart. This is best accomplished by connecting the electrodes to an isolation device. 4) The manner of induction of ventricular paired pacing to control tachyarrhythmia probably is performed best as described in this case report: Using a paired-pulse stimulator, the S1-S2 interval should be set at an interval which is somewhat shorter than the cycle length of the spontaneous ventricular rate, and the S2-S3 interval should be set at twice the S1-S2 interval. This will produce a regular paced rhythm which is just faster than the spontaneous ventricular rate. After the paired-pulse stimulator is connected to the electrodes and pacing is initiated, the stimulus strength.
should be increased gradually until constant ventricular capture is obtained. The S1-S2 interval then is shortened gradually and carefully until the S2 stimulus produces an electrical response (QRS complex) for which there is no effective mechanical response. The mechanical response is monitored best by directly recording the arterial pulse pressure using standard techniques. 5) An ECG should be monitored continuously during the ventricular paired pacing, and preferably simultaneously with an arterial pulse pressure.

2. A-V Junctional Automatic Tachycardia

The following observations strongly suggest that this tachycardia resulted from an ectopic automatic focus firing very rapidly: a) the rate of the spontaneous A-V junctional tachycardia increased and later decreased gradually over quite a long period of time; b) during ventricular paired pacing, a critical interval for reset of the A-V junctional focus was noted. When the S1-S2 interval was too short or, conversely, when the S2-S3 interval was too long, the A-V junctional rhythm escaped; c) the spontaneous A-V junctional rate was slowed by the administration of intravenous lidocaine; d) the spontaneous A-V junctional rhythm demonstrated overdrive suppression, a property characteristic of spontaneous pacemakers; e) the spontaneous A-V junctional tachycardia was not interrupted by pacing the ventricles at rates faster than the spontaneous rate, suggesting, though clearly not proving, that the spontaneous rhythm was not a re-entrant tachycardia. Therefore, it seems a reasonable conclusion that this ectopic tachycardia was most likely an automatic rather than a re-entrant tachycardia.

It is of considerable interest that an ectopic pacemaker can fire at a rate of over 300 beats per minute. For instance, one of the arguments used to suggest that supraventricular tachycardias such as atrial flutter are most likely re-entrant rhythms was the notion that ectopic pacemakers could not fire as rapidly as 300 beats/min (the usual atrial rate in atrial flutter). However, this case indicates that at least in children, ectopic pacemakers certainly are capable of firing as rapidly as 285 beats/min. Thus, the argument that atrial flutter is too fast per se to be an automatic tachycardia no longer seems valid.

In summary, we have presented a unique case in which we were able to document the presence of an automatic A-V junctional tachycardia. Initially, effective treatment was only possible by utilizing ventricular paired pacing. This case demonstrates the usefulness of ventricular paired pacing to control tachycardias resistant to usual modes of therapy, and suggests that ventricular paired pacing may be a useful therapeutic adjunct to improve ventricular function in children.

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Figure 6 Twelve-lead electrocardiogram recorded on the eighth postoperative day demonstrating a spontaneous sinus rhythm.
Left Ventricular Diverticulum and Mitral Incompetence in Asymptomatic Children

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SUMMARY Two children with congenital apical left ventricular diverticulum and significant mitral incompetence are reported. The angiographic and anatomic findings of the few previously reported patients with congenital diverticula and aneurysm were analyzed and a new classification differentiating between the two was proposed.

The clinical data analyzed in apical diverticula and aneurysm are similar to those patients with anomalous origin of the left coronary artery from the pulmonary artery. Left ventriculography is the best diagnostic tool.

The place of surgery in the treatment of the congenital apical diverticula with or without mitral incompetence in asymptomatic children is not clear. Further observations are needed to reveal its natural history.

ANEURYSM OR ANEURYSMAL DILATION of the left ventricle may complicate anomalous origin of the left coronary artery from the pulmonary artery. However, true congenital aneurysm of the left ventricle in children is very rare. Only a few scattered reports are available and in these the aneurysm was attributed to cardiomyopathy or to a congenital muscular defect of the left ventricle.

In previous communications the terms aneurysm and diverticulum have been used interchangeably. Serious attempts were not made to differentiate between congenital left ventricular aneurysm and the different types of diverticula. However, from a review of the few previously reported cases, it is possible to classify the diverticula and to differentiate them from a true congenital aneurysm.

This report presents two asymptomatic children with left ventricular apical diverticulum of undetermined cause accompanied by severe mitral incompetence, a combination never previously observed in children. Anatomic and angiographic differences between aneurysm and diverticula are defined and the appropriateness of surgery in such patients is discussed.

Case 1

This two-year-old bedouin girl was hospitalized in February, 1974, for kerosene pneumonitis. According to this history she was the product of a normal pregnancy and delivery. Prior to present admission she was hospitalized three additional times for recurrent gastroenteritis and pneumonitis, at the age of four and ten months, respectively. (Unfortunately we do not have any information concerning her cardiovascular status during these three hospitalizations in 1972.)

On admission in 1974, the physical examination revealed a well-developed girl without cyanosis or signs of congestive failure. The systolic blood pressure was 90 mm Hg. The peripheral pulses were equal and normal on palpation. The heart size was enlarged with a widespread, lifting, cardiac impulse palpable at the 6th left intercostal space in the anterior axillary line. There were no thrills, but a definite left ventricular heave was present. The first heart sound was normal, the second sound at the second left intercostal space was single and did not split with respiration. A loud third

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_Circulation_. 1976;53:176-181
doi: 10.1161/01.CIR.53.1.176

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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