Single-chamber cardiac pacing with activity-initiated chronotropic response: evaluation by cardiopulmonary exercise testing

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ABSTRACT In this study, sequential cardiopulmonary exercise testing was used to assess the physiologic benefits of a single-chamber ventricular pacing system that utilizes a piezoceramic sensor to adjust heart rate by detecting “physical activity.” An initial exercise test was conducted with the pacemaker programmed (based on a randomization table) to either the fixed rate (VVI, 70 beats/min) or rate-variable (VVI-Act) mode, and the results were compared with those obtained during a second exercise test in which the pacemaker was programmed to the alternate pacing mode. A 1.5 to 2 hr rest period was permitted between exercise tests, each of which consisted of a symptom-limited constant speed (3.0 mph) Balke protocol with 2 min stages commencing at 0.0% grade with increments of 2.5% at end of each stage. Compared with findings during fixed-rate VVI pacing, VVI-Act pacing was associated with (1) greater exercise-induced positive chronotropic response (mean maximum heart rate VVI-Act 128 ± 15.3 beats/min vs VVI 90 ± 28.4 beats/min; p < .01), (2) prolongation of exercise duration (VVI-Act 10.2 ± 3.8 min vs VVI 7.7 ± 2.5 min; p < .01), (3) increased peak oxygen consumption (VVI-Act 1617 ± 656 ml O2/min vs VVI 1325 ± 451 ml O2/min; p < .01), and (4) onset of anaerobic threshold at a higher oxygen consumption (VVI-Act 1208 ± 343 ml O2/min vs VVI 1064 ± 377 ml O2/min; p < .01). Additionally, of 44 comparable exercise stages tested in the two pacing modes, perceived exertion (assessed by a numerical grading system) was lower in 38 of 44 instances during VVI-Act compared with VVI pacing. Thus provision of chronotropic response during exercise by single chamber rate-variable ventricular pacing substantially improved exercise tolerance and therefore may be of benefit for patients with bradycardia-related symptoms in whom associated sinoatrial disease or difficulty establishing or maintaining atrial sensing preclude optimum function of conventional atrial-tracking pacing systems.


OPTIMAL CARDIAC PACING provides both atrioventricular (AV) synchrony and a physiologically appropriate heart rate.1,2 To achieve these goals, both atrial synchronous (VDD) and universal (DDD) pacing modes track native atrial rate and pace the ventricles after a preset AV interval. However, in many potential candidates for placement of cardiac pacemakers, abnormal sinoatrial function may preclude appropriate operation of atrial-tracking devices.3 For example, in patients with sinus bradycardia and subnormal response in heart rate during exercise,4 usefulness of atrial tracking is limited by inability of the native atrial rate to attain levels appropriate to the degree of exertion. Similarly, in patients with complete or intermittent AV block in conjunction with chronic atrial fibrillation, refractory primary atrial tachycardia,5 or inexcitable atria, conventional pulse generators are incapable of providing either AV synchrony or consistently appropriate heart rate responses during physical exertion. Consequently, there has been considerable interest in development of pacemakers that provide heart rate responsiveness independently of atrial function.6–17 This study used graded treadmill exercise to assess the potential physiologic benefits of a single-chamber ventricular pacing system that adjusts pacing rate by detecting and tracking physical activity.11–17 To
The signal processor. In this system, a low threshold permits the device to respond to relatively minor body movements, whereas a high threshold excludes recognition of low-amplitude vibrations and permits recognition only of signals associated with more intense body activity.

A programmable "rate response" setting determines the relationship between detected body activity signals and the resulting pacing rate of the pulse generator. Ten rate response settings are available; a response of 10 permits greatest pacing rate change in response to body activity, whereas a response set at 1 permits the least pacing rate change. The range of pacing rates are restricted by a programmable lower or "base" rate (60, 70, 80 pulses/min), below which the pulse generator pacing rate is not allowed to fall, and a similarly adjustable upper rate (100, 125, 150 pulses/min), which is the maximum pacing rate permitted (figure 1).

Exercise treadmill protocol. In 11 of 12 patients an initial familiarizing exercise treadmill test was carried out within 1 to 2 weeks before pacemaker implantation. Patient 9 did not undergo this procedure because of severe angina pectoris. In addition, after implantation of the pulse generator, but before the sequential exercise tests reported below, activity threshold and rate response characteristics were established by assessing pacing response during low-grade treadmill exercise activity (1.5 mph, 0.0%, and 2.5% grade) in all 12 patients. Thus patients were given the opportunity to become at ease with both treadmill exercise testing procedures and laboratory surroundings.

Exercise studies were conducted in an environmentally controlled laboratory (20° to 22°C, 40% to 50% humidity) with patients in the postprandial state (3 to 4 hr). Subjects were not permitted to use side rails while on the treadmill. Before commencement of the first of the two sequential cardiopulmonary exercise tests described below, the implanted pulse generator was programmed (based on a previously determined randomization table) to either a fixed rate (VVI, 70 ppm) pacing mode or the activity-initiated rate-responsive pacing mode (VVI-Act). The second of the sequential studies was undertaken after a 1.5 to 2 hr rest period, employing the previously untested pacing mode. Patients were not informed of which pacing mode was programmed.

A modified symptom-limited constant speed (3.0 mph) treadmill (Quinton Model 18-60) was used. The protocol consisted of 2 min stages starting at a grade of 0.0%, with grade increments of 2.5% after completion of each stage until volitional maxi-

**Figure 1.** Schematic representation of circuitry and operating characteristics of the activity-initiated variable-rate pacing system used in this study.

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mum was achieved. Peak oxygen uptake (VO₂ peak) and oxygen uptake at anaerobic threshold (VO₂-AT) were determined during these relatively brief exercise tests. This method minimizes both patient fatigue and recovery time, thereby permitting repeat exercise testing to evaluate the effect of a change in pacing mode.

The effects of exercise on cardiopulmonary variables were measured with a computerized breath-by-breath technique (System 2000, Medical Graphics Corp., St. Paul), which employed an infrared CO₂ analyzer, a zirconium cell oxygen analyzer, and a pneumotachometer. All analyzers were calibrated and lag times calculated immediately before each exercise test. With this technique, minute ventilation (VE), oxygen uptake (VO₂), and carbon dioxide output (VCO₂) were determined for each breath by direct analysis of expired air. Respiratory exchange ratio, ventilatory equivalents for VO₂ and VCO₂, and end-tidal PO₂ and PCO₂ were calculated from these measurements.

Anaerobic threshold (AT) was determined noninvasively by incorporating three of the methods that have been described for its estimation: (1) the association between VE and VO₂, with AT being defined as the point of departure from linearity of this relation, (2) the point at which there is an increase in the ventilatory equivalent for VO₂ (VE/VO₂) without a concomitant increase in the ventilatory equivalent for VCO₂ (VE/VCO₂), and (3) the break point in the systemic increase in end-tidal PO₂. In each case, close agreement (< 5% variation) was found among the three methods.

Throughout exercise, arterial blood pressure was recorded at regular intervals with a Puritan-Bennet Infrasonde D400 blood pressure monitor, and the electrocardiogram was monitored continuously (leads aVF, V₁, and V₅), with recordings obtained at 30 sec intervals. In addition, perceived exertion during exercise was assessed by a numerical grading system in which the patient’s perception of exercise intensity was measured on a scale of 6 to 20.6 was considered to be minimal intensity, 13 was moderate intensity, and 20 was extremely heavy exercise. This scale correlates well with several exercise-induced changes in physiologic variables, is helpful in assessing the influence of symptoms in limiting exercise tolerance, and permits comparison of symptoms with objective measures of cardiopulmonary status.27-29

Data analysis. All data are presented as mean ± SD. The statistical significance of pacing mode–related changes in systemic blood pressure, heart rate, exercise duration, cardiopulmonary variables, and perceived exertion were determined with Student’s t test for paired data. Statistical significance was set at p < .05.

Results

Between April 1984 and March 1986, activity-initiated rate-variable pacemakers were implanted in 17 patients at the University of Minnesota Hospitals. Results of sequential exercise testing with measurement of cardiopulmonary variables in 12 of these patients (seven females, five males; ages 11 to 71 years) comprise the basis of this report. Five patients (one female, four males; ages 6 to 72 years) were not included in the treadmill exercise protocol. Orthopedic limitations precluded treadmill exercise testing in three, sequential cardiopulmonary exercise studies were refused by one, and one patient, a 6-year-old boy, was not believed to be capable of manipulating the apparatus for measurement of metabolic variables while undergoing treadmill exercise.

Clinical findings in the 12 individuals included in this study are summarized in table 1. In these patients, provision of an appropriate response in heart rate during exercise was not achievable with conventional atrial-tracking devices because of medically refractory chronic or paroxysmal atrial tachyarrhythmias (four patients), inadequate sinoatrial heart rate response during exercise (seven patients), or an inexcitable atrium (one patient). In all but three patients (patients 6, 10, and 12), the presence of bidirectional AV block and/or atrial fibrillation precluded concerns regarding the potential adverse effects of retrograde (VA) conduction during ventricular pacing. In these three individuals, VA conduction was observed only in patient 6 and was evident only at heart rates less than approximately 80 beats/min.

Based on a randomization table, exercise testing was carried out in the fixed rate VVI mode first in seven patients; in the remaining five patients the rate-variable VVI-Act mode was tested first (table 2). During exercise in the VVI-Act mode, a lower rate limit of 60 beats/min was used in all patients, and with the exception of patients 3 and 9 the upper rate limit was set at 150 beats/min. Patient 3 was evaluated at a time when the maximum upper rate limit was restricted to 125 beats/min. Patient 9 had symptoms of angina pectoris, and the upper rate setting of 100 beats/min was selected to minimize risk of exacerbation of symptoms. Figure 2 illustrates electrocardiographic findings during rest and exercise in patient 1 with the pacemaker programmed to the VVI-Act mode.

Blood pressure, heart rate, and exercise duration. Systemic arterial pressure, measured immediately before commencement of treadmill exercise with the patient in the standing position, did not differ significantly for the two phases of the sequential exercise protocol (baseline systolic pressure, VVI 126 ± 18.6 mm Hg vs VVI-Act 132 ± 27.2 mm Hg, p = NS; baseline diastolic pressure, VVI 72 ± 10.7 mm Hg vs VVI-Act 78 ± 10.4 mm Hg, p = NS) (figure 3). However, during exercise in the fixed-rate VVI pacing mode systolic pressure tended to rise initially, then to plateau after 4 min of exercise. Diastolic pressure exhibited a tendency to fall in those patients exercising 8 min or longer. Exercise with the pulse generator programmed to the VVI-Act mode resulted in a more consistent systolic pressure rise throughout the exercise period, whereas diastolic pressures remained essentially unchanged from baseline values (figure 3).

Figure 4 illustrates individual heart rate responses
### Clinical features

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)/sex</th>
<th>Symptoms</th>
<th>ECG Findings</th>
<th>Cardiac diagnosis</th>
<th>Pacemaker settings (VVI-Act mode)</th>
<th>Activity threshold</th>
<th>Rate response</th>
<th>Follow-up</th>
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<tbody>
<tr>
<td>1</td>
<td>44/F</td>
<td>Palpitations</td>
<td>1° At tach</td>
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<td>Junct. rhythm</td>
<td>CAD, inexcitable, atrium</td>
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<td>At fibr, CHB</td>
<td>CAD</td>
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<td>12</td>
<td></td>
</tr>
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<td>Sinus brady</td>
<td>SND, inadequate exercise HR</td>
<td>High</td>
<td>9</td>
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<td>24/M</td>
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<td>VVI paced</td>
<td>TGV, CHB,(^b) inadequate exercise HR response</td>
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<td>11</td>
<td></td>
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<tr>
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<td>28/F</td>
<td>Exer intol</td>
<td>Junct. rhythm</td>
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<tr>
<td>9</td>
<td>58/F</td>
<td>Palpitations</td>
<td>Parox at fibr</td>
<td>CAD, CHB,(^a) BTS</td>
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</table>

\(^a\) At tach = primary atrial tachycardia; \(^b\) at fibr = atrial fibrillation; BTS = bradycardia-tachycardia syndrome; CAD = coronary artery disease; CHB = complete heart block; CV = common ventricle; EOL = end of life; Exer intol = exercise intolerance; HR = heart rate; SND = sinus node dysfunction; TGV = complete transposition of great vessels.

Table 2

### Findings during sequential cardiopulmonary exercise tests: comparison of fixed rate and activity-initiated rate-responsive ventricular pacing

<table>
<thead>
<tr>
<th>Patient</th>
<th>Exercise duration (min)</th>
<th>Peak HR - SBP</th>
<th>Peak VO(_2) (ml/min)</th>
<th>VO(_2)-AT (ml/min)</th>
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<tr>
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<td>VVI/Act</td>
<td>VVI</td>
<td>VVI/Act</td>
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<td>4</td>
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<td>7.5</td>
<td>9.5(^a)</td>
<td>9,000</td>
<td>20,900</td>
</tr>
<tr>
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<td>4.3</td>
<td>15,120</td>
<td>31,250</td>
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<tr>
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<td>8.6(^a)</td>
<td>9.8</td>
<td>10,950</td>
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<tr>
<td>8</td>
<td>6.4</td>
<td>7.7(^a)</td>
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<td>6.2(^a)</td>
<td>9.4</td>
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<td>11.6</td>
<td>18.9(^a)</td>
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<tr>
<td>12</td>
<td>11.1(^a)</td>
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<tr>
<td>Mean</td>
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<td>10.2</td>
<td>14,600</td>
<td>24,672</td>
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<tr>
<td>± SD</td>
<td>± 2.5</td>
<td>± 3.8</td>
<td>± 4,519</td>
<td>± 5,848</td>
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</table>

\(^a\) indicates the pacing mode tested first.

HR-SBP = heart rate-systolic blood pressure product.
A Standing (Pre Treadmill Exercise)

B Treadmill Exercise (4 Minutes)

Patient #1

FIGURE 2. Electrocardiographic tracings obtained in patient 1 while standing before exercise (A) and while walking on the treadmill (B). Vertical bars denote atrial activity (P waves). With the patient standing, the atrial rate is approximately 90 beats/min and the ventricular rate is 92 beats/min. At 4 min of exercise the atrial rate is approximately 129 beats/min and the ventricular paced rate is 138 beats/min.

achieved at each 2 min stage of treadmill exercise during both VVI (panel A) and VVI-Act (panel B) mode pacing. For comparable exercise stages, heart rates were significantly greater (p < .01, paired t test) during VVI-Act pacing (128 ± 15.3 beats/min) than during VVI pacing (90 ± 28.4 beats/min). Additionally, maximum heart rate–systolic blood pressure product achieved was greater during VVI-Act pacing (p < .01, table 2). Of note, in patient 9 angina pectoris occurred at approximately equivalent heart rate–systolic blood pressure products during treadmill exercise in each pacing mode. However, selection of a relatively conservative rate response of 5 and an upper rate limit of 100 beats/min during VVI-Act pacing (table 1) delayed her achieving the critical rate-pressure product and contributed to improved exercise tolerance and longer treadmill exercise duration. Overall, pacing in VVI-Act mode was associated with prolongation of treadmill exercise duration in 11 of 12 (92%) patients (table 2). For the group, a mean increase of exercise duration of 32.5% was achieved (VVI-Act 10.2 ± 3.8 min vs VVI 7.7 ± 2.5 min; p < .01).

VO₂ peak and AT. VO₂ peak and VO₂-AT are summarized for each patient in table 2 and illustrated in figure 5. Compared with findings during VVI mode pacing, exercise during VVI-Act pacing was accompanied by an increased VO₂ peak in 11 of 12 (92%) patients. Overall, mean VO₂ peak increased 22% (VVI-Act 1617 ± 656 ml O₂/min vs VVI 1325 ± 451 ml O₂/min; p < .01).

VO₂-AT was achieved in nine of 12 (75%) patients exercising during VVI pacing and in 10 of 12 (83%) patients exercising during VVI-Act pacing. Compared with findings during exercise with VVI pacing, exercise in the VVI-Act mode was associated with a mean increase in VO₂-AT of 13.5% (VVI-Act 1208 ± 343 ml O₂/min vs VVI 1064 ± 377 ml O₂/min; p < .01).

Perceived exertion. The mean level of perceived exertion at each exercise stage (estimated by a numerical grading system, see above) was consistently lower during VVI-Act pacing than during VVI pacing (figure 6). Indeed, for each of the total of 44 exercise stages completed by the 12 patients while being paced in the fixed-rate VVI mode, the comparable exercise stage during VVI-Act pacing was reported to be associated with a lower perceived exertion level in 38 of 44 (86%) instances and equivalent in four of 44 (9%) instances.

FIGURE 3. Graphs depicting relative effects of VVI and VVI-Act pacing on systolic and diastolic blood pressure during each stage of treadmill exercise. Blood pressure (normalized with respect to baseline value) is indicated on the ordinate and exercise duration is noted on the abscissa. During VVI-Act pacing, systolic blood pressure tended to rise throughout exercise, whereas diastolic pressure rose initially and plateaued. On the other hand, during VVI pacing both systolic and diastolic blood pressures tended to decline after an initial exercise-induced rise.
Follow-up. In each of the 12 patients, pulse generators have remained programmed to the VVI-Act mode throughout the follow-up period (range 2 to 24 months, table 1). In patients 3 and 10, pacemaker reprogramming was necessary during the follow-up period; in patient 11 a pacing system problem was encountered. Patient 3 experienced periodic transient sensations of excessively rapid heart rates (90 to 110 beats/min) while in bed at night. These episodes occurred only when he lay in such a manner that the pulse generator was pressed against the mattress. This difficulty, presumably arising from closer mechanical coupling of the device to the body (thereby increasing its sensitivity to ambient pressure wave signals), was alleviated by programming the activity threshold from medium to high. Thus the sensitivity of the device to low-amplitude pressure waves was diminished. Patient 10 noted a tendency for her heart to beat at a faster rate than she felt appropriate when riding in a motor vehicle. In this patient the activity threshold had been changed from high to medium after the exercise treadmill tests. The problem was resolved by reprogramming the activity threshold to high. In patient 11, dislodgment of the pacing lead occurred within 4 days of pacemaker implantation. The lead was repositioned and no further problem was encountered.

Discussion

This study used graded treadmill exercise with measurement of cardiopulmonary indexes to compare exercise tolerance during single-chamber activity-initiated chronotropic response ventricular pacing (VVI-Act) with that achieved during conventional fixed-rate ventricular pacing (VVI). Findings indicate that VVI-Act pacing consistently (1) extended exercise duration, (2) increased VO₂ peak, (3) delayed achievement of anerobic threshold (VO₂ - AT) to a higher level of oxygen consumption, and (4) decreased level of perceived exertion during comparable stages of exercise.

Contributions of AV synchrony and chronotropic response to exercise tolerance. Compared with findings during fixed-rate ventricular pacing (VVI), provision of both AV synchrony and heart rate responsiveness in paced patients has been associated with improvements in both cardiovascular hemodynamic indexes (e.g., cardiac output, arteriovenous oxygen differences) and exercise tolerance. However, available evidence suggests that it is the positive chronotropic response that provides by far the greater contribution to this augmentation of exercise performance. For example, despite an overall threefold increase in cardiac output between rest and exercise, Karlof observed that only approximately 8% of this increment was attributable to preservation of AV synchrony. More recently, Fananapazir et al. reported that the approximate 40% increase in exercise capacity associated with heart rate responsiveness in their patients (compared with fixed-rate ventricular pacing) was independent of whether rate responsiveness was provided in conjunction with AV synchrony. The latter observations have been confirmed by Ausubel et al. although these investigators noted that at comparable rates the increased cardiac output achieved with single-chamber rate-responsive ventricular pacing was associated with a need to invoke compensatory contractile reserve as evidenced by

FIGURE 4. Heart rate response at the end of each stage of treadmill exercise for the 12 patients studied. In each panel the ordinate indicates ventricular rate (beats/minute) and the abscissa depicts exercise duration (min). Note that during exercise in the VVI-Act mode, "breakthrough" of native cardiac rhythm occurred in patients 4, 10, and 12 (indicated by daggers).
a smaller end-diastolic volume and an increased ejection fraction.

The availability of piezoceramic sensor-activated rate-responsive pulse generators appears to provide the opportunity to improve exercise capacity substantially for patients with single-chamber pacing systems. In this study, increments in exercise duration and VO2 peak of 33% and 22%, respectively, were achieved during VVI-Act pacing, findings similar to but of a greater magnitude than those observed by Humen et al.12 in six patients (mean age 69 ± 14.3 years) who exercised under comparable conditions. The better exercise performance observed in our study may have been due in part to a younger patient group (43 ± 19 years) but in large measure was due to the fact that noncardiac conditions (peripheral vascular disease, arthritis, chronic obstructive pulmonary disease) commonly limited exercise performance in Humen’s patients.

Clinical implications. In potentially active patients with pacemakers, provision of both AV synchrony and an appropriate response in heart rate is desirable when feasible. On the other hand, for patients in whom primary atrial arrhythmias (e.g., atrial fibrillation, inexcitable atria) preclude maintenance of AV synchrony or in whom abnormal sinoatrial function results in symptoms caused by inadequate chronotropic responsiveness, a cardiac pacing system that provides an activity-initiated heart rate response permits quantitative (oxygen consumption, exercise duration) and qualitative (perceived exertion) improvements in exercise tolerance. Potentially similar improvement in exercise tolerance may be achieved in a broader category of patients in whom technical difficulties prevent obtaining and/or maintaining adequate long-term atrial sensing or pacing (e.g., growing children) or in whom an already existing single-chamber pacing lead is to be reused when a pacemaker generator is replaced.

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