CARDIAC PACING

Rate-Modulated Cardiac Pacing Based on Transthoracic Impedance Measurements of Minute Ventilation: Correlation With Exercise Gas Exchange

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The relation of pacing rate to physiologic variables of metabolic demand was examined in 10 consecutive patients with a minute ventilation-sensing, rate-modulating ventricular pacemaker implanted for complete heart block. All patients had paroxysmal (seven patients) or chronic (three patients) atrial fibrillation and were referred for catheter ablation of the atrioventricular junction. Treadmill exercise testing with measurement of expired gas exchange and respiratory flow was performed before ablation and 4 weeks after pacemaker implantation, with the pacemaker programmed to both the fixed-rate VVI and rate-modulating minute ventilation VVIR pacing modes in random sequence. The relation of pacing rate to oxygen consumption (VO₂), expired carbon dioxide concentration (VCO₂), respiratory quotient, tidal volume, respiratory rate and minute ventilation was determined during exercise in the rate-modulating minute ventilation pacing mode. Pacing rate was highly correlated with minute ventilation (r = 0.89), respiratory quotient (r = 0.89), VCO₂ (r = 0.87), tidal volume (r = 0.87), VO₂ (r = 0.84) and respiratory rate (r = 0.84).

The mean exercise duration increased from 8.3 ± 2.8 min in the fixed rate pacing mode to 10.2 ± 3.4 min in the rate-modulating, minute ventilation mode (p = 0.0001). The maximal VO₂ increased from 13.4 ± 3.4 to 16.3 ± 4.1 cc/kg per min (p = 0.0004). The maximal heart rate achieved in the minute ventilation pacing mode was 136 ± 9.7 beats/min, similar to that observed in the patient's intrinsic cardiac rhythm before ablation (134.9 ± 30.1 beats/min, p = NS). Isoproterenol infusion at rates of 1 and 2 µg/min produced no change in pacing rate, suggesting that the rate-modulating algorithm of the pacemaker was capable of discriminating changes in transthoracic impedance related to respiration from those related to right ventricular stroke volume.

Thus, rate-modulating pacemakers that measure minute ventilation by transthoracic impedance provide increases in pacing rate during exercise that closely parallel physiologic variables of metabolic demand.

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Although the normally functioning sinus node is the most physiologic sensor for rate-modulated ventricular pacing and provides for atrioventricular (AV) synchrony, some patients requiring a permanent pacemaker for the treatment of symptomatic bradycardia are not suitable for atrial synchronous, dual-chamber (DDD) pacing because of atrial tachyarrhythmias or sinus node dysfunction (1). Several sensors have been developed to modulate the rate of cardiac pacemakers and the clinical benefits of ventricular-inhibited, rate-modulated (VVIR) as compared with fixed rate ventricular inhibited (VVI) pacing have been clearly demonstrated (2-8). Because respiratory flow is linearly related to oxygen consumption and the rate of the normal sinus node during aerobic exercise (8-12), pacing systems that accurately measure minute ventilation are likely to provide physiologic modulation of heart rate (8,12,13). Exercise (11,12) in normal volunteers and patients with congestive heart failure have shown that minute ventilation correlates with oxygen consumption in a variety of cardiac disorders. Previous reports (14-16) have demonstrated that measurements of transthoracic impedance with standard bipolar catheters or auxiliary subcutaneous leads have a close relation to variables of respiratory flow,
including tidal volume, respiratory rate and minute ventilation.

The present study prospectively examined the heart rate response to incremental treadmill exercise in patients with a new rate-modulated pacemaker that senses minute ventilation from measurements of transthoracic impedance (17) in a consecutive series of patients with complete heart block after catheter ablation of the AV junction. The relation of heart rate during exercise to several physiologic variables of metabolic demand, including respiratory flow, oxygen consumption and expired carbon dioxide concentration was determined for patients in their intrinsic rhythm before ablation and in both the fixed rate VVI and rate-modulated minute ventilation VVIR pacing modes.

Methods

Study patients. Ten consecutive patients with chronic or paroxysmal atrial fibrillation referred for catheter ablation of the AV junction were prospectively studied. The study protocol was approved by the institutional review board for research involving human subjects of the University of Alabama at Birmingham. All patients gave written informed consent. Catheter ablation was performed after sedation with intravenous thiopental. Two direct current shocks of 300 joules each were delivered from the distal pole of a specially designed 8F bipolar electrode catheter (Bard Electrophysiology) at a site recording the largest bipolar His bundle potential (18). Permanent complete heart block was achieved in all patients.

The day after catheter ablation, a permanent bipolar, ventricular-inhibited, rate-modulated (VVIR) pacing system was implanted under local anesthesia with standard implantation techniques. A bipolar permanent pacing lead was inserted into the left cephalic vein and positioned at the right ventricular apex in all patients. A Teletronics 1202 MV pulse generator (Teletronics and Cordis Pacings Systems) was then implanted in a prepectoral pocket.

Description of the pulse generator. The 1202 MV pulse generator calculates transthoracic impedance every 50 ms (20 Hz) by emitting an impulse of known current (1 mA, pulse duration 15 microseconds) from the proximal ring electrode of the bipolar pacing lead and measuring resultant voltage between the distal tip electrode and the pulse generator case. Because transthoracic impedance increases with inspiration and decreases with expiration, the respiratory rate can be calculated from the frequency of excursions of the impedance signal. The tidal volume is estimated from the amplitude of each impedance signal excursion. The product of the frequency and amplitude of changes in transthoracic impedance is an estimate of minute ventilation. Filtering of the impedance signal with a low pass filter of 1 Hz is designed to discriminate changes in transthoracic impedance related to respiration from those related to right ventricular stroke volume. The impedance signal is averaged over two time periods, one of 1 min duration, the other with a duration of 1 h, each updated every 50 ms. The difference between the 1 min and 1 h impedance averages is used to modulate changes in pacing rate. The pacing rate is modulated so that 50% of the difference between the present pacing rate and the target rate (derived from the impedance measurements) is achieved over a period of 35 s. Thus, changes in pacing rate occur exponentially. The slope of the relation between the change in pacing rate for a given change in the impedance signal is a programmable parameter (termed the rate-response factor), with 59 values available.

The pulse generator must be programmed to the fixed rate, "adaptive" mode for at least 1 h before activation of the rate modulating minute ventilation mode. In the adaptive mode, the impedance impulses are emitted and the baseline impedance measurements at rest are averaged, allowing automatic calibration of the pulse generator for the rest transdiaphragmatic impedance excursions of the individual. Changes in transthoracic impedance detected by the pulse generator can be determined by telemetry of the device. A treadmill exercise test with the pulse generator programmed to the adaptive mode allows measurement of the change in transthoracic impedance from rest to peak exercise, and a rate response factor to be used when the pacemaker is programmed to the minute ventilation mode can be telemetered from the pulse generator. The pulse generator can then be programmed to the rate-modulated minute ventilation mode with the appropriate rate-response factor.

Exercise testing protocol. In all patients, treadmill exercise testing with measurement of respiratory flow and expired gas exchange was performed on the day before catheter ablation. Beta-adrenergic blocking drugs and calcium channel antagonists were discontinued before baseline exercise testing. Type I antiarrhythmic medications and digoxin were continued. On the day after pacemaker implantation, treadmill exercise testing (modified Bruce protocol) was performed with the pacemaker programmed to the adaptive mode to determine the appropriate rate-modulating rate-response factor for the pacemaker. The pulse generator was programmed to the minute ventilation mode, with a lower pacing rate of 70 beats/min and an upper rate equal to 85% of the age-predicted maximal heart rate (calculated from the formula 220 - age in years). Four weeks after discharge, the patients were asked to perform paired, symptom-limited treadmill exercise tests with measurement of expired gas exchange using the modified Bruce protocol. The pulse generator was programmed to the fixed rate adaptive mode (70 beats/min) for one exercise test and to the rate-modulating minute ventilation mode for the other test. The minimal rest period between the exercise tests was 30 min, and the sequence of programmed pacing modes for each test was chosen at random. Patients did not know the pacing mode used during paired exercise testing.
Expired gas exchange. During paired exercise tests, patients were connected to a pneumotachometer by a rubber mouthpiece with a unidirectional three-way valve for the measurement of respiratory flow and expired gas exchange. Oxygen consumption (VO$_2$, expressed as ml O$_2$/kg per min) was determined by a galvanic cell (Applied Electrochemistry, Inc.). Expired carbon dioxide concentration (VCO$_2$) was determined by mass spectroscopy (Applied Electrochemistry, Inc.). Anaerobic threshold was determined by plotting minute ventilation and VCO$_2$ versus VO$_2$, and was considered to have been reached when the ratio of both minute ventilation and VCO$_2$ to VO$_2$ increased.

Isoproterenol infusion. The ability of the rate-modulating algorithm of the pacemaker to discriminate changes in transthoracic impedance related to respiration from those related to right ventricular stroke volume was investigated by measurement of pacing rate and the telemetered change in transthoracic impedance from the pulse generator during intravenous infusion of isoproterenol. With the pulse generator programmed to the minute ventilation mode, continuous electrocardiographic monitoring and measurement of expired gas exchange were performed for 5 min at rest and for two 5 min periods during infusion of isoproterenol at 1 and 2 $\mu$g/min. The pulse generator was interrogated every 60 s to determine changes in transthoracic impedance measured by the device.

Data analysis. Oxygen consumption (VO$_2$), carbon dioxide production (VCO$_2$), respiratory rate, tidal volume, minute ventilation and respiratory quotient measured from expired gas exchange analysis were compared with the telemetered transthoracic impedance measurements from the pulse generator and the actual pacing rate observed for each patient during exercise, using Pearson’s correlation coefficient. The correlation coefficients of each physiologic variable with the transthoracic impedance measurements and pacing rate from all patients in the study were averaged. Correlation between heart rate and physiologic variables of metabolic demand during exercise testing was examined in the patient’s intrinsic rhythm before ablation and in the rate-modulated minute ventilation pacing mode after ablation. Comparison of continuous data between the pacing modes was performed with the Wilcoxon rank sum test.

Results

Patient characteristics. Ten consecutive patients referred for catheter ablation of the AV junction for paroxysmal (seven patients) or chronic (three patients) atrial fibrillation refractory to medical therapy were included in the study. Permanent complete heart block was produced with catheter ablation in all patients. The mean age of the study patients was 62.6 ± 16.7 years; there were seven men and three women. One patient had mild compensated congestive heart failure. Clinically significant obstructive pulmonary disease was present in two patients, both requiring therapy with inhaled bronchodilators, oral theophylline and intermittent corticosteroids.

Exercise tolerance and oxygen consumption (Table 1). The results of paired exercise testing with the pacemaker programmed to the fixed rate adaptive mode and the rate-modulated minute ventilation mode are shown in Table 1. The mean exercise duration increased from 8.3 ± 2.8 min in the adaptive mode to 10.2 ± 3.4 min in the minute ventilation mode ($p = 0.001$). The maximal heart rate achieved with exercise was 136 ± 9.7 beats/min with the minute ventilation pacing mode, similar to that observed at baseline: 134.9 ± 30.1 beats/min, $p = NS$). The target heart rate (85% of age-predicted maximum) of 133.8 ± 14.2 beats/min was not significantly different from the maximal heart rate achieved in the minute ventilation pacing mode ($p = 0.59$). All patients maintained a pacing rate of 70 beats/min in the adaptive pacing mode. The maximal VO$_2$ attained increased from 13.4 ± 3.4 ml/kg per min in the adaptive mode to 16.3 ± 4.1 ml/kg per min in the minute ventilation mode ($p = 0.004$). Anaerobic threshold was achieved during exercise in the minute ventilation mode by 7 of the 10 patients. The underlying rhythm at the time of the baseline exercise test before catheter ablation was sinus in six patients and atrial fibrillation in four patients. The mean exercise duration before ablation was 7.47 ± 4.4 min and increased to 10.16 ± 3.4 min 4 weeks after ablation with the pacemaker programmed to the rate-modulating minute ventilation mode ($p = 0.016$).

Relation of pacing rate to expired gas exchange and respiratory flow. (Table 2). The relation of the pacing rate to respiratory flow, VO$_2$, VCO$_2$ and respiratory quotient for all patients with the pacemaker programmed to the rate-modulating minute ventilation mode is shown in Table 2. The pacing rate correlated strongly with the expired minute ventilation ($r = 0.89$) (Fig. 1 and 2). Respiratory rate and tidal volume were also linearly related to the change in pacing rate of the pacemaker ($r = 0.84$ and 0.87, respectively). The correlation of pacing rate with expired gas analysis was similar, with correlation coefficients of 0.84 for VO$_2$, 0.87 for VCO$_2$ and 0.89 for respiratory quotient. Changes in transthoracic impedance measured by the pulse generator showed a somewhat stronger correlation with each of the physiologic variables than did the actual pacing rate.
Table 2. Correlation of Pacing Rate (minute ventilation mode) With Physiologic Variables in 10 Patients

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)</th>
<th>Gender</th>
<th>VO₂*</th>
<th>VCO₂*</th>
<th>RQ*</th>
<th>MV*</th>
<th>TV*</th>
<th>RR*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>56</td>
<td>M</td>
<td>0.85</td>
<td>0.87</td>
<td>0.88</td>
<td>0.93</td>
<td>0.89</td>
<td>0.84</td>
</tr>
<tr>
<td>2</td>
<td>74</td>
<td>M</td>
<td>0.82</td>
<td>0.84</td>
<td>0.91</td>
<td>0.95</td>
<td>0.98</td>
<td>0.89</td>
</tr>
<tr>
<td>3</td>
<td>78</td>
<td>F</td>
<td>0.57</td>
<td>0.58</td>
<td>0.61</td>
<td>0.55</td>
<td>0.52</td>
<td>0.37</td>
</tr>
<tr>
<td>4</td>
<td>66</td>
<td>M</td>
<td>0.92</td>
<td>0.94</td>
<td>0.97</td>
<td>0.97</td>
<td>0.99</td>
<td>0.89</td>
</tr>
<tr>
<td>5</td>
<td>23</td>
<td>F</td>
<td>0.86</td>
<td>0.90</td>
<td>0.98</td>
<td>0.94</td>
<td>0.96</td>
<td>0.87</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>M</td>
<td>0.92</td>
<td>0.94</td>
<td>0.79</td>
<td>0.99</td>
<td>0.99</td>
<td>0.91</td>
</tr>
<tr>
<td>7</td>
<td>85</td>
<td>M</td>
<td>0.74</td>
<td>0.83</td>
<td>0.90</td>
<td>0.70</td>
<td>0.56</td>
<td>0.81</td>
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<tr>
<td>8</td>
<td>60</td>
<td>M</td>
<td>0.95</td>
<td>0.95</td>
<td>0.95</td>
<td>0.97</td>
<td>0.98</td>
<td>0.96</td>
</tr>
<tr>
<td>9</td>
<td>60</td>
<td>M</td>
<td>0.83</td>
<td>0.92</td>
<td>0.96</td>
<td>0.93</td>
<td>0.89</td>
<td>0.96</td>
</tr>
<tr>
<td>10</td>
<td>64</td>
<td>M</td>
<td>0.95</td>
<td>0.95</td>
<td>0.95</td>
<td>0.95</td>
<td>0.98</td>
<td>0.92</td>
</tr>
<tr>
<td>Mean</td>
<td>62.6</td>
<td></td>
<td>0.84</td>
<td>0.87</td>
<td>0.89</td>
<td>0.89</td>
<td>0.87</td>
<td>0.84</td>
</tr>
<tr>
<td>±SD</td>
<td>16.7</td>
<td></td>
<td>0.12</td>
<td>0.11</td>
<td>0.11</td>
<td>0.14</td>
<td>0.18</td>
<td>0.17</td>
</tr>
</tbody>
</table>

*All values are Pearson's correlation coefficients; † patients with chronic obstructive pulmonary disease. F = female; M = male; MV = minute ventilation; RQ = respiratory quotient; RR = respiratory rate; TV = tidal volume; VCO₂ = expired carbon dioxide; VO₂ = oxygen consumption.

The correlation coefficient for the relation of the transthoracic impedance estimate of minute ventilation from the pulse generator (rate-response factor) with the actual measured minute ventilation was 0.93. The pacing rate correlated well with physiologic variables of metabolic demand in the two patients with chronic obstructive lung disease (included in Table 2).

Comparison of rate modulation in the minute ventilation pacing mode with sinus rhythm (Table 3). The relation of heart rate to VO₂, VCO₂, respiratory quotient, minute ventilation, tidal volume and respiratory rate was examined during sinus rhythm and in the minute ventilation pacing mode in the six patients with sinus rhythm at the time of the baseline exercise test. The minute ventilation pacing mode provided physiologic rate increases that closely paralleled those observed during exercise in sinus rhythm before catheter ablation (Fig. 3). The mean correlation coefficient for the relation of heart rate to minute ventilation during the baseline exercise test was 0.95 for patients with sinus rhythm (p = NS compared with the minute ventilation pacing mode). The relation of heart rate to VO₂ was also similar in both sinus rhythm and minute ventilation pacing mode (r = 0.85 sinus, r = 0.83 minute ventilation mode, p = NS).

Effect of isoproterenol on pacing rate in the minute ventilation pacing mode (Table 4). The ability of the signal-processing algorithm of the pacemaker to discriminate changes in transthoracic impedance related to respiration from those related to contractility was assessed during isoproterenol infusion. With isoproterenol infusion there

Table 3. Relation of Heart Rate to Physiologic Variables During Exercise. Comparison of Minute Ventilation Pacing Mode With Sinus Rhythm

<table>
<thead>
<tr>
<th></th>
<th>Sinus Rate (n = 6)*</th>
<th>MV Pacing Rate (n = 6)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂</td>
<td>0.85</td>
<td>0.83</td>
</tr>
<tr>
<td>VCO₂</td>
<td>0.89</td>
<td>0.85</td>
</tr>
<tr>
<td>RQ</td>
<td>0.88</td>
<td>0.88</td>
</tr>
<tr>
<td>MV</td>
<td>0.95</td>
<td>0.88</td>
</tr>
<tr>
<td>TV</td>
<td>0.83</td>
<td>0.89</td>
</tr>
<tr>
<td>RR</td>
<td>0.82</td>
<td>0.80</td>
</tr>
</tbody>
</table>

*All values are Pearson's correlation coefficients. Abbreviations as in Table 2.
Figure 3. Patient 4. A. Relation of heart rate (HR) to minute ventilation (MV), oxygen consumption (VO₂), and carbon dioxide production (VCO₂) during sinus rhythm and treadmill exercise testing before catheter ablation of the atrioventricular junction. B. Relation of pacing rate in the minute ventilation mode to the same physiologic variables during treadmill exercise in the same patient after catheter ablation. Note that although the maximal heart rate achieved during the two exercise tests (A and B) was the same, the slope of the heart rate response was steeper in the minute ventilation pacing mode than during sinus rhythm. The slope of the minute ventilation, VO₂, and VCO₂ curves during both tests were similar. Other abbreviations as in Figure 1.

were no significant changes from baseline in pacing rate or telemetered transthoracic impedance estimates of minute ventilation (rate-response factor).

Table 4. Effect of Isoproterenol Infusion on Pacing Rate and Transthoracic Impedance (minute ventilation pacing mode) in 10 Patients

<table>
<thead>
<tr>
<th>Isoproterenol Dose</th>
<th>Pacing Rate (beats/min)</th>
<th>MV-TTI</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>70.5</td>
<td>58.2</td>
<td></td>
</tr>
<tr>
<td>1 µg/min</td>
<td>70.8</td>
<td>58.0</td>
<td>NS</td>
</tr>
<tr>
<td>2 µg/min</td>
<td>72.2</td>
<td>56.6</td>
<td>NS</td>
</tr>
</tbody>
</table>

MV-TTI = transthoracic impedance estimate of minute ventilation telemetered from the pulse generator in the minute ventilation pacing mode (lower values reflect increases in minute ventilation).

Discussion

Relation of pacing rate to physiologic variables. An ideal rate-modulating pacemaker should increase the heart rate in direct proportion to metabolic demand. Previous studies (9–13) of normal volunteers and patients with structural heart disease have indicated a linear correlation between heart rate and minute ventilation during aerobic exercise. The results of the present study suggest that rate-modulated pacemakers that measure transthoracic impedance changes related to respiration increase the pacing rate with exercise in a manner that closely parallels physiologic variables of metabolic demand. The results of linear regression analysis indicate a close correlation between the transthoracic impedance estimate of minute ventilation and the actual measured minute ventilation during exercise (r = 0.93). The rate-modulating algorithm of the pacemaker provided increases in heart rate that were highly correlated with VO₂, VCO₂, and minute ventilation. As observed in previous studies (2–6) of rate-modulated pacing, the ability to increase the heart rate in response to metabolic demand resulted in clinically significant improvements in exercise capacity and oxygen consumption as compared with values obtained with the fixed rate pacing mode.

Factors influencing the relation of minute ventilation to oxygen consumption. Minute ventilation increases during exercise in parallel with increases in carbon dioxide production (VCO₂) (19). Oxygen consumption (VO₂) is also linearly related to minute ventilation at work loads below anaerobic threshold (11). However, several factors may distort the relation of minute ventilation to oxygen consumption. Because the kinetics of VCO₂ are slower than for VO₂ (9,20), changes in work load result in a transient dissociation of these variables until a new steady state can be reached. At work loads exceeding anaerobic threshold, buffering of lactic acid production results in disproportionate increases in VCO₂ and minute ventilation compared with VO₂. Differences in substrate utilization during exercise affect the relation of VCO₂ and minute ventilation to VO₂. For example, metabolism of fat (respiratory quotient 0.7) results in a lower ratio of minute ventilation to VO₂ than does metabolism of carbohydrate (respiratory quotient 1.0). The pulmonary function of the individual also has an impact on the relation of minute ventilation to VO₂. Patients with an increased ratio of ventilatory dead space to tidal volume have a higher level of minute ventilation in relation to VO₂ than that observed for individuals with greater ventilatory efficiency (19). The "set point" for the arterial concentration of carbon dioxide is an important factor relating minute ventilation to VO₂, with greater respiratory flow required for maintenance of lower arterial carbon dioxide concentrations (21). Because the fitness of the individual and the presence of cardiac disease determine anaerobic threshold (12), these
factors have important effects on the relation of \( \dot{V}CO_2 \) and minute ventilation to \( VO_2 \).

Comparison of heart rate during rate-modulated pacing with sinus rhythm. An important finding of this study is the favorable comparison of the rate-modulating minute ventilation pacing mode with that observed during sinus rhythm in the same individuals. The correlation of heart rate with minute ventilation and \( VO_2 \) was similar for patients during sinus rhythm and in the minute ventilation pacing mode. It should be emphasized that the slope of the increase in heart rate during VVIR pacing was somewhat different from that during sinus rhythm for each patient (Fig. 3). Despite these differences in the slope of the heart rate response, the exercise capacity and maximal \( VO_2 \) observed with the minute ventilation pacing mode exceeded those observed during exercise testing before ablation of the AV junction in the patient’s intrinsic heart rhythm. The maximal heart rate achieved with the pacemaker was very similar to that observed during the baseline exercise test in sinus rhythm. However, improvements in exercise capacity and quality of life after catheter ablation of the AV junction and rate-modulating pacemaker implantation are not specific to the minute ventilation pacemaker, having been observed with rate-modulating pacemakers that sense activity as well (18). This finding probably relates to several factors, including withdrawal of antiarrhythmic drugs and improved physical conditioning after control of symptomatic atrial fibrillation with catheter ablation.

Discrimination of changes in impedance related to respiration from those related to stroke volume. Because transthoracic impedance is related to both right ventricular stroke volume and respiration, the ability to discriminate between these factors has important implications for the design of pacing systems. The fact that isoproterenol infusion produced no changes in pacing rate or telemetered transthoracic impedance suggests that the signal-processing circuit of the present device can discriminate between these two components of the impedance signal. However, this conclusion must be qualified because right ventricular stroke volume was not directly measured.

Limitations. Several important limitations of this study should be emphasized. First, the study protocol included a relatively small series of patients. Second, the exercise capacity of patients in the minute ventilation pacing mode cannot be directly compared with that observed in the intrinsic cardiac rhythm before catheter ablation because the baseline exercise tests were performed while patients were receiving therapy with digoxin and type I antiarrhythmic drugs for disabling atrial arrhythmias. After ablation of the AV junction, tachyarrhythmias were under control in all patients and they were able to discontinue antiarrhythmic drugs. Thus, changes in exercise capacity before and after ablation are likely to be related to several factors that are independent of the pacing system. Third, comparison of the relation of heart rate response to physiologic indicators of metabolic demand during the minute ventilation pacing mode with that observed during sinus rhythm must be interpreted cautiously because the study group consisted of patients with paroxysmal atrial arrhythmias. Although all patients achieved a maximal heart rate of \( \geq 85\% \) of the age-predicted maximum during exercise, the chronotropic competence of the sinus node in these patients may not have been normal. However, the close correlation of the sinus rate with measured minute ventilation suggests that the heart rate response of the study group was well preserved. Finally, the apparent specificity of the signal-processing algorithm of the pacemaker for changes in transthoracic impedance related to respiration was measured indirectly, without direct measurements of right ventricular function.

Conclusion. The results of this prospective study suggest that rate-modulating pacemakers based on minute ventilation measured by transthoracic impedance provide increases in heart rate that closely parallel physiologic variables of metabolic demand during exercise.

References


