Hemodynamic Results of Right Atrial Pacing in
33 Normal Subjects*

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In the course of studies on the physiologic bases of electronic pacing, the effect of increments in heart rate upon cardiovascular dynamics in normal and cardiac subjects was studied. The rate increases were achieved by bipolar right atrial catheter pacing at rates above the control sinus rhythm heart rates. Prior studies from this laboratory had demonstrated the physiologic superiority of atrial and sequential atroventricular pacing to ventricular pacing. Previous studies on the effect of increments in heart rate by right atrial pacing, on cardiac output had resulted in somewhat divergent results. The purpose of this paper is to report our results in 33 normal subjects.

Methods and Materials

A bipolar electrode catheter was passed into the right atrium to permit control of the heart rate (at levels above the initial sinus rhythm) with a Medtronic paired pulse stimulator or a standard Medtronic pacemaker. A second catheter, usually a 100 cm long Lehman catheter, was passed into the right heart to permit right heart pressure determination and to serve as a vehicle for indocyanine green administration for indicator dilution cardiac output determination. For the latter purpose, blood was aspirated from a systemic artery via an indwelling Courand needle using a Harvard constant infusion-withdrawal apparatus and a Gilford densitometer. The dye curves were recorded on an 8 channel Electronics for Medicine recorder at a paper speed of 10 mm/sec. The Hamilton-Stewart semilogarithmic replots were used to calculate cardiac output. Three dye-in-blood concentration levels were used to calibrate the dye curves. Output determination was usually made at two minutes after institution of a given atrial pacing rate. Previous studies from this laboratory have proved that a stable cardiac output is achieved by this time.

Control output and pressure data are available in 33 subjects adjudged to be normal after complete study, in 73 patients with rheumatic heart disease, or congenital heart disease without intracardiac or extracardiac shunts, and in 63 subjects with pulmonary emphysema for a total of 169 in all. The data in the two patient groups will be presented elsewhere. After control data were obtained, repeat studies were made at rates above the control sinus rate. The data were then analyzed in four groups: control heart rate levels, and rates of 60-89, 90-109 and 110-140 during atrial pacing. Not all subjects were investigated at all three pacing rates. Six of the normal subjects were studied at control rates and all three experimental rate levels.

Results

The data in six normal subjects studied with control data and during all three pacing rates of 60-89, 90-109 and 110-140 are shown in Table 1. The average ventricular rate, cardiac index and stroke volume in the control group are 68 beats per minute, 2.65 L/min/M² BSA, and 69 ml/beat; the corresponding data in the 60-89 group are 80, 2.90 and 64; the corresponding data in the 90-109 group are 100, 3.09 and 55; the corresponding data in the 110-140 group are 113, 3.01 and 47. The cardiac index difference between the control and the 60-89 rate group is significant at the 0.05 level, P=0.05. The same is true...
for the cardiac index difference between the control data and the data at rates 90-
109 and control levels and indices at rates 110-140. The indices at rates 60-89 versus
90-109 show no significant difference from zero; the same is true of comparison of the
index data at rates 60-89 versus 110-140 and 90-109 versus 110-140. Thus, the in-
dices rise slightly but significantly when control data (average heart rate 68) are
compared to atrial pacing average rates of 80, 100 and 113 respectively, but no further
rise occurs when the 60-89 index level is compared to the two higher rates or the
90-109 index level is compared to the 110-
140 level. The increases in rate from one
group to another faster rate group are gen-
erally, of course, significant as are the stroke volume decreases as the rate rises. The
decrements in stroke volume demonstrate that the cardiac output rises less propor-
tionately than the rate increments as the heart rate is elevated by atrial pacing.

In nine normal subjects, comparison of heart rate, cardiac index and stroke vol-
ume are available under control conditions

### Table 1—Hemodynamic Data in 6 Normal Subjects

<table>
<thead>
<tr>
<th>Cath No.</th>
<th>Control (VR, CI, SV)</th>
<th>60-89 (VR, CI, SV)</th>
<th>90-109 (VR, CI, SV)</th>
<th>110-140 (VR, CI, SV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1223</td>
<td>66 VR, 6 CI, 69 SV</td>
<td>78 VR, 2.50 CI, 60 SV</td>
<td>102 VR, 2.72 CI, 50 SV</td>
<td>120 VR, 2.62 CI, 41 SV</td>
</tr>
<tr>
<td>1244</td>
<td>69 VR, 2.83 CI, 78 SV</td>
<td>84 VR, 3.12 CI, 70 SV</td>
<td>105 VR, 3.02 CI, 54 SV</td>
<td>115 VR, 2.92 CI, 48 SV</td>
</tr>
<tr>
<td>1575</td>
<td>66 VR, 3.16 CI, 80 SV</td>
<td>88 VR, 3.40 CI, 70 SV</td>
<td>100 VR, 3.02 CI, 50 SV</td>
<td>110 VR, 3.32 CI, 50 SV</td>
</tr>
<tr>
<td>1579</td>
<td>71 VR, 2.21 CI, 54 SV</td>
<td>78 VR, 2.36 CI, 52 SV</td>
<td>101 VR, 2.97 CI, 51 SV</td>
<td>110 VR, 2.79 CI, 44 SV</td>
</tr>
<tr>
<td>1603</td>
<td>74 VR, 2.78 CI, 66 SV</td>
<td>78 VR, 2.88 CI, 65 SV</td>
<td>100 VR, 3.33 CI, 59 SV</td>
<td>120 VR, 3.18 CI, 47 SV</td>
</tr>
<tr>
<td>1634</td>
<td>64 VR, 2.47 CI, 65 SV</td>
<td>80 VR, 3.13 CI, 66 SV</td>
<td>90 VR, 3.46 CI, 65 SV</td>
<td>100 VR, 3.22 CI, 54 SV</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td>68 VR, 2.65 CI, 69 SV</td>
<td>80 VR, 2.90 CI, 64 SV</td>
<td>100 VR, 3.09 CI, 55 SV</td>
<td>113 VR, 3.01 CI, 47 SV</td>
</tr>
</tbody>
</table>

and atrial pacing rates of 60-89. The control rate is 68 and the experimental atrial
pacing rate is 80, P=0.001. The control cardiac index is 2.63, that during atrial
pacing 2.94, a difference significant at the 0.05 level. The control stroke volume is 68,
that during pacing 64; this latter difference is not significant.

In 27 normal subjects, similar data are
available for comparison of control data and
atrial pacing data at rate 90-109. The con-
trol average atrial rate, index and stroke vol-
ume is 78, 2.92 and 66 respectively. The
corresponding data during atrial pacing are
99, 3.26 and 57. All three parameters are
significantly different at the 0.001 P level.
Comparison of control data and atrial pac-
ing data at rate 110-140 are also available in
27 normal subjects. The control and pacing
data (rate, index and stroke volume) are
81, 3.17 and 65, and 116, 3.31 and 49 re-
spectively. All three parameters again differ at the 0.001 P level. The control car-
diac index data is therefore significantly
lower than the data during each of the
three levels of atrial pacing.

### Table 2—Hemodynamic Data in 33 Normal Subjects

<table>
<thead>
<tr>
<th>Group</th>
<th>VR (L/Min/M²)</th>
<th>CI</th>
<th>SV (ml)</th>
<th>VR (L/Min/M²)</th>
<th>CI</th>
<th>SV (ml)</th>
<th>VR (L/Min/M²)</th>
<th>CI</th>
<th>SV (P Values)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.</td>
<td>68 VR, 2.63 CI, 68 SV</td>
<td>80 VR, 2.94 CI, 64 SV</td>
<td>0.001</td>
<td>0.05</td>
<td>.........</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B.</td>
<td>78 VR, 2.92 CI, 66 SV</td>
<td>99 VR, 3.26 CI, 57 SV</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.</td>
<td>81 VR, 2.97 CI, 65 SV</td>
<td>116 VR, 3.31 CI, 49 SV</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D.</td>
<td>81 VR, 3.04 CI, 66 SV</td>
<td>100 VR, 3.18 CI, 57 SV</td>
<td>0.001</td>
<td>.........</td>
<td>0.05</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E.</td>
<td>80 VR, 2.90 CI, 64 SV</td>
<td>113 VR, 3.01 CI, 47 SV</td>
<td>0.001</td>
<td>.........</td>
<td>0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F.</td>
<td>100 VR, 3.19 CI, 55 SV</td>
<td>116 VR, 3.16 CI, 47 SV</td>
<td>0.001</td>
<td>.........</td>
<td>0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

A. Control Data Vs. Atrial Pacing Rate 60-89: 9 Subjects
B. Control Data Vs. Atrial Pacing Rate 90-109: 27 Subjects
C. Control Data Vs. Atrial Pacing Rate 110-140: 27 Subjects
D. Atrial Pacing Rates 60-89 Vs. 90-109: 8 Subjects
E. Atrial Pacing Rates 60-89 Vs. 110-140: 6 Subjects
F. Atrial Pacing Rates 90-109 Vs. 110-140: 22 Subjects

*Signifies no significant difference.*
In eight normal subjects, the mean heart rates, cardiac indices, and stroke volumes during rates of 60-89 were 81, 3.04 and 66; the corresponding data at rates 90-109 are 100, 3.18 and 57. The rates differ significantly, P=0.001, the stroke volume differs at the 0.05 level, but there is no difference in the cardiac index level. In six normal subjects, corresponding data at rates of 60-89 and 110-140 are 80, 2.90 and 64, and 113, 3.01 and 47 at rates of 110-140. The heart rate and stroke volume data are significantly different, P=0.001, but the index difference is not significantly different from zero. In 22 normal subjects, the corresponding data at rates of 90-109 average 100, 3.19 and 55, and at rates of 110-140, 116, 3.16 and 47. The rates and stroke volume are significantly different, P=0.001, but the indices are not different in the two rate levels.

In summary, therefore, the control cardiac indices in all four sets of observations outlined above are significantly lower than those of the atrial pacing rates, but the lower rate atrial pacing indices fail to rise further as the atrial pacing rate is increased. The overall data are summarized in Table 2.

**Discussion**

Ross et al studied the hemodynamic effects of heart rate changes in man induced by electrical stimulation of the right atrium at a rate exceeding that of the sinus node. Of the 17 patients investigated, six were considered to have only functional heart murmurs after detailed study. The data for the normal subjects above are not available in Ross' paper, but for the group as a whole, an increase in heart rate from 80 to 121 beats/min resulted in virtually no output changes, 3.67 to 3.72 L/min/M². Further increases in the heart rate to an average of 148 resulted in a slight fall in flow to 3.21 L/min/M². Stein et al studied ten normal men by right atrial pacing at rates above the sinus rate. The individual data are not given in Stein's paper, but the graphed data reveal little increase in output as the rate is increased. Benchimol and Liggett analyzed data in eight normal subjects during right atrial pacing and observed a slight initial fall in output followed by a slight rise to levels above the control level as the atrial stimulation rate was further increased. Again, the data are available only in graphic form. A secondary fall in output was observed at rates above 150/min. Cobb et al increased the resting heart rate by right atrial pacing in 18 normal subjects. As the heart rate rose from 68 to 104/min, cardiac output rose 5 to 63 per cent, average 0.9 L/min/M². Further rate increments produced no further alterations in output. Continuation of atrial pacing for 10-50 minutes demonstrated persistent elevation of output above control levels; output returned to control levels promptly after cessation of atrial pacing. Afonso et al noted an 11 per cent increase in cardiac output in ten dogs studied during control heart rates (average 77/min) and tachycardia induced by right atrial stimulation, average rate of 179/min. Warren et al investigated the effect of heart rate increments produced by intravenous atropine administration in six normal men. The control cardiac index of 2.9 L/min/M² rose to an average level of 5.1 within three to five minutes after drug administration.

The data obtained in the present study demonstrate a small but significant increment in cardiac output as heart rate is increased by right atrial pacing in normal subjects. Further increases in heart rate produced by right atrial pacing are, however, not accompanied by further flow increments. A plateau in the relationship between cardiac index and heart rate is thus produced early in the course of right atrial stimulation. These observations demonstrate that cardiac output is in part controlled by the level of the heart rate as postulated by Rushmer.¹¹

**Summary**

The effect of increasing heart rate above control sinus rate was studied in 33 normal subjects by right atrial bipolar electrode catheter pacing. At all atrial pacing rates, significant cardiac output increments are
noted compared to the outputs during the control sinus rate. Further heart rate elevation produced by electrical pacing fails to elevate cardiac output above the levels noted at the lower atrial pacing rates.

Resumen
El efecto de la aceleración del ritmo cardíaco por encima del ritmo sinusal de control ha sido estudiado en 33 sujetos normales, mediante la regulación por medio del catéter electrónico bipolar situado en la aurícula derecha.

El ritmo atrial inducido determinó niveles de rendimiento cardíaco superiores a los observados con el ritmo sinusal de control. La aceleración del ritmo producido por regulación eléctrica no determinó un rendimiento cardíaco mayor al obtenido con ritmos mas lentos de regulación atrial.

Résumé
L’effet produit en accélérant le rythme cardiaque au-dessus du rythme basal du sinus a été étudié chez 33 sujets normaux au moyen d’une stimulation par cathéter bipolar dans l’oreillette droite. A tous les rythmes de stimulation de l’oreillette, on a remarqué des augmentations significatives du débit cardiaque lorsqu’on les compare aux débits pendant le ritmo sinusal de contrôle; mais une augmentation ultérieure du rythme par stimulation électrique n’èléve pas le débit cardiaque au-dessus des niveaux notés à des rythmes auriculaires obtenus par stimulations moins rapides.

Zusammenfassung

References

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