The Changes in Cardiac Output with Reversion of Atrial Fibrillation to Sinus Rhythm

By James J. Morris, Jr., M.D., Mark Entman, M.D., William C. North, M.D., Yihong Kong, M.D., and Henry McIntosh, M.D.

ONE of the aims in reverting atrial fibrillation to sinus rhythm is to improve cardiac function. Previous studies comparing the cardiac output during atrial fibrillation and following the restoration of sinus rhythm have demonstrated no change to as much as 140 per cent increase. Because of the experimental design of these studies, the validity of the observation that an increase in cardiac output was due to change in rhythm alone is questionable. It therefore seemed timely to restudy the changes in cardiac output with the change from atrial fibrillation to sinus rhythm under resting and exercise conditions. By use of the technic of cardioversion and with modification of the experimental design some of the objections of former studies can be overcome, and the observation can be more readily attributed to changes in rhythm alone.

Method

Twelve patients were included in this study. The clinical information pertinent to the group is shown in Table 1. The age of the patients ranged from 19 to 66 years. Six had rheumatic heart disease, five ischemic heart disease, and one postoperative congenital heart disease. The duration of the episode of atrial fibrillation treated in this study varied from 4 months to 17 years. All were on steady daily doses of various digitalis preparations and had adequate control of the ventricular rate. The functional classifications (New York Heart Association) were as follows: two patients class I, six patients class II, and four patients class III. The patients were observed in the hospital several days after attaining a stable weight and being edema-free. All were free from clinical evidence of congestive heart failure at rest during this period. Hyperthyroidism was excluded in each patient by suitable laboratory tests.

Cardioversion, as described by Low and associates was used to revert atrial fibrillation to sinus rhythm. Quinidine therapy for maintenance of normal sinus rhythm was not instituted before or after reversion to sinus rhythm until the completion of our observations. Cardioversion was accomplished with a commercially available direct-current defibrillator.* The procedure was carried out under light thiobarbiturate anesthesia (sodium thiopental); the dosage varied from 160 to 300 mg. In eight patients succinyl choline was also used in doses from 60 to 160 mg. The time of anesthesia, from induction to full recovery, varied from 12 to 30 minutes, the average time being 20.5 minutes.

Nothing by mouth was allowed for 7 hours prior to the study. No medications other than the anesthetic agents were employed. The following protocol was used in each study. From hour 0 to 1 routine right heart catheterization was performed with positioning of the venous catheter in the main pulmonary artery. The patient was then allowed 2 hours of undisturbed bed rest. At hours 3 to 3% metabolic rates, oxygen consumption, and cardiac outputs were determined. The patient was then anesthetized and reverted from atrial fibrillation to sinus rhythm. After full recovery from anesthesia the patient was again allowed 2 hours of undisturbed bed rest. At hours 6 to 6% repeat metabolic rates and oxygen consumption were determined. When these measurements were stable and equal to those recorded in the morning period (hours 3 to 3%) repeat observations were made of metabolic rate, oxygen consumption, and cardiac output.


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From the Cardiovascular Laboratory, Department of Medicine and the Department of Anesthesiology, Duke University Medical Center, Durham, North Carolina.

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Eleven patients were studied by this protocol while in a resting state; in four patients the observations were also carried out with exercise before and after reversion of atrial fibrillation to sinus rhythm. One patient was studied during exercise alone. The exercise studies were done according to the same protocol, with the additional observations of metabolic rate, oxygen consumption, and cardiac output at exercise during hours 3 to 3% while in atrial fibrillation and hours 6 to 6% while in sinus rhythm. The oxygen consumption recorded during steady-state exercise in the morning period was duplicated by adjusting the workload in the afternoon study, at which time the cardiac output was determined.

Cardiac output was determined in all studies by the Fick method, with the pulmonary and radial artery as the sampling sites. Blood samples were analyzed for oxygen content by the spectrophotometric method of Hickam and Frayzer. Actual determinations of the oxygen consumption were done by collecting Douglas bag samples for 2-minute periods with exercise and 3-minute periods during rest. To estimate the oxygen consumption and metabolic rates and thus to allow determinations of cardiac outputs at comparable levels of oxygen consumption, consecutive intervals were monitored in the morning and afternoon sessions, both for the minute ventilation and the oxygen content of expired air by an oxygen analyzer. The readings were taken at 30-second intervals and by use of an analog computer, the oxygen consumption and the metabolic rate were determined immediately at each 30-second interval. These approximations were used to determine when oxygen consumption was at the desired level; then the pulmonary and radial artery samples as well as the expired air were collected for precise calculation of the arteriovenous oxygen difference and oxygen consumption. Under these conditions both resting and exercise levels of oxygen consumption were very similar in the studies both before and after reversion.

The reported determinations of cardiac outputs are the average of duplicate or quadruplicate determinations of the arteriovenous oxygen difference and the oxygen consumption. Exercise studies were performed during supine bicycle pedaling. At least 3 minutes of stable oxygen consumption were observed before actual determinations of cardiac output. To allow sufficient leeway for adjustment of oxygen consumption in the afternoon to that recorded in the morning the exercise period was actually 12 to 15 minutes in both the morning and the afternoon periods, with the samples being collected in the last 4 minutes. In this manner slight adjustments of the workload could be made during the afternoon study in order to give a stable level of oxygen consumption equal to that recorded in the morning and with time remaining for at least a 3-

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**Clinical Information**

<table>
<thead>
<tr>
<th>Pt. no.</th>
<th>Age</th>
<th>Race, sex</th>
<th>Diagnosis</th>
<th>Functional class</th>
<th>Duration of atrial fibrillation</th>
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<tbody>
<tr>
<td>1</td>
<td>53</td>
<td>C, F</td>
<td>HVD</td>
<td>I</td>
<td>4 yr.</td>
</tr>
<tr>
<td>2</td>
<td>43</td>
<td>C, M</td>
<td>HVD</td>
<td>II</td>
<td>6 mo.</td>
</tr>
<tr>
<td>3</td>
<td>49</td>
<td>C, M</td>
<td>IHD</td>
<td>II</td>
<td>6 mo.</td>
</tr>
<tr>
<td>4</td>
<td>25</td>
<td>C, M</td>
<td>RHD-MS (post-op.)</td>
<td>II</td>
<td>5 yr.</td>
</tr>
<tr>
<td>5</td>
<td>34</td>
<td>N, F</td>
<td>RHD-MI (severe)</td>
<td>III</td>
<td>15 mo.</td>
</tr>
<tr>
<td>6</td>
<td>38</td>
<td>C, M</td>
<td>RHD-MS (severe)</td>
<td>III</td>
<td>1 yr.</td>
</tr>
<tr>
<td>7</td>
<td>44</td>
<td>C, F</td>
<td>RHD-MS (severe)</td>
<td>III</td>
<td>2 yr.</td>
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<tr>
<td>8</td>
<td>41</td>
<td>C, M</td>
<td>RHD-MS (post-op.)</td>
<td>I</td>
<td>2 yr.</td>
</tr>
<tr>
<td>9</td>
<td>66</td>
<td>N, M</td>
<td>RHD-MI (moderate)</td>
<td>III</td>
<td>17 yr.</td>
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<tr>
<td>10</td>
<td>52</td>
<td>N, M</td>
<td>HVD</td>
<td>II</td>
<td>1 yr.</td>
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<tr>
<td>11</td>
<td>59</td>
<td>N, M</td>
<td>IHD</td>
<td>II</td>
<td>1 yr.</td>
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<tr>
<td>12</td>
<td>19</td>
<td>C, M</td>
<td>CHD (post-op.)</td>
<td>II</td>
<td>4 mo.</td>
</tr>
</tbody>
</table>

Abbreviations. Race: C, Caucasian; N, Negro. Sex: M, male; F, female. Diagnosis: HVD, hypertensive vascular disease; IHD, ischemic heart disease; RHD, rheumatic heart disease; MS, mitral stenosis; MI, mitral insufficiency; TS tricuspid stenosis; AI, aortic insufficiency; CHD, congenital heart disease.

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*Model E2 O₂ analyzer, Beckman Instruments, Inc., Fullerton, California.

*Circulation, Volume XXXI, May 1965
minute period of stable exercise before the oxygen consumption was determined.

Results

Oxygen Consumption and Metabolic Rate

Oxygen consumption and metabolic rate during rest with atrial fibrillation and after restoration of sinus rhythm were similar (table 2). Differences in metabolic rates in individual patients before and after cardioversion were within ±10 per cent in seven patients and ±15 per cent in 10 patients. In the five patients studied during supine exercise, first in atrial fibrillation and then sinus rhythm, again the values for oxygen consumption and metabolic rate changed little with reversion to sinus rhythm (table 3). All five patients had metabolic rates within ±20 per cent before and after restoration of sinus rhythm. Individual comparisons of patients are shown in figure 1.

It should be recalled that these determinations of metabolic rate were not recorded under “basal conditions.” In fact, the initial observations were purposely recorded in a condition that would allow duplication of the metabolic rate in the postcardioversion study.

Heart Rate

The heart rates shown in figure 1 are the actual 2-minute averages of the ventricular rate recorded electrocardiographically during the determinations of oxygen consumption. In sinus rhythm, therefore, this represents the true rate of effective systoles per minute, whereas in atrial fibrillation, because of a slight pulse deficit, the reported heart rates may be slightly higher than the number of effective ventricular systoles. The peripheral pulse was monitored by manual palpation in several instances and pulse deficits of 5 to 10 per minute at rest and approxi-

<table>
<thead>
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<th>Rest Studies</th>
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<tbody>
<tr>
<td>Pt. no.</td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>1</td>
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<tr>
<td></td>
</tr>
<tr>
<td>2</td>
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<tr>
<td></td>
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<tr>
<td>3</td>
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<tr>
<td></td>
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<tr>
<td>4</td>
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<td>11</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Average</td>
</tr>
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<td></td>
</tr>
</tbody>
</table>

“P” value >5% >5% <5% <5% <5% <5% >5% <1%

AF, atrial fibrillation; SR, sinus rhythm; PA, pulmonary artery; AO systemic artery; AV arteriovenous; P value comparison AF and SR.

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Exercise Studies*

<table>
<thead>
<tr>
<th>Pt. no.</th>
<th>Rhythm</th>
<th>Oxygen consumption, ml./min./m²</th>
<th>Metabolic rate, %</th>
<th>Ventricular rate, beats/min.</th>
<th>Cardiac output, L./min.</th>
<th>Cardiac index, L./min./M.²</th>
<th>Oxygen content, vol. per cent</th>
<th>AV O₂ diff., vol. %</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>AF</td>
<td>785 (454)</td>
<td>+284</td>
<td>131</td>
<td>7.3</td>
<td>4.2</td>
<td>7.26</td>
<td>18.02</td>
</tr>
<tr>
<td></td>
<td>SR</td>
<td>753 (435)</td>
<td>+269</td>
<td>132</td>
<td>8.4</td>
<td>4.9</td>
<td>7.19</td>
<td>16.12</td>
</tr>
<tr>
<td>2</td>
<td>AF</td>
<td>1295 (578)</td>
<td>+340</td>
<td>98</td>
<td>10.2</td>
<td>4.5</td>
<td>5.51</td>
<td>18.21</td>
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<tr>
<td></td>
<td>SR</td>
<td>1339 (598)</td>
<td>+355</td>
<td>128</td>
<td>11.9</td>
<td>5.3</td>
<td>6.47</td>
<td>17.69</td>
</tr>
<tr>
<td>3</td>
<td>AF</td>
<td>1265 (586)</td>
<td>+353</td>
<td>139</td>
<td>10.7</td>
<td>4.9</td>
<td>9.13</td>
<td>21.03</td>
</tr>
<tr>
<td></td>
<td>SR</td>
<td>1280 (593)</td>
<td>+359</td>
<td>122</td>
<td>12.2</td>
<td>5.6</td>
<td>9.59</td>
<td>20.13</td>
</tr>
<tr>
<td>5</td>
<td>AF</td>
<td>1048 (557)</td>
<td>+352</td>
<td>86</td>
<td>8.3</td>
<td>4.4</td>
<td>4.12</td>
<td>16.75</td>
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<tr>
<td></td>
<td>SR</td>
<td>995 (529)</td>
<td>+332</td>
<td>98</td>
<td>9.6</td>
<td>5.1</td>
<td>6.43</td>
<td>16.87</td>
</tr>
<tr>
<td>12</td>
<td>AF</td>
<td>616 (346)</td>
<td>+142</td>
<td>98</td>
<td>5.3</td>
<td>3.0</td>
<td>9.60</td>
<td>21.30</td>
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<tr>
<td></td>
<td>SR</td>
<td>573 (322)</td>
<td>+125</td>
<td>110</td>
<td>6.9</td>
<td>3.9</td>
<td>11.75</td>
<td>20.12</td>
</tr>
<tr>
<td>Average</td>
<td>AF</td>
<td>1002</td>
<td>+294</td>
<td>110</td>
<td>8.36</td>
<td>4.2</td>
<td>7.12</td>
<td>19.06</td>
</tr>
<tr>
<td></td>
<td>SR</td>
<td>988</td>
<td>+288</td>
<td>118</td>
<td>9.80</td>
<td>4.96</td>
<td>8.29</td>
<td>18.19</td>
</tr>
</tbody>
</table>

*Same abbreviations as in table 3.

The average oxygen consumption per minute at rest and at exercise was approximately 10 to 20 beats per minute at exercise.

At rest, after cardioversion nine patients increased their heart rate, one remained unchanged, and one decreased the ventricular rate (table 2). While the differences in ventricular rates were significant ($p<0.05$) at rest, and not statistically significant at exercise, there was no relationship between the change in heart rate and cardiac output.

![Graph](https://example.com/graph1.png)

**Figure 1**

Oxygen consumption (left) and ventricular rate (right) before and after cardioversion in 16 patients. The lower 11 values are the resting observations. The upper five points are the exercise values. The means of each group are shown. The lines connect the same patients before and after restoration of sinus rhythm.

Arterial and Pulmonary Artery Oxygen Content

At rest five patients had an increase in arterial oxygen content following cardioversion and five had a decrease, and in one patient it was unchanged after cardioversion (table 2). The changes were less than 1.0 volume per cent in eight of the 11 patients, two decreased oxygen content more than 1 volume per cent, and one patient increased oxygen content 1.8 volumes per cent.

Nine of the 11 patients had an increase in pulmonary artery oxygen content after restoration of sinus rhythm. One showed no change, and one patient a decrease of 0.95 volume per cent.

Ten of the 11 patients showed a decrease in arteriovenous oxygen difference with restoration of sinus rhythm, one patient (no. 8) showed an increase in arteriovenous oxygen difference of 0.41 volume per cent (table 2).

At exercise the somewhat higher average arterial oxygen content with atrial fibrillation than with sinus rhythm was not of statistical significance by analysis of variance although the $p$ value was $<0.05$. The arteriovenous oxygen difference fell significantly with change to sinus rhythm ($p<0.01$). This narrowing of the arteriovenous oxygen difference was made up almost equally from...
Cardiac output in 11 patients studied at rest first in atrial fibrillation and then in sinus rhythm. The first patient showed a decrease in cardiac output, the next three did not show significant changes. The remaining seven patients increased cardiac output 0.6 L./min. or greater. The functional classification is listed below each individual patient.

Cardiac Output

If ± 0.5 L./min. is assumed to be a significant change in cardiac output, then seven of the 11 patients had a significant rise in cardiac output of from 0.6 to 3.2 L./min. (fig. 2); one patient (no. 8) had a decrease in cardiac output of 0.6 L./min.; and three patients (nos. 6, 9, and 11) had no significant change.

Five patients were studied during exercise before and after the restoration of sinus rhythm (fig. 3): all had an increase in cardiac output. All four patients in whom both resting and exercise observations were made before and after restoration of sinus rhythm had an increase of cardiac output both at rest and during exercise (patients 1, 2, 3, and 5). The percentage increase in cardiac output with restoration of sinus rhythm was greater in three patients during rest than at exercise and greater in one patient during exercise. One patient (no. 12) was studied only at exercise. Unfortunately, none of the four patients failing to show increase in cardiac output at rest was studied at exercise.

Cardiac Index, Functional Classification, and Basal Metabolic Rate

In general, in this small series the higher values for resting oxygen consumptions were associated with higher cardiac indices. This rule was modified by the functional classifications. The two patients recorded as functional class I had the highest resting cardiac indices, and three of the lowest four cardiac indices were recorded in the patients in class III, despite roughly equal oxygen consumptions.

The only patient decreasing cardiac output (no. 8) with restoration of sinus rhythm was in functional class I and had the highest resting cardiac index. Two of the three patients who showed no significant change in cardiac index with the restoration of sinus...
rhythm were in functional class III and the other was in class II. Of the seven patients who increased cardiac output with the restoration of sinus rhythm one was in functional class I, two were in functional class III, and four were in functional class II.

The duration of the atrial fibrillation, the etiology of the heart disease, the time of anesthesia, and the number or energy of the countershocks did not appear to influence the results.

**Discussion**

Previous studies have shown that restoration of sinus rhythm resulted in an increase in cardiac output in a majority of patients. Table 4 shows the results of various studies in the literature. The acceptance of these observations has been questioned for several reasons. First, in almost all the studies quinidine has been the agent used for restoration of sinus rhythm. Not only are the results of studies of the influence of quinidine on cardiac output variable, but some authors have even suggested that the observed increase in cardiac output with restoration of sinus rhythm by quinidine was due to the drug’s action on the peripheral circulation.\(^6\) Secondly, by the nature of the method of quinidine conversion the observations first in

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**Table 4**

*Changes in Cardiac Output with Reversion of Atrial Fibrillation to Sinus Rhythm*

<table>
<thead>
<tr>
<th>Author and reference</th>
<th>Year</th>
<th>Method of estimating cardiac output</th>
<th>No. patients</th>
<th>Change in CO(^+)</th>
<th>No. patients with significant CO increase(^+)</th>
<th>Comment</th>
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</thead>
<tbody>
<tr>
<td>Meakins(^1)</td>
<td>1923</td>
<td>Carbon dioxide</td>
<td>1</td>
<td>—</td>
<td>+40%</td>
<td>1</td>
</tr>
<tr>
<td>Smith(^2)</td>
<td>1930</td>
<td>Carbon dioxide</td>
<td>3</td>
<td>0 to 30%</td>
<td>+40%</td>
<td>2 of 3</td>
</tr>
<tr>
<td>Kerkhof(^3)</td>
<td>1936</td>
<td>Acetylene</td>
<td>8</td>
<td>+14 to 36%</td>
<td>+25%</td>
<td>6 of 8</td>
</tr>
<tr>
<td>Hecht(^4)</td>
<td>1951</td>
<td>Fick</td>
<td>14</td>
<td>?</td>
<td>Approx. 1/3</td>
<td></td>
</tr>
<tr>
<td>Kory(^5)</td>
<td>1951</td>
<td>Fick</td>
<td>8</td>
<td>0 to ?</td>
<td>+32%</td>
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<tr>
<td>Hansen(^6)</td>
<td>1952</td>
<td>Indicator dilution</td>
<td>14</td>
<td>—12 to 135%</td>
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<td>Storstein(^7)</td>
<td>1955</td>
<td>Fick</td>
<td>5</td>
<td>—4 to +22%</td>
<td>+11%</td>
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<tr>
<td>Broch(^8)</td>
<td>1957</td>
<td>Fick</td>
<td>20</td>
<td>?</td>
<td>+21%</td>
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<td>Gilbert(^9)</td>
<td>1963</td>
<td>Indicator dilution</td>
<td>3</td>
<td>+14 to 57%</td>
<td>+42%</td>
<td>2 of 3</td>
</tr>
<tr>
<td>Oram(^10)</td>
<td>1963</td>
<td>Fick</td>
<td>10</td>
<td>—46 to 30%</td>
<td>+140%</td>
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<tr>
<td>Graettinger(^11)</td>
<td>1963</td>
<td>Fick and indicator dilution</td>
<td>18</td>
<td>?</td>
<td></td>
<td></td>
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<tr>
<td>Wade(^12)</td>
<td>1952</td>
<td>Fick</td>
<td>15</td>
<td>+20%</td>
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<td></td>
</tr>
<tr>
<td>Selzer(^13)</td>
<td>1960</td>
<td>Fick</td>
<td>15</td>
<td>—</td>
<td>+18%</td>
<td></td>
</tr>
</tbody>
</table>

\(^*\)Only rest studies; for exercise values, see reference.  
\(^\dagger\)CO = cardiac output, L./min.  
\(^\ddagger\)0.5 L./min. or greater increase in cardiac output.  
\(^\S\)Oral quinidine sulfate administered until reversion occurred.  
\(^\dagger\)Matched groups of patients with atrial fibrillation and sinus rhythm; see text.
atrial fibrillation and then in sinus rhythm were frequently separated by days or months, making a comparison of results difficult. Third, one of the well-recognized important influences on the level of cardiac output is the level of oxygen consumption. Some of the studies have failed to report the level of oxygen consumption at the time of cardiac output determinations and, therefore, comparisons do not seem valid.

Another approach taken by Wade et al. and Selzer has been to compare the cardiac outputs in groups of patients with atrial fibrillation and sinus rhythm. These patients were matched for the type of heart disease, the severity, age, duration, etc. It would seem to be more reliable to compare the same patient first in atrial fibrillation and then sinus rhythm than to introduce errors inherent in matching patients.

By the experimental design outlined above these four objections can be partially overcome. The only drug employed was a brief period of light anesthesia and thus the added effects of quinidine were avoided. The observations on cardiac output in atrial fibrillation and in sinus rhythm also were done in the same patient within a matter of 3 hours, by which time it was hoped that the effect of the anesthetic was dissipated. Finally, with continuous measurement of oxygen consumption it was assured that observations on cardiac output were recorded at similar levels of oxygen consumption.

In this or the other studies it was impossible to control the effect of differences in circulating levels of catecholamines, the mental attitude of the patient, etc. All efforts were made, however, to have the patient at ease and familiar with the experimental design.

A second objection to a comparison of cardiac output in atrial fibrillation and in sinus rhythm is that the number of effective ventricular contractions even at similar heart rates may, because of a pulse deficit, be less in atrial fibrillation. In this study the difference in heart rate was small, and the amount of change in cardiac output in any given patient was not proportional to the differences in heart rate.

The major objection would appear to be that the procedure of countershock itself induces a change in cardiac performance. We have no control observations on this phenomenon. No relationships in the individual patients were discernible between the number (one to six discharges) or energy of countershocks or the length of anesthesia and the change in cardiac output with the restoration of sinus rhythm.

In the 11 patients studied at rest, the most consistent change we noted was a decrease in arteriovenous oxygen difference (10 of 11 patients). Since arterial oxygen content remains constant in atrial fibrillation and in sinus rhythm, this was accomplished by an increase in oxygen content of mixed venous blood. Since oxygen consumption remained constant, this was reflected by an increase in cardiac output in nine of the 11 patients studied at rest. The technic of the Fick method of cardiac output has inherent errors and variations, and in this laboratory we consider the variability to be approximately ±0.5 L./min. With this limitation, seven of the 11 patients studied at rest showed a significant increase in cardiac output with conversion to sinus rhythm. This was an absolute increase of 0.6 to 3.2 L./min. In these seven patients the average increase was 1.5 L./min. or a rise of 34 per cent in cardiac output. In the five patients studied at exercise, all decreased their arteriovenous oxygen difference by over 1.0 volume per cent (average 2.04 volumes per cent) and all increased cardiac output from 1.1 to 1.8 L./min., average 1.4 L./min., or 17 per cent.

In the four patients in whom studies were performed, both at rest and during exercise there was a greater percentage increase in cardiac output at rest than during exercise in three. Unfortunately no observations were made during exercise on the four patients who did not increase cardiac output at rest to see if the stress of exercise would make this evident. Such an observation has been suggested by Hecht et al. following studies.
made with quinidine conversion. They noted
that certain patients failed to show an in-
crease in cardiac output at rest, but did with
exercise.

In this small series the change or lack of
change in cardiac output did not appear to
depend on the type of heart disease, the type
of valvular disease, the duration of atrial
fibrillation, or the functional classification.

The most obvious reason for an improve-
ment in cardiac output with restoration of
sinus rhythm would appear to be the addi-
tion of the “atrial pump mechanism” for in-
creasing the diastolic volume of the ventricle.
This mechanism has been well demonstrated
in dogs by Skinner et al.17 Further evidence
for the “inefficiency” of atrial fibrillation was
offered by Braunwald and Frahm.18 who
noted an increase in mean left atrial pressure
relative to left ventricular end-diastolic pres-
sure when this arrhythmia occurred during
cardiac catheterization. Graettingen et al.11
failed to note changes in cardiac output after
“cardioversion” but did offer data suggesting
improved ventricular function with restoration
of sinus rhythm. This failure may have been
because the patients were under anesthesia
during the study. The only other reported
study of cardiac output before and after “car-
dioversion” of atrial fibrillation is that of
Oran et al.10 These authors only briefly men-
tion their results, and their findings are sim-
ilar to those reported here. Thus, the addition
of the atrial pump mechanism and perhaps an
improvement in myocardial function may be
the explanation for the increased cardiac out-
put.

It is important to define what gains can
reasonably be considered with the re-es-
ablishment of sinus rhythm. Four such con-
siderations are (1) removal of the subjective
awareness of atrial fibrillation, (2) removal
of the inappropriate rate response to exer-
cise noted with atrial fibrillation,19 (3) re-
duction in systemic embolization, and (4)
improvement in cardiac function. This last
point is partially answered by this study. At
least under these acute experimental condi-
tions cardiac output can be reasonably antici-
ipated to increase. Seven of 11 patients in-
creased cardiac output 34 per cent at rest, and
all five patients studied at exercise increased
cardiac output 17 per cent.

Summary

Twelve patients were reverted from atrial
fibrillation to sinus rhythm by the technic of
“cardioversion.” Brief, light anesthesia was
the only drug employed. Under conditions of
comparable oxygen consumption cardiac out-
put was measured first in atrial fibrillation
and then in sinus rhythm. Eleven patients
were studied at rest and five during exercise.
Ten of the 11 patients studied at rest
showed a reduced arteriovenous oxygen dif-
fERENCE with sinus rhythm and seven of the
11 increased cardiac output 0.6 L./min. or
greater, an average increase of 34 per cent.
All five patients studied at exercise decreased
the arteriovenous oxygen difference with sinus
rhythm and cardiac output rose 1.1 L./min.
or greater in all, an average increase of 17
per cent.

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The Mother of Invention

Necessity is not the mother of invention; knowledge and experiment are its parents. This is clearly seen in the case of many industrial discoveries; high-speed cutting tools were not a necessity which preceded, but an application which followed, the discovery of the properties of tungsten chromium-iron alloys; so, too, the use of titanium in arc lamps and of vanadium in steel were sequels to the industrial preparation of these metals, and not discoveries made by sheer force of necessity.—W. R. Whitney.
The Changes in Cardiac Output with Reversion of Atrial Fibrillation to Sinus Rhythm

JAMES J. MORRIS, JR., MARK ENTMAN, WILLIAM C. NORTH, YIHONG KONG and HENRY MCINTOSH

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