Alterations in Cardiac Function Immediately Following Electrical Conversion of Atrial Fibrillation to Normal Sinus Rhythm

By William Shapiro, M.D., and Garner Klein, M.D.

SUMMARY
An assessment was made of cardiac function by means of right heart catheterization before and immediately after conversion of atrial fibrillation to sinus rhythm in 11 studies on 10 human subjects. Comparable data during exercise were available in five studies. Medical management of patients in this study was identical to that employed routinely for this procedure. After conversion to normal sinus rhythm cardiac output response was significantly increased only during exercise. The relationship of mean right atrial and mean pulmonary wedge pressures to right and left ventricular stroke work was uniformly improved both because of decreases in these pressures and increases in work. Indirect evidence of mitral valvular regurgitation was diminished to absent after conversion. "A" waves and the pulse pressure of atrial contraction were very small in the right atrial tracings after conversion and these contours were usually inapparent on pulmonary wedge tracings. It is concluded that over-all cardiac performance was improved with re-establishment of sinus rhythm and that diminutions in atrial-venous pressure behind each ventricle may be important in explaining symptomatic improvement following electroconversion. Measures of both pressure and flow were required for complete description of the alterations induced by this intervention.

Additional Indexing Words:
Cardiac output
Atrial function
Mitral stenosis
Intracardiac pressures
Right heart catheterization
Pressure-flow relationships
Ventricular function

Carefully controlled investigations have elucidated clearly the contribution made by a properly timed, effective atrial systole to cardiac performance and helped explain some of the deleterious effects of its absence in atrial fibrillation. Although consideration of the manifest hazards of the presence of atrial fibrillation in patients favors conversion to sinus rhythm, alterations in human circulation that accompany such therapy are still under investigation and to some extent in dispute. The availability of a safe, effective means of conversion, in the form of direct current countershock, has stimulated studies designed to resolve some aspects of these issues.

In general, investigations performed prior to the availability of electroshock therapy suffer from the fact that negative inotropic effects of quinidine were potentially present after, but not before, the administration of this drug. Recent studies, however, were performed without quinidine in order to

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define the pure effects of the change from atrial fibrillation to normal sinus rhythm. These studies used principally cardiac output determinations and showed that in most, but not all, patients a distinct increase in cardiac output occurred shortly after establishment of the sinus mechanism; additional evidence indicates that there is further progressive improvement in cardiac function with the passage of time. Since ordinary clinical use of direct current countershock often includes the administration of quinidine as well as barbiturate anesthesia, and because measurement of cardiac output alone does not precisely describe alterations in cardiac function, the present study was done in an attempt to assess the earliest effects of changing atrial fibrillation to normal sinus rhythm under the usual clinical conditions in which this procedure has been carried out. Furthermore, to evaluate the induced changes, right heart catheterizations were performed before and after these alterations in cardiac rhythm and, whenever possible, additional observations were made during leg exercise in the supine position.

Methods

Eleven studies were carried out in 10 patients. Seven studies were performed on the six patients in group A (table 1) whose mean age was 62 years (range 46 to 74). Patients representing group A studies 4, 5, and 6 had ischemic heart disease by virtue of previous myocardial infarction. The etiology of congestive failure in group A studies 1 and 2 was considered most likely to be due to ischemic heart disease. Studies 3 and 7 of group A were performed 9 months apart in a 46-year-old man who had diffuse myocardial disease of unknown etiology. The duration of the arrhythmia in this group of patients ranged from 3 weeks to 5 years. All had had cardiomegaly and congestive heart failure on admission to hospital. Group B consisted of four patients who had undergone mitral comissurotomy 2 to 7 months prior to study. Their mean age was 42 years (range 32 to 51). Each had had classical clinical, hemodynamic, and operative findings of significant mitral stenosis; patients 1, 2, and 3 had improvement demonstrated by postoperative reduction in pulmonary arterial and wedge pressures. Patient 4 of this group, 3 months postoperative, had no improvement in symptoms or hemodynamic findings. Patient 1 had complicating angina pectoris and intermittent claudication. All patients in group B had been known to have had atrial fibrillation continuously for at least 2 to 7 months.

Prior to study each patient had been treated optimally with digitalis and diuretics when indicated. In the 24 to 48 hours prior to study, digitalis had been discontinued but quinidine sulfate was given orally in doses of 0.2 g every 6 hours. During the period of this study four additional prospective study patients were converted on this regimen but were lost to follow-up. Medical management in this study was identical to that utilized clinically in our hospital in patients being prepared for elective conversion by external countershock.

Right heart catheterizations were carried out in the conventional manner during the postabsorptive state, but premedication with sedative or analgesic drugs was not given. Cardiac output was determined by the Fick principle. Following resting determinations, leg exercise in the supine position was carried out for 4 to 6 minutes on a bicycle-type ergometer. Control pulmonary wedge pressures at rest were measured with the legs elevated in the exercising position; continuous recordings were taken during exercise. At the end of the second minute of exercise, the catheter was withdrawn into the pulmonary artery and mean pulmonary artery pressure tracings were recorded. During the third to fifth minute, cardiac output was measured. Following completion of the measurements obtained during atrial fibrillation, the patient was anesthetized with an ultra short-acting barbiturate (sodium pentothal or sodium methohexitol) to a light plane of anesthesia. Synchronized electrical DC countershock was applied as described by Lown and associates. Shocks of 150 to 300 watt seconds were required for conversion. Following establishment of normal sinus rhythm, the patients became responsive within 20 to 30 minutes. At this point the determinations were repeated.

Analysis of data was carried out on at least two separate occasions with care taken to average at least 10 cardiac cycles to obtain heart rate and pressure levels, both during atrial fibrillation and sinus rhythm. Vascular resistance was expressed in units. Stroke work was calculated according to the formula of Mitchell and co-workers. Calculations substituting pulmonary wedge mean pressure for left ventricular end-diastolic pressure may have underestimated actual left ventricular stroke work in those patients in group B who had possible residual left atrial-left ventricular end-diastolic pressure gradients (most likely for group B, patient 4). Statistical analyses
Comparison of Metabolic and Circulatory Data before and after Electrical Conversion of Atrial Fibrillation to Normal Sinus Rhythm

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Results

Data at Rest

The individual and mean data as well as the results of paired t-test analyses are presented in tables 1 to 3, which indicate that the metabolic status of the entire series was almost identical during both periods of study. There were no material differences in mean basal metabolic rate, oxygen consumption, heart rate, systemic vascular resistance, and pulmonary vascular resistance. In addition, resting cardiac output, stroke volume, arteriovenous oxygen difference, and right ventricular stroke work were unchanged shortly after establishment of normal sinus rhythm. Conversely, the mean pulmonary wedge pressure for the entire series (group A+B, table 1) declined from 17.7 to 13.1 mm Hg (P < 0.01), indicating paired t-tests were carried out by standard methods14 with the aid of a digital computer.

BMR = basal metabolic rate; A-V O2 = arteriovenous O2 difference; R = rest; E = exercise.

Table 1

![Figure 1](shapiro.png)

Comparison of mean pressures at rest before and after establishment of sinus rhythm for entire series. P values are below each pair of bars. PWm = pulmonary wedge mean; PAm = pulmonary artery mean; RVEDP = right ventricular end-diastolic pressure; RAm = right atrial mean; MABP = mean arterial blood pressure; NS = not significant.

Circulation, Volume XXXVIII, December 1968
ALTERATIONS IN CARDIAC FUNCTION

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|--------------------|------------------------|                         |             |             |             |             |             |             |
| 54                  | 130                    | 75, 98                  | 97           | —           | 95          | 103         | 5.4         | 4.2         | 10.9        |
| 110                 | 129                    | 84, 102                 | 60           | 72          | 76          | 108         | 5.0         | 8.6         | 4.9         | 8.2         |
| 65                  | 96                     | 63, 90                  | 72           | 75          | 71          | 82          | 5.0         | 9.0         | 5.5         | 8.8         |
| 80                  | 99                     | 70, 84                  | 74           | 97          | 96          | 125         | 5.1         | 8.9         | 5.0         | 8.1         |
| 77.3                | 113.5                  | 73, 93.5                | 75.8         | 81.3        | 84.5        | 105         | 5.1         | 8.8         | 4.9         | 8.3         |
| 24.3                | 18.5                   | 8.8, 8.1                | 15.5         | 13.7        | 12.9        | 21.7        | 0.2         | 0.2         | 0.5         | 0.4         |

|                    | Before                  | After                   | Before       | After       | Before      | After       | Before      | After       |
|--------------------|------------------------|                         |             |             |             |             |             |             |
| 76.2                | 103                    | 77.7, 90.8              | 69.0         | 80.2        | 74.4        | 103.2       | 5.61        | 10.2        | 5.55        | 9.8         |
| 17.5                | 21.7                   | 12.2, 12.8              | 16.9         | 11.5        | 22.7        | 31.3        | 0.84        | 2.4         | 1.33        | 2.6         |

|                    |                        |                         |             |             |             |             |             |             |
| 54                  |                        |                         |              |             |              |             |             |             |

| Right ventricular end-diastolic pressure decreased from 7.2 to 6.5 mm Hg (P < 0.02), right atrial mean pressure decreased from 6.5 to 3.3 mm Hg (P < 0.01), whereas the average pulmonary artery mean and radial arterial mean pressures did not change significantly (fig. 1).

Mean data from group A alone revealed significant decreases in right ventricular end-diastolic pressure from 8.8 to 7.0 mm Hg (P < 0.05), and right atrial mean pressure from 8.1 to 6.0 mm Hg (P < 0.05). Mean pulmonary wedge pressure declined 3 mm Hg, and cardiac output increased 0.6 L/min, but these changes were not of statistical significance.

Significant mean changes in group B included an increase in left ventricular stroke work from 62.2 to 76.7 g-m (P < 0.01), a decrease in mean pulmonary wedge pressure from 14.8 to 7.8 mm Hg (P < 0.01), and a decline in right atrial mean pressure from 3.5 to −1.5 mm Hg (P < 0.02). Mean cardiac output also increased 0.6 L/min in group B from 5.6 to 6.2 L/min and stroke volume increased from 76 to 85 ml/beat, but these changes did not achieve significance upon t-test analysis.

**Figure 2**

Mean pulmonary wedge pressure at rest and after 1 and 2 minutes of exercise during atrial fibrillation (AF, solid line) and normal sinus rhythm (NSR, broken line) in a group B patient.
Table 2
Comparison of Systemic and Pulmonary Pressures and Resistances before and after Electrical Conversion of Atrial Fibrillation to Normal Sinus Rhythm

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<th>Mean PA pressure (mm Hg)</th>
<th>Pulmonary vascular resistance (units)</th>
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R = rest; E = exercise.

Data during Exercise

Five complete studies during exercise were obtained (group A 5, 7; group B 2, 3, 4). Of importance, the level of exercise was at the same level before and after conversion as judged from the level of oxygen consumption. During exercise mean cardiac output was 1.16 L/min higher ($P < 0.05$) after conversion; A-V $O_2$ difference was 0.4 vol % less ($P < 0.05$), and pulmonary wedge mean was 2.6 mm Hg lower ($P < 0.05$) in these five patients. Compared with their respective resting values before and after conversion, the mean postconversion wedge pressure rose 2 mm Hg more during exercise (figure 2 presents an example) while the mean pulmonary artery pressure rose 4 mm Hg less than during atrial fibrillation. Left ventricular stroke work was greater, rising from 66.8 to 89.4 g-m. Although heart rate in the five studies was slower during exercise after conversion, 90.8 versus 103.0/min, this change was not statistically significant. Analysis limited to the three group B patients did reveal the preconversion and postconversion heart rates (113.5 versus 93.5) to be significantly less ($P < 0.05$).

**Figure 3**
Right atrial pressure contour in a group B patient at comparable heart rates during atrial fibrillation (AF, left panel) and after successful countershock therapy (NSR, right panel).
Table 3
Comparison of Pressures and Stroke Work before and after Electrical Conversion of Atrial Fibrillation to Normal Sinus Rhythm

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<td>&lt;0.02</td>
<td>&lt;0.01</td>
<td>NS</td>
<td>&lt;0.01</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

RVEDP = right ventricular end-diastolic pressure; RA mean = right atrial mean pressure; RVS = right ventricular stroke work; LVSW = left ventricular stroke work; R = rest; E = exercise.

Atrial Pulse Contour Analysis

The height of the right atrial “a” wave during the period immediately after conversion was small, averaging 3.2 ± 3.8 mm Hg for the entire group (table 4). The mean for group A was 3.9 ± 3.8, and the mean for group B was 1.8 ± 3.9. The pulse pressure of the “a” wave was small, the average for the group as a whole being 1.8 ± 1.5, for group A 1.4 ± 0.8, and for group B 2.7 ± 2.3.

Figure 3 presents a right atrial pressure tracing from a patient in group B and shows the appearance of recognizable atrial contours and a diminutive “a” wave pulse pressure following establishment of sinus rhythm.

In more than half of the patients consistent late systolic waves identifiable as C-V waves were noted in the preconversion resting pulmonary wedge contour tracings. An example is seen in figure 4, which also illustrates the marked attenuation of these waves following establishment of sinus rhythm.

Analyses of Cardiac Function

Figure 5 illustrates the relationship between right ventricular end-diastolic pressure and right ventricular stroke work measured during the resting state before and after cardioversion. The group as a whole, as well as each of the two subgroups, showed shifts in this relationship following conversion resulting from both apparent increases in right ventricular stroke work and decreases in right ventricular end-diastolic pressure. Figure 6 relates the right atrial mean pressure and the right ventricular stroke work, and it may be seen that the mean right atrial pressures at the observed levels of right ventricular work were reduced after restoration of sinus rhythm.
Table 4
Analysis of Right Atrial A Waves Immediately after Electrical Conversion of Atrial Fibrillation to Normal Sinus Rhythm

<table>
<thead>
<tr>
<th>Group and study</th>
<th>RA “A” pulse (mm Hg)</th>
<th>RA “A” height (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group A</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>3.3</td>
</tr>
<tr>
<td>3</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>12</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>7</td>
<td>1.5</td>
<td>11.0</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td>1.4</td>
<td>3.9</td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td>0.8</td>
<td>3.8</td>
</tr>
<tr>
<td><strong>Group B</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>1.6</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>-1.4</td>
<td>-1.4</td>
</tr>
<tr>
<td>3</td>
<td>1.6</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>7.5</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td>2.7</td>
<td>1.8</td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td>2.3</td>
<td>3.9</td>
</tr>
<tr>
<td><strong>Group A+B</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td>1.8</td>
<td>3.2</td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td>1.5</td>
<td>3.8</td>
</tr>
</tbody>
</table>

The availability of mean pulmonary wedge pressures, which are assumed to approximate mean left atrial pressure, allows an analysis of over-all left ventricular function. Figure 7 illustrates the relationship between pulmonary wedge pressure and cardiac output, both at rest and during exercise, with normal sinus rhythm and atrial fibrillation in one of the group B patients. In figure 8 the same relationships are presented for one of the patients of group A. The reduction of mean right atrial pressure together with slight rises in right ventricular stroke work is seen for the entire series (n = 11) as well as the subgroups after rhythm conversion.

**Figure 4**
Pulmonary wedge pressure contour before and after conversion. The prominent waves during atrial fibrillation (AF, left panel) were diminished after conversion (NSR, right panel).

**Figure 5**
Relationship between mean right ventricular end-diastolic pressure and mean right ventricular stroke work at rest during atrial fibrillation (circles) and normal sinus rhythm (triangles). Group A is n = 7; group B is n = 4; entire series is n = 11.
ALTERATIONS IN CARDIAC FUNCTION

A given level of cardiac output was observed at lower mean pulmonary wedge pressures after establishment of sinus rhythm in this group B patient.

The three complete rest and exercise studies in group B revealed that after restoration of normal sinus rhythm more left ventricular stroke work was performed at lower pulmonary wedge pressures both at rest and during exercise. The data at rest from the entire group and for group A also show increased work with lower mean wedge pressures.

Linear regression analyses were carried out to determine whether these data could be summarized in terms of statistical significance. Significant differences were not noted in...
either the before and after slopes or y intercepts of the raw data. Since each pair of data represents a distinctly different subject, construction of before and after slopes shows great overlap, precluding the success of this type of analysis in showing significant differences. Table 3 shows that while right and left ventricular stroke work only tended to increase with sinus rhythm, the right ventricular end-diastolic, mean right atrial, and mean pulmonary wedge pressures all declined significantly when subjected to paired t-test analyses.

Discussion

It is clear that complete evaluation of circulatory alterations after interventions such as those here requires analysis of pressure-flow relationships, since gross alterations in cardiac output may not always be observed following cardioversion,6,7 nor may changes in cardiac output necessarily completely describe the nature of what has occurred. The limited number of studies describing pressure-flow relationships immediately before and after electrical conversion of atrial fibrillation to normal sinus rhythm have yielded somewhat conflicting results with regard to the changes in cardiac output, left atrial pressure, and the importance of a decrease in heart rate.7,15 Predictions of results based on atrial pressure contours in the absence of simultaneous flows have also been made.16

In the present study mean cardiac output did increase in each group at rest and during exercise, but these alterations were significant only for the five exercise studies (P < 0.05).

If one considers the mean right or left atrial pressure associated with a given level of ventricular work (that is, the “hemodynamic price” of ventricular work), it is clear that these pressures were diminished in the patients in the present study following establishment of the sinus mechanism (figs. 6 to 9). The pressures behind each ventricle at any given level of work appear to be important to an individual patient’s sense of well being. The role of the rises in left atrial-pulmonary venous pressure in the production of dyspnea in patients with disease distal to the pulmonary capillary bed seems clear17 and elevations of right atrial pressure lead to a variety of debilitating phenomena. Reductions in pulmonary wedge pressure associated with re-establishment of sinus rhythm, especially during exercise, may aid in prevention of pulmonary edema and dyspnea.17 Also, reductions in intracardiac pressures have reportedly been associated with more favorable prognoses in patients with heart failure when contrasted with those patients who maintain high pulmonary vascular pressures in the presence of similar levels of cardiac output.18

The hemodynamic “stimulus” to ventricular work, that is, ventricular end-diastolic pressure itself may be distinguished from the “price” of that work.19 The data in figure 5 show reductions in right ventricular end-diastolic pressure and increases in right ventricular stroke work after conversion. These alterations may be interpreted as being due to positive inotropic effects associated with the establishment of the sinus mechanism. It is unclear from detailed analyses of factors regulating ventricular function19 why such left shifts should have occurred. It would rather have been expected that ventricular function should move upward on the same curve with increases in stroke work. A possible alternate explanation of these apparent inotropic effects may be inferred from the pressure tracing in figure 4. If it be accepted that atrioventricular valvular regurgitation occurred during atrial fibrillation, as has been shown by Skinner and colleagues3 (and which may be reflected in figure 4 and the many similar tracings in other patients in this series), then the analysis of present data would have underestimated stroke work during the arrhythmia, and the shift to a different function curve represents an artifact. The type of data obtained precludes quantitative expressions of the amount of such regurgitation. Other explanations of an actual or apparent shift to a new curve (improved ventricular function) include (1) increases

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in catecholamine levels; (2) change in pressure-volume relationships of the right ventricle; and (3) overestimation of right ventricular end-diastolic pressure during fibrillation because of the varying nature of the pressures under these conditions.

The failure to observe clear "a" waves in pulmonary wedge pressure tracings following conversion was not unexpected since at best, pulmonary wedge contours represent damped, unpredictable, distorted images of left atrial pressure contours despite the almost uniform similarity of wedge and left atrial mean pressures. Analysis of right atrial "a" waves, however, revealed them to be small and to have small pulse pressures. Follow-up studies would have been of interest to observe any associations between increases in these atrial pressure contours and over-all cardiac performance, but such studies could not be carried out in the patients under discussion. Despite the small "a" waves, declines in mean atrial and right ventricular end-diastolic pressures occurred and were associated with what are interpreted as improvements in over-all cardiac function. It would appear, therefore, that assessments based on the small amplitude or unimpressive form of this type of atrial contour alone may be misleading in this situation.

These observations reveal that, especially under adverse circumstances, normal sinus rhythm is advantageous for cardiac performance. These data provide examples of failure to find improvement in cardiac function if evaluation had been made only on the basis of increases in cardiac output. They do not agree with the explanation that diminution in heart rate is the major factor leading to improved ventricular function in patients such as those in this series. With the likely exceptions of patients with advanced mitral stenosis and patients without organic heart disease, whose autonomic system can provide adequate compensation (though perhaps not under maximal exercise stresses), it may be concluded that normal sinus rhythm permits a lowered hemodynamic "price" to be paid for any given level of ventricular work when contrasted with atrial fibrillation.

Acknowledgment

The authors are grateful to Dr. N. S. Skinner for his critical review of this work.

References


Early Transplantation Experiments (circa 1750)

The success of this operation [transplanting teeth] is founded on a disposition in all living substances, to unite when brought into contact with one another; although they are of a different structure; and even although the circulation is only carried on in one of them.

This disposition is not so considerable in the more perfect or complex animals, such as quadrupeds, as it is in the more simple or imperfect; nor in old animals, as in young; for the living principle in young animals, and those of simple construction, is not so much confined to, or derived from one part of the body; so that it continues longer in a part separated from their bodies, and even would appear to be generated in it for some time; while a part, separated from an older, or more perfect animal, dies sooner, and would appear to have its life entirely dependent on the body from which it was taken.

Taking off the young spur of a cock, and fixing it to his comb, is an old and well known experiment.

I have also frequently taken out the Testis of a cock and replaced it in his belly, where it has adhered, and has been nourished; nay, I have put the Testis of a cock into the belly of a hen with the same effect.—John Hunter: The Natural History of the Human Teeth. London, Robert Hardwicke, 1865, p. 156.
Alterations in Cardiac Function Immediately Following Electrical Conversion of Atrial Fibrillation to Normal Sinus Rhythm
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