Acute Effects of Countershock Conversion of Atrial Fibrillation upon Right and Left Heart Hemodynamics

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RENEWED INTEREST in the therapy of atrial fibrillation has emerged from the successful clinical application of external countershock. This method is being used with increasing frequency, and an evaluation of its effectiveness should take into consideration whether significant hemodynamic improvement is induced by restoration of sinus rhythm.

Several studies have been reported comparing the circulatory behavior in atrial fibrillation and in sinus rhythm. Investigations were usually performed either on parallel series of arrhythmic and rhythmic patients or in the same subjects following quinidine conversion to normal rhythm. Both methods introduce extraneous factors due to the unavoidable pathophysiologic diversities between different patients or to the changes that may occur during the time interval between separate studies on the same individuals, independent of the rhythm.

External countershock defibrillation provides a simple means of restoring sinus rhythm within seconds, without the adjunction of pharmacologic interference, with each patient serving as his own control under closely comparable conditions in a single procedure. It seemed therefore of interest to apply the method to the hemodynamic study of the fibrillating and rhythmic heart.

Material and Methods

The present report concerns 12 patients, seven female and five male, aged 24 to 50 years. All had isolated mitral valvular disease. Mitral commissurotomy had been performed on seven patients, 20 days to 8 years prior to this study. In this group varying degrees of residual valvular dysfunction remained, and in two patients significant mitral regurgitation had been created at surgery. The remaining cases were studied before operation; predominant mitral stenosis was present in two and major mitral insufficiency in three. There was no clinical or hemodynamic evidence of aortic valve involvement in any instance.

Under light plane anesthesia, which allowed for spontaneous respiration of room air, catheters were advanced to the main pulmonary artery via the right antecubital vein, to the left atrium by the transseptal approach, and to the left ventricle by the percutaneous retrograde arterial technic. The right brachial arterial pressure was obtained through an indwelling Cournand needle. Left atrial, left ventricular, systemic, and pulmonary arterial pressures were recorded simultaneously or in rapid succession while the patient was breathing into an oxygen consumption circuit. Mixed venous and arterial blood samples were then withdrawn for Fick determination of cardiac output, and immediately thereafter right ventricular and right atrial pressures were recorded. The entire procedure was repeated in an exactly similar fashion approximately 30 minutes after sinus rhythm had been restored by synchronized direct-current countershock. Mean pressures were obtained by electrical integration. All measurements were made on a minimum of 10 beats and averaged. Ventricular stroke work was calculated according to the formula:

\[
\text{Stroke Index} \times 1.055 \times 13.6 \times (\text{mean pulmonary or mean brachial arterial pressure} - \text{right or left ventricular end-diastolic pressure})
\]

Oxygen content of blood samples was determined by the method of Van Slyke and Neill.

Results

Heart rate during sinus rhythm was within

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*C23G Statham strain gages; DR8 recorder, Electronics for Medicine.
†Corbin-Farnsworth DC defibrillator.

Circulation, Volume XXXII, August 1965

214
ACUTE HEMODYNAMIC EFFECTS OF COUNTERSHOCK

A maximum of 10 beats per minute faster or slower than the rate during fibrillation, being increased in four cases, decreased in three, and unchanged in five after conversion.

The systolic pressure in the right ventricle and pulmonary artery decreased in seven patients, increased in four, and was unchanged in one case (variations ranged from −23 to +24 per cent, group average −6 per cent). The pulmonary arterial mean pressure was decreased in six, increased in three, and unchanged in three patients (from −33 to +28 per cent, average −6 per cent). The variations observed were often of minimal degree. The systolic pressure in the left ventricle and brachial artery increased in seven patients, decreased in four, and was unchanged in one, the increments being more significant than the decrements (from −7 to +20 per cent, average +6 per cent). The beat-to-beat variations of peak systemic pressure secondary to the changing duration of diastole during fibrillation disappeared when sinus rhythm was restored. The brachial arterial mean pressure increased in seven and decreased in the remaining cases (from −14 to +28 per cent, average +1 per cent). Thus there appeared to be a tendency for the ejection pressures to decrease in the right heart and increase in the left heart (fig. 1).

The end-diastolic pressure in the right ventricle (RVEDP) decreased in eight patients.

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**Figure 1**

Graphic representation of changes in pulmonary arterial systolic (PA syst) and mean (PAM) pressures and in brachial arterial systolic (BA syst) and mean (BAM) pressures. In this figure and in figures 2, 3, and 4, for each measurement the values obtained during atrial fibrillation are plotted on the left-hand side, and those obtained during sinus rhythm on the right-hand side. Each solid line represents one patient. Broken lines represent group averages expressed in per cent variation.

*Circulation, Volume XXXII, August 1965*
Right ventricular end-diastolic (RVEDP), mean right atrial (MRAP), left ventricular end-diastolic (LVEDP), and mean left atrial (MLAP) pressures in atrial fibrillation and sinus rhythm.

The mean right atrial pressure (MRAP) was likewise usually lower during sinus rhythm (from −60 to +33 per cent, average −12 per cent). The mean left atrial pressure (MLAP) decreased in four cases, increased in six, and was unchanged in two (from −29 to +22 per cent, average 0 per cent) (fig. 2). In the six patients in whom MLAP was augmented, it appeared to be related to a prominent re-appearing “a” wave when some degree of mitral valvular obstruction was present, or to a significant rise of the “v” wave in two cases with severe mitral regurgitation. On the other hand, significant decreases of MLAP occurred only in the patients with the lower transmitial pressure gradients in the absence of severe regurgitation. The mean linear diastolic pressure gradient between the left atrium and the left ventricle remained practically unchanged in all cases after conversion to sinus rhythm.

Oxygen saturation of mixed venous blood was augmented in six cases, practically unchanged in five cases and decreased in one (from −9 to +20 per cent, average +5 per cent), whereas significant changes in arterial blood saturation (4 per cent or more) occurred in five cases, each of which showed
increased values after countershock (from −3 to +13 per cent, average +3 per cent). The resulting effect was a decrease in the arteriovenous oxygen difference in half the patients (from −26 to +24 per cent, average −2 per cent).

The oxygen consumption variations ranged from −10 to +54 per cent (average +7 per cent), with a tendency to higher values during sinus rhythm. Consequently, there was an increase in cardiac output and index in eight cases, and in stroke volume and index in nine. Cardiac output changes ranged from −20 to +41 per cent, average +7 per cent; stroke volume changes ranged from −16 to +67 per cent, average +7 per cent; and stroke index changes ranged from −16 to +76 per cent, average +15 per cent (fig. 3).

The interplay of output and pressures was such that a decrease in pulmonary vascular resistance (from −79 to +59 per cent, average −33 per cent) and systemic resistance (from −34 to +58 per cent, average −10 per cent) resulted in most cases (fig. 3).

The stroke work of the right ventricle was augmented in eight patients, variations ranging from −25 to +80 per cent, average +30 per cent, and that of the left ventricle was increased in nine cases (from −16 to +90 per cent, average +22 per cent) after conversion (fig. 4).

**Discussion**

In the present study, each patient being his own control, the possible interference of extraneous factors tending to invalidate the comparison of hemodynamic data before and after restoration of sinus rhythm was reduced. In contrast to investigations performed on separate series of patients, the anatomic lesions and the degree of compensation at the time of examination were exactly comparable in each set of determinations. No drugs were administered between studies which may have an effect of their own upon the circulation, such as quinidine. Direct-current countershock in itself does not seem to affect the myocardium and its functions, although rare

![Figure 3](http://circ.ahajournals.org/)

**Figure 3**

Cardiac output (C.O.), stroke volume (S.V.), pulmonary vascular resistance (P.V.R.), and systemic resistance (S.R.) in atrial fibrillation and sinus rhythm.
instances of electrocardiographic signs of the lesion have been reported. None of the patients in our series exhibited such changes. The use of general anesthesia throughout the procedure is open to criticism, since it will tend to depress respiration. However, this practice was adopted after the first four patients studied (not included in the present series), in whom general anesthesia was given only at the time of countershock, had shown consistently a marked delay in resuming basal conditions upon awakening. It was deemed that a fundamental requisite was to maintain all factors besides the rhythm as stable as possible throughout the procedure and the technic selected seemed best to meet these requirements. Thus, the results obtained should represent the direct effects of conversion to sinus rhythm. The increase or decrease in heart rate was in no instance sufficient to be held responsible for the hemodynamic changes observed. In the only patient in whom heart rate during atrial fibrillation was above 100 beats per minute it remained elevated during sinus rhythm.

Most previous investigations have emphasized the increase in cardiac output brought about by restoration of sinus rhythm. Recently, Burchell, referring to unpublished data obtained at the Mayo Clinic and to the observations of Graettinger et al., who found no significant increase in cardiac output after countershock conversion, voiced the opinion that such an alleged increase should not be considered as the main reason for attempting to restore sinus rhythm. Our data do not seem entirely to support this view: six patients of 12 showed an increase in cardiac output of over 10 per cent above the fibrillation values, as opposed to a significant decrease in two patients. The augmented flow resulted in most cases from the combination of increased oxygen consumption and decreased arteriovenous difference. The latter finding was emphasized by Storstein and Tveten, who attributed it to a peripheral vasodilating effect of quinidine, and by Broch and Müller, who regarded the lower arteriovenous difference as a sign of improved conditions of oxygen supply and tension in the tissues during sinus rhythm. There is no reason to believe that dye-curve measurements of cardiac output would have yielded different results than Fick determination in our cases, in terms of relative changes in the single patients from atrial fibrillation to sinus rhythm. Moreover, our patients were studied only at rest, and it is generally agreed that the response of cardiac output to exercise is definitely more adequate during sinus rhythm than during atrial fibrillation. It is true that there are other considerations than those of flow in evaluating the usefulness of restoring sinus rhythm.

In agreement with most investigators, systolic and mean pressures in the ventricles and in the pulmonary artery and brachial artery were not systematically influenced by cardioversion. However, there appeared to be a trend for the systolic pressures to decrease in the lesser circulation and increase in the systemic circulation. As would be expected, the fixed duration of diastole in sinus rhythm conferred a greater stability to the systemic peak arterial pressure from beat to beat.

In the right heart, where obstruction to flow from atrium to ventricle was not a com-

Figure 4

Left ventricular (LVSW) and right ventricular (RVSW) stroke work in atrial fibrillation and sinus rhythm.
plicating factor, the observed decrease in mean right atrial pressure accompanied a drop in RVEDP in most cases. The lower MRAP could also have been due partly to the suppression of that degree of functional tricuspid regurgitation which, according to Ferrer and Harvey, is almost invariably present during atrial fibrillation in spite of a normal valve.

The behavior of MLAP is less readily explained. An increased MLAP, as occurred in half of our cases, could be expected in patients with some degree of mitral valvular obstruction: an augmented transvalvular flow would in effect raise the diastolic pressure gradient across a fixed valve area. However, this did not occur in our patients in whom the mean linear gradient remained practically unchanged. Nor would the planimetric gradient be expected to behave differently in the absence of significant changes in the average duration of the ventricular filling period. This implies that the transvalvular flow was not increased sufficiently to raise the pressure gradient or that the element of stenosis was unimportant.

In the patients with mitral obstruction, the increase in MLAP correlated with the height of the reappearing “a” wave. On the other hand, in those patients in whom MLAP decreased with sinus rhythm, the transvalvular gradients were the lowest of the series, indicating the absence of significant obstruction. In these same patients there was a parallel decrease of MLAP and LVEDP similar to the changes described on the venous side. It appears therefore that the augmented MLAP could be explained as a consequence of the more forceful contraction of the left atrium against an obstructed mitral valve. In the presence of mitral insufficiency, the enhanced left ventricular stroke volume will manifest itself as an increase in forward as well as backward flow, thus altering the pressure-volume relationship of the left atrium during ventricular systole and raising the “v” wave and the MLAP. It is important to note that a higher MLAP was in no instance accompanied by a significant increase in pulmonary arterial pressure.

The most significant pressure changes occurred at the time of ventricular filling. It must be noted here that a particular feature of this group of patients was the high incidence of abnormally elevated end-diastolic pressure in both right and left ventricles: RVEDP was above 5 mm. Hg and LVEDP was above 10 mm. Hg in 11 of the 12 patients. The higher RVEDP values were observed in the patients with mitral insufficiency and in those with mitral stenosis complicated by marked pulmonary hypertension. The initial elevation of LVEDP above 10 mm. Hg could be explained in the five cases with mitral regurgitation and in four patients studied less than 30 days after mitral commissurotomy. In the latter situation, the left ventricle may still be in the phase of its adjustment to the suddenly increased load caused by the augmented diastolic filling secondary to the removal of the valvular obstruction. The remaining two patients showing a higher than 10 mm. Hg LVEDP while in atrial fibrillation had pure mitral stenosis and were studied before operation. There was no apparent clinical or hemodynamic explanation for this abnormal finding other than a hypothetical left myocardial disease. Whatever the reason for the initial elevation of ventricular end-diastolic pressure, restoration of sinus rhythm was accompanied by a reduction of RVEDP in eight and of LVEDP in nine of 12 patients.

On the venous side of the heart, in the face of unchanged pulmonary arterial pressure and of decreased pulmonary vascular resistance, the hemodynamic changes observed after return to sinus rhythm were indicative of an improved function obtained at a lower level of both MRAP and RVEDP.

An analysis of the behavior of left ventricular end-diastolic pressure in relation to systemic pressure, cardiac output, and work appears pertinent to the evaluation of the effect of restoring sinus rhythm. The work of Sarnoff et al. and of Braunwald et al. has emphasized the importance of these measures of

Circulation, Volume XXXII, August 1963
cardiac performance, and in particular of the transport function of the atrium and of the relation between mean left atrial and left ventricular end-diastolic pressures. In the words of Braunwald and Frahm, the level of LVEDP represents the stimulus that determines the force of ventricular contraction and the MLAP the price that the organism must pay to provide this stimulus. It has been shown that when LVEDP is elevated, the MLAP is maintained at a lower level by the contribution of left atrial systole. In this respect, normal sinus rhythm will have a beneficial effect in those patients without significant mitral valvular obstruction. Although, as stressed by Braunwald and Ross, an elevated LVEDP should not be considered in itself indicative of ventricular failure, it is apparent that the markedly increased LVEDP observed in most of our patients while in atrial fibrillation must represent failure. In the majority of these patients, in fact, the significant decrements of LVEDP occurring after restoration of sinus rhythm were accompanied by parallel increases rather than decreases of systemic systolic pressure and of stroke index (figs. 5 and 6). However, according to Sarnoff, whereas a constant relationship exists between atrial pressure (in the absence of valvular obstruction) and the stroke work of the ventricle, such a relationship is not consistently found between atrial pressure and stroke volume and cardiac output. Therefore, stroke work is the value to consider in the analysis of ventricular performance. Figure 7 shows the behavior of left ventricular stroke work in respect to LVEDP in our cases. Of nine patients who showed a reduced LVEDP with sinus rhythm, left ventricular stroke work was augmented in eight and slightly diminished in one. Conversely, stroke work decreased in the two patients in whom res-

![Figure 5](http://circ.ahajournals.org/)

**Figure 5**

Correlation of left ventricular end-diastolic pressure (LVEDP) with the brachial arterial systolic pressure (BA syst). In this figure and in figures 6 and 7 each line represents one patient, the closed circles marking the values obtained during atrial fibrillation and the open circles the values recorded during sinus rhythm.
Correlation of left ventricular end-diastolic pressure (LVEDP) with the stroke index.

Restoration of sinus rhythm was accompanied by an increased LVEDP. Thus, it appears from these observations that the change from atrial fibrillation to sinus rhythm resulted in a shift of the ventricular function curve to an area of improved function in the majority of patients. If the higher levels of LVEDP during atrial fibrillation had not represented some degree of impaired performance, decrements of this value would have been accompanied by decreases in ventricular stroke work, as a consequence of a shift to the left along the same ventricular function curve.21, 27

In conclusion, the data obtained indicate that the over-all performance of the heart can be definitely enhanced by the return to sinus rhythm. It is realized that in some of our patients who appeared to be incompletely compensated at the time of investigation, hemodynamic improvement might have been obtained by further therapeutic regimen alone without conversion to sinus rhythm. However, suppression of atrial fibrillation was indeed capable of achieving immediate beneficial effects. Furthermore, it should be noted that the present study was concerned with acute changes. It is not unlikely that further improvement of cardiac performance will occur after a more prolonged stabilization of the new hemodynamic situation is achieved.

**Figure 6**

**Correlation of left ventricular end-diastolic pressure (LVEDP) with the stroke index.**

**Summary**

Right and left heart hemodynamics were studied in 12 patients with mitral valvular disease and atrial fibrillation before and immediately after external direct-current countershock resulting in restoration of normal sinus rhythm. The change of rhythm was accompanied by the following hemodynamic variations in the majority of cases: Systolic and mean pressures in the right and left ventricles and in the pulmonary and brachial arteries were not systematically altered, with a tendency, however, to a decrease in ejection pressures in the right heart and increase in the left heart. Mean right atrial and right ventricular end-diastolic pressures as well as left ventricular end-diastolic pressure decreased significantly. The behavior of the mean left atrial pressure appeared to be influenced by the degree of mitral valvular disease. Cardiac output and index and stroke volume and index increased; pulmonary vascular and systemic resistances decreased.

The stroke work of the ventricles also increased in the majority of patients.

An analysis of the changes in left ventricular end-diastolic pressure in relation to the left ventricular stroke work indicated that sinus rhythm resulted usually in a shift of the ven-

**Figure 7**

**Correlation of left ventricular end-diastolic pressure (LVEDP) with the stroke work of the left ventricle (LVSW).**

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tricular function curve to an area of improved performance.

References
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