Echographic Assessment of Atrial Transport, Mitral Movement, and Ventricular Performance Following Electroversion of Supraventricular Arrhythmias

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SUMMARY

Controversy attends the extent and temporal sequence of improvements in hemodynamic function resulting from the return of atrial contraction following cardioversion of supraventricular arrhythmias. Thus, mitral, left atrial (LA) and left ventricular (LV) echograms were obtained before and one hour after conversion of supraventricular arrhythmias to normal sinus rhythm by direct current countershock in patients with chronic coronary disease or cardiomyopathies without valvular dysfunction. The duration of the rhythm disturbance varied from one day to five years in 22 patients and was indeterminate in 13. Atrial systole immediately produced prominent mitral “A” waves with anterior valve excursion of 7.5 mm (range 3 to 12) in 33 of the 35 patients (94%). The two patients with atrial electromechanical dissociation reverted to atrial fibrillation within one week. Cardioversion caused a decline in LA diameter (3.5 to 3.2 cm, \( P < .001 \)) and a rise in LV end-diastolic dimension (5.2 to 5.5 cm, \( P < .001 \)) while LV end-systolic dimension was unchanged (4.2 cm). Thereby stroke volume rose. Heart rate fell an average of 16 beats/min. Depressed cardiac output was improved + 0.84 L/min/m². Thus, in the majority of patients with acute or chronic supraventricular arrhythmias without mitral valve disease, cardioversion promptly restores effective atrial contraction, decreases LA size, and results in substantial hemodynamic benefit.

Additional Indexing Words:

- Atrial fibrillation
- Left atrial contraction
- Left atrial size
- Cardiomyopathy
- Cardioversion
- Left ventricular dimensions
- Mitral valve motion
- Left ventricular volumes
- Coronary heart disease
- Echocardiography

DIRECT CURRENT CARDIOVERSION of supraventricular arrhythmias is frequently performed in an attempt to improve cardiac performance by restoring the atrial contribution to ventricular filling. However, previous investigations have failed to demonstrate evidence of effective atrial contraction in many patients following the return of sinus rhythm by electroversion.1-5 Moreover, considerable controversy continues regarding the extent of improvement in ventricular performance resulting from the restoration of atrial transport function.1-18

Echocardiography provides a sensitive, noninvasive technique for the evaluation of atrial contraction and cardiac performance. Effective atrial contraction can be confirmed by the opening motion of the mitral leaflets,13 while cardiac function can be assessed by means of left heart chamber dimensions and derived stroke volume and cardiac index.14-20 Thus, this study was undertaken to evaluate the effects of electroversion of supraventricular arrhythmias upon atrial transport function and cardiac performance as determined by echocardiography in patients with chronic coronary artery disease or cardiomyopathy without valvular heart disease.

Methods

Thirty-five patients without clinical evidence of mitral valve disease undergoing elective direct current cardioversion of supraventricular arrhythmias were included in this study. The group was comprised of 20 males and 15 females ranging in age from 36 to 68 years with a mean age of 51. The cardiac diagnosis was cardiomyopathy in 16 cases, coronary atherosclerosis in 13, hypertensive cardiovascular dis-
ease in 4, atrial septal defect in 1, and 1 patient exhibited no other evidence of heart disease. The electrocardiogram revealed atrial fibrillation in 31 cases, atrial flutter in 3, and paroxysmal atrial tachycardia in 1. The duration of arrhythmia varied from 1 day to 5 years in 22 patients with a mean duration of 14 months and could not be determined in the remaining 13 patients.

Electroversion was performed according to the method of Lown utilizing a Hewlett-Packard Model 7802D defibrillator. Intravenous diazepam in doses from 0.1 to 0.5 mg/kg was the sole agent utilized for anesthesia and was given primarily for the purpose of inducing amnesia. All patients were medicated with 200–400 mg quinidine intramuscularly 30 minutes prior to the procedure and 28 patients were also receiving digitalis. All patients were alert and responsive within one hour following electroschok.

Echocardiography was performed in the supine position with an Ekoline 20 echograph utilizing a 0.5 inch diameter 2.25 MHz transducer focused at 10 cm with a repetition rate of 1000 impulses/sec. The signal from the echograph was displayed and recorded on an Electronics for Medicine DR8 multichannel oscilloscope recorder. Ultrasonic scans were obtained from apex to base and echocardiograms were recorded with rigid adherence to the technique and criteria previously stressed for determining left ventricular dimensions which provide optimal relation to left ventricular volumes. Briefly, along the left sternal border at the location of the most prominent cardiac impulse or in the third or fourth intercostal space, the transducer was directed posteriorly and then angled until the characteristic echo of the anterior mitral leaflet was recorded. The transducer was then directed slightly inferior and lateral and the echograph gain adjusted until echoes were obtained from both left ventricular septal and endocardial surfaces just below the full excursions of the mitral leaflets in the area of the chordae tendineae. The transducer was then maintained in this position and recordings were obtained at the constant respiratory phase of held mid-expiration.

Echocardiograms were carried out prior to the administration of anesthesia and one hour following cardioversion in all patients, and were also recorded immediately following cardioversion in 20 patients. Mitral valve echograms were performed in all patients and records of satisfactory quality for the determination of left heart chamber dimensions and derived stroke volume were also obtained in 12 of these patients (table 1).

All echocardiograms were calibrated and measurements were taken before and after conversion from identical areas of the cardiac chambers as confirmed by echographic scans. Amplitude of the “A” wave of the mitral valve echo was measured from the onset to the peak of the maximal “A” deflection recorded from the anterior leaflet. Although a previous study has demonstrated the correlation of left atrial size determined echographically with that determined angiographically utilizing measurements obtained in ventricular end-systole, the left atrial dimension in this study was measured at ventricular end-diastole to evaluate the size of the left atrium following atrial contraction. The left atrial dimension in end-diastole was measured from the inner border of the posterior wall of the aorta to the endocardial surface of the posterior left atrial wall 0.04 sec after the onset of the QRS complex in the initial sector traversed during vertical scanning in an inferior to superior arc through the aorta and left atrium. Examination of identical areas of the left atrium was confirmed by vertical scanning and/or by identification of aortic leaflets as landmarks. The endocardial left ventricular end-diastolic dimension (EDD) was measured 0.04 sec after onset of the QRS complex and the left ventricular end-systolic dimension (ESD) was determined by the nearest approximation of the septal and posterior internal walls during systole. Left ventricular volumes in end-diastole and end-systole were calculated as the cube of the respective dimensions. Stroke volume was the difference between these volumes; stroke volume multiplied by heart rate determined cardiac output. Heart rate was obtained from simultaneously performed electrocardiograms.

Results

Heart Rate

Although electroversion resulted in a reduction in heart rate in 32 of the 35 patients studied, the change was usually minor (table 1 and fig. 1A). Thus, mean heart rate for the group prior to cardioversion of

| Table 1 |
| Cardiac Measurements Before and After Electroversion |

<table>
<thead>
<tr>
<th>Patient</th>
<th>Heart rate (beats/min)</th>
<th>Left atrial dimension (cm)</th>
<th>End diastolic dimension (cm)</th>
<th>End systolic dimension (cm)</th>
<th>End diastolic volume (cc)</th>
<th>End systolic volume (cc)</th>
<th>Cardiac index (L/min/m²)</th>
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<tr>
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<td>4.2 4.2</td>
<td>150 168</td>
<td>86 84</td>
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90 ± 9 (SEM) beats/min was reduced to 74 ± 3 beats/min (P < .01) following the restoration of sinus rhythm.

"A" Wave Amplitude

Normal diastolic motion of the anterior mitral leaflet consists of an initial rapid anterior movement as the leaflet opens at the onset of diastole, and a second forward movement as the leaflet opens in response to atrial contraction. Both anterior deflections are followed by posterior motion as the leaflet floats toward a closed position in response to ventricular filling. In the presence of slow heart rates, the mitral valve may open slightly wider during the interval between these two anterior deflections. However, this motion is gradual and of small amplitude and thus may easily be distinguished from the motion caused by atrial contraction. Although the resulting "M" shape configuration may be distorted by the presence of mitral stenosis or rapid heart rates, in the absence of these conditions atrial contraction invariably produces an "A" deflection in the mitral valve echogram; thus the "A" wave may be utilized as an indicator of effective atrial mechanical function. Figure 2 illustrates the echogram of a patient with marked sinus arrhythmia with junctional escape beats. Note that when the P wave of the electrocardiogram occurs in normal relation to the QRS, an "A" deflection is recorded on the mitral valve echogram. When atrial activation does not precede ventricular...
depolarization, an “A” deflection is not registered.

Echograms of the anterior mitral leaflet revealed minor random oscillations in all 35 patients prior to cardioversion. These random oscillations or flutter-fibrillatory waves could be distinguished from “A” waves by their smaller and variable amplitude, random occurrence, or occurrence synchronous with flutter waves on the electrocardiogram. Following electroversion, prominent “A” waves were recorded in 33 of 35 patients. Figure 3 shows the ultrasound tracing obtained in one patient prior to and following successful electroversion. The electrocardiogram in the top panel reveals atrial fibrillation which had been the documented cardiac rhythm for four years, while the echogram of the anterior mitral leaflet demonstrates the absence of “A” waves. The bottom panel, obtained one hour after cardioversion, reveals normal sinus rhythm and the presence of prominent “A” waves in the mitral valve echogram. In contrast, figure 4 shows the ultrasound tracing of one patient in whom the return of normal sinus rhythm was not accompanied by an “A” deflection of the anterior mitral leaflet. Figure 1B illustrates the mean value of the amplitude of the “A” deflection for the entire group of patients studied. The “A” wave amplitude ranged from 3 to 12 mm and the mean value for the group was 7.5 mm ± 0.76 ($P < 0.001$). Twenty-eight of the 33 patients exhibiting atrial contraction on mitral echogram remained in sinus rhythm for three weeks and 18 sustained conversion for at least six months. The two patients who manifested electromechanical dissociation reverted to atrial fibrillation within one week.

Left Atrial Dimension

The “A” deflection in the mitral echogram indicates the presence of atrial contraction; however, actual forward movement of the posterior left atrial wall in atrial systole may frequently be difficult to identify on echogram. Figure 5 demonstrates the return of prominent contractions of the atrial wall following electroversion in one of our patients. The panel on the left illustrates the echogram through the plane of the aorta with the left atrium posteriorly, obtained during atrial fibrillation. No contractions of the left atrial posterior wall are noted and the average left atrial dimension in end-diastole is 3.2 cm. The panel on the right, obtained from a similar echo plane confirmed by scanning, reveals normal sinus rhythm with prominent contractions of the left atrial wall with an

![Figure 3](image-url)

_Echocardiograms obtained pre (top panel) and post (bottom panel) electroversion in patient WD with idiopathic cardiomyopathy and left bundle branch block. Atrial fibrillation prior to conversion was unaccompanied by “A” waves on the mitral valve echogram, although slightly wider mitral opening following rapid ventricular filling is noted (arrows). The restoration of normal sinus rhythm postcardioversion resulted in return of “A” deflections of the mitral valve (arrow). AMV = anterior mitral leaflet; PMV = posterior mitral leaflet; IS = interventricular septum._

![Figure 4](image-url)

_Echocardiogram obtained in patient CC with sick sinus syndrome in whom the restoration of normal sinus rhythm following electroversion was not accompanied by concomitant appearance of “A” waves of the anterior mitral leaflet (arrow). V1 = ECG precordial lead V1._

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attendant left atrial dimension of 2.9 cm. This motion of the posterior left atrial wall following direct current cardioversion was noted in one-third of our patients.

In contrast, figure 6 illustrates the echo traversing the plane of the left atrium in another patient in whom left atrial contractions were not visualized following cardioversion. Despite the lack of diastolic motion of the atrial wall, the average left atrial dimension of 3.5 cm prior to cardioversion was reduced to 3.2 cm following electroversion.

**Figure 5**

Pre (left panel) and post (right panel) electroversion echocardiograms through the plane of the aorta and left atrium (LA) in a patient with an atrial septal defect. The area of the left atrium examined is that immediately superior to the sector of the anterior mitral leaflet and was confirmed to be similar both before and after conversion by echographic scans. Following the return of normal sinus rhythm, prominent anterior movement of the left atrial posterior wall during atrial systole was noted (arrows) resulting in diminution of LA end-diastolic dimension. AO = ascending aorta.

**Figure 6**

Echogram through the plane of the aorta and left atrium pre (left panel) and post (right panel) cardioversion in patient MT with coronary disease in whom movement of the left atrial wall was not observed during atrial systole following return of normal sinus rhythm. Portions of the aortic leaflets are noted in both panels which confirm identical areas of measurement before and after electroversion.
The average decrease in left atrial dimension in response to cardioversion for the group of patients studied is shown in table 1 and figure 1C. The left atrial dimension at end-diastole of 3.5 ± 0.21 cm prior to cardioversion was significantly reduced (P < .001) to a mean value of 3.2 ± 0.19 cm following the restoration of normal sinus rhythm.

**Left Ventricular Dimensions**

Left ventricular dimensions in end-systole and end-diastole were determined on echogram from the sector just below the mitral leaflets. The respective ventricular volumes, obtained as a cube function of these dimensions, have been demonstrated to correlate closely with those recorded by cineangiography.18-20

Figure 7 demonstrates the left ventricular dimensions obtained prior to and following electroversion in a patient with atrial fibrillation of 2 years duration. During atrial fibrillation, the end-diastolic dimension averaged 4.4 cm while the end-systolic dimension was 3.1 cm. Upon restoration of normal sinus rhythm, the end-diastolic dimension increased to 4.8 cm, while the end-systolic dimension increased to 3.4 cm, which represented an increase in stroke volume from 55.4 to 71.2 cc.

Table 1 and figure 8 illustrate the mean values for left ventricular dimensions pre and postelectroversion for our group of patients. End-diastolic dimension increased significantly from 5.2 ± 0.22 to 5.5 ± 0.28 cm (P < .001) following cardioversion (fig. 8A). End-systolic dimension, meanwhile, exhibited no significant change, being 4.2 ± 0.26 pre and 4.2 ± 0.23 cm postcardioversion (fig. 8B).

The effects of the changes in ventricular dimensions on derived ventricular volumes are shown in table 1 and figure 9. End-diastolic volume pre and postcardioversion is indicated by the top line, while end-systolic volume is shown on the bottom line with the resultant stroke volume demonstrated by the diagonal lines. Calculated end-diastolic volume increased significantly from 150 ± 18 to 168 ± 16 cc (P < .001) following electroversion, while end-systolic volume remained unaltered, 86 ± 15 to 84 ± 14 cc. Thus, stroke volume increased significantly after the return of normal sinus rhythm and atrial contraction from 64 ± 6 to 84 ± 5 cc (P < .001).

**Figure 7**

Pre (top panel) and post (bottom panel) cardioversion echograms through the plane of the left ventricle just below the maximal excursion of the mitral valve leaflets in patient MT with coronary disease. Similar echoes are obtained in both tracings confirming the similarity of the areas examined. Following electroversion, there was an increase in both end-diastolic and end-systolic ventricular dimensions. LVW = left ventricular posterior wall; MV = mitral valve.

**Figure 8**

Average left ventricular end-diastolic (A) and end-systolic (B) dimensions pre and postcardioversion.
Cardiac Index

The response of the mean cardiac index to electroversion for all patients in this study is shown in table 1 and figure 10. Although cardiac index was augmented in 9 of the 12 patients, resulting in an increase in the mean volume from 3.00 ± 0.36 to 3.32 ± 0.22 L/min/m², this change was not statistically significant (fig. 10A). However, when the response to electroversion was analyzed for the subgroup of 5 patients whose control cardiac index was below normal (less than 2.5 L/min/m²), cardiac index increased in all patients and resulted in a significant increase in mean values from 2.13 ± 0.23 to 2.86 ± 0.33 L/min/m² (P < .01) (fig. 10B).

Discussion

The importance of atrial contraction to ventricular filling was first recognized by William Harvey in 1628. Subsequent experimental and clinical studies have confirmed the role of the atria as “booster pumps” which contribute substantially to cardiac output. Thus, the advent of the ability to convert supraventricular arrhythmias to sinus rhythm safely and practically by direct current cardioversion was accompanied by the expectation that atrial mechanical function would return concomitantly with normal electrical activation and would thereby benefit cardiac hemodynamics. However, recent studies have failed to substantiate the restoration of atrial contraction following successful electroversion during the early postconversion period in a large percentage of patients. In addition, previous investigations have not yielded uniform conclusions regarding the benefit conferred upon ventricular performance by the return to normal sinus rhythm. The present study, utilizing echocardiography, demonstrates that atrial transport function returns promptly following electroversion and may provide a significant increment in cardiac output in patients with reduced ventricular function without primary left ventricular volume overloading.

Prior investigations have employed a variety of methods to detect the return of atrial contraction following cardioversion. Thus, diminutive or absent “A” waves have been noted in left atrial pressure recordings, pulmonary wedge pressure recordings and external precordial pulse tracings during the immediate postelectroversion period in the majority of patients studied. In contrast, one recent study, utilizing kinetocardiography, demonstrated the prompt return of atrial mechanical activity in nearly all patients. Echocardiography, because it can record opening of the mitral leaflets in response to atrioventricular flow, provides a particularly sensitive method for the detection of atrial contraction. Indeed, even the minimal augmentation of ventricular filling induced by atrial contraction which occurs in response to atrial flutter or coarse atrial fibrillation is capable of producing a deflection in the mitral valve echogram. Thus, it is not surprising that ultrasound may be capable of detecting atrial mechanical activity that would go unrecognized by other modalities. Therefore, although our population differs from that of most earlier reports in that we excluded patients with mitral valve disease, the detection of “A” wave deflections in 33 of 35 patients in the first hour following electroversion appears to indicate that atrial transport function is restored without delay more frequently than has been previously recognized.
Although mitral valve diastolic contour on echogram may be altered by heart rate and diastolic filling, thereby causing a diminution of "A" wave deflection, the mean value for postconversion "A" amplitude of 7.5 mm is less than the 13 mm amplitude obtained in normal subjects in our laboratory; thus the postconversion amplitude likely represents decreased force of left atrial contraction.

Two patients in this study failed to exhibit evidence of atrial contraction on mitral echogram despite the appearance of prominent P waves in ECG upon the return of normal sinus rhythm (fig. 4). One of these patients manifested the typical features of the sick sinus syndrome, and atrial fibrillation had been present in both subjects for over two years. Sinus rhythm was sustained for only one week in each case. By comparison, 84% of our patients exhibiting atrial contraction on mitral echogram remained in sinus rhythm for three weeks and 54% sustained conversion for at least six months. Thus, although our number of patients is quite small, it appears that atrial electromechanical dissociation may convey a poor prognosis for the maintenance of sinus rhythm and warrants further evaluation as a prognostic indicator.

The reduction in left atrial size and increase in left ventricular end-diastolic dimension noted in this study (figs. 1C, 6–8), even in patients with a postconversion reduction of heart rate of less than 10 beats/min, suggest that the atrial contractions observed in our patients were responsible for a substantial increase in atrioventricular flow. Although the reduction in atrial dimension was most marked in those patients manifesting diastolic motion of the left atrial wall during atrial systole, it was noted in all but one of the remaining patients as well. Left ventricular performance was thereby enhanced by means of the greater volume of blood available for ejection and augmentation of contractile force by means of the Frank Starling mechanism.

Echocardiography affords a reliable means for the determination of left ventricular dimensions. Moreover, ventricular volumes derived from these measurements have been shown to correlate closely with those obtained by cineangiography by several investigators. However, it should be noted that the echocardiographic beam may traverse a limited area of the ventricle which correlates poorly with actual ventricular volumes. This difficulty may be encountered in dilated hearts and in coronary heart disease with segmental abnormalities of ventricular contraction. Nevertheless, in a patient study of an intervention such as the present investigation, evaluation of directional changes remains valid since the individual serves as his own control.

The finding of augmented stroke volume (table 1 and fig. 9) is in agreement with previous studies, although it differs somewhat from the findings of Graettinger et al. who could relate an increase in stroke volume only to a diminution of heart rate. Although increase in stroke volume can result solely from a decrease in heart rate, the observation in our study of similar increments in patients with a decline in heart rate of less than 10 beats/min suggests that the return of atrial contraction played a role in the augmentation of stroke volume. Although stroke volume increased significantly in our group of patients, heart rate decreased (fig. 1A). The net rise in cardiac index failed to achieve statistical significance for the entire group (fig. 10A). However, an already normal cardiac output would not be expected to improve following cardioversion despite the return of effective atrial contraction and thus evaluation of cardiac output alone is not a sensitive measure of atrial transport function. Accordingly, alterations in cardiac output induced by electroversion were analyzed for those patients with preconversion depression of cardiac index below 2.5 L/min/m² and revealed a uniform improvement in all of the patients in this subgroup (fig. 10B). Thus, considerable benefit accrued to our patients with reduced cardiac function following the electroversion of supraventricular arrhythmias.

The response of chamber dimension to electroversion was subsequently examined in each patient in reference to the amplitude of the mitral "A" wave and the preconversion left ventricular end-diastolic volume. The "A" wave amplitude on mitral echogram induced by conversion to normal sinus rhythm could not be correlated with changes in left atrial and ventricular chamber size. It is likely that this lack of correlation reflects the role of heart rate and early diastolic ventricular filling in determining the movement of the mitral leaflets and thus the prominence of the "A" deflection of the mitral echogram. Chamber size, in the one patient with electromechanical dissociation in whom it was recorded, showed no change following cardioversion. In contrast to the lack of correlation between amplitude of mitral "A" wave deflection and hemodynamic benefit, the patients with the greatest left ventricular end-diastolic volumes manifested the least increment in cardiac performance. Therefore, the return of atrial transport function was least efficacious in the presence of left ventricular enlargement. It is postulated that postconversion hemodynamic benefit is small in patients with markedly dilated hearts, even with low initial cardiac output, since the ventricles may be operating near the apex of depressed and flattened function curves with their Starling reserve mechanisms nearly expended. It was not possible to fully test this hypothesis in the present study, since our patient sub-
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group with low initial cardiac output had only mild to moderate ventricular enlargement. However, the present findings and mechanisms suggested are consistent with previous reports which indicate that improvement in pump performance usually does not result from cardioversion in patients with primary abnormalities of left ventricular volume overloaded such as in chronic mitral regurgitation.

In conclusion, this study demonstrates that atrial contraction, as manifested by the “A” wave deflection of the mitral echogram, promptly returns following electroversion of supraventricular arrhythmias in nearly all patients without mitral valve disease. In addition, the return of atrial contraction is accompanied by an increase in left ventricular filling as indicated by a decrease in left atrial dimension and an increase in left ventricular end-diastolic dimension. These alterations suggesting augmented left ventricular filling as a consequence of atrial contraction occurred in all patients and were reflected by an increase in cardiac output in patients with reduced cardiac output prior to electroversion.

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