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# Electromechanical correlation of left atrial function after cardioversion

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*To study the left atrial function after cardioversion, left ventricular apex cardiogram, electrocardiogram, unipolar and bipolar oesophageal electrocardiogram, and cardiac catheterization were done after 39 procedures in 37 patients with rheumatic heart disease and 2 with atrial septal defect. An 'a' wave in the apex cardiogram representing left atrial contraction appeared in 31 of the 32 records taken 8 hours after cardioversion. Oesophageal electrocardiograms were recorded in 18 patients and in each instance showed separate right and left atrial components of P wave in the unipolar records and large left atrial waves in the bipolar records. In the pressure tracings an 'a' wave appeared in 19 of the 20 patients in the right atrium and 13 of the 16 subjects in the pulmonary arterial wedge immediately (20 to 40 minutes) after cardioversion. The mean pulmonary arterial wedge pressure decreased after cardioversion in each case. In spite of this the left ventricular stroke work index increased relatively in 12 of the 15 patients, showing improved cardiac performance. It is concluded that there is no true disparity between electrical and mechanical activity of the left atrium, and both are usually restored soon after cardioversion.*

Electrical cardioversion to restore normal sinus rhythm is now an accepted elective procedure and is widely practised (Lown, 1967; Resnekov and McDonald, 1968). It has however been suggested that normal atrial electrical activity may not be accompanied by significant left atrial mechanical activity (Braunwald, 1964), that this may be more usual in rheumatic heart disease (Logan *et al.*, 1965), and it may appear 3 to 6 days after cardioversion (Ikram, Nixon, and Arcan, 1967, 1968).

The present study was designed to assess the return of mechanical activity of the left atrium after cardioversion by left ventricular apexcardiogram and correlate it with the return of electrical activity in left atrium and with 'a' waves of the right and left atrial pressure pulses as recorded during cardiac catheterization.

## Material and methods

Studies were done after 39 cardioversion procedures. There were 37 patients with rheumatic heart disease; 35 had undergone closed mitral valvotomy and two were unoperated upon with moderate degree of mitral regurgitation. Eight of the operated cases had significant mitral regurgitation and one had associated aortic regurgitation at the time of cardioversion. The remaining two had atrial septal defect.

Of the 39 patients, 26 were men and 13 were women. Their age and sex distribution is shown in Table 1 and duration of atrial fibrillation in Table 2.

TABLE 1 *Age and sex distribution*

| *Age* | *Men* | *Women* | *Total* |
|---|---|---|---|
| 19 and below | 2 | 3 | 5 |
| 20–29 | 9 | 1 | 10 |
| 30–39 | 9 | 4 | 13 |
| 40–49 | 4 | 6 | 10 |
| 50 and above | 1 | 0 | 1 |
| Total | 25 | 14 | 39 |

TABLE 2 *Duration of atrial fibrillation*

| *Duration* | *No.* |
|---|---|
| Less than 2 months | 3 |
| 2 to 6 months | 7 |
| 6 months to 1 year | 5 |
| 1 year to 2 years | 4 |
| More than 2 years | 5 |
| Not known correctly, but at least 6 months | 15 |
| Total | 39 |

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Standard electrocardiograms were recorded before and after cardioversion in each case. A bipolar electrode catheter was passed through the nose into the oesophagus, and oesophageal electrocardiograms were recorded at distances of 50, 45, 40, 35, and 30 cm. from the tip of the nares in 18 patients. Unipolar as well as bipolar tracings were recorded. A left ventricular apex cardiogram was recorded at the point of maximal impulse with the patient in a left lateral position during mid-expiration (Dimond, Duenas, and Benchimol, 1966) in 37 instances. Using a funnel with a diameter of 2·0 cm. and piezoelectric crystal microphone (Sanborn No. 374), the tracings were recorded at a paper speed of 75 mm. per second on a multichannel photographic recorder (Electronics for Medicine, DR 8).

Right heart catheterization was done in 20 patients. Pressure tracings of right atrium and pulmonary artery wedge reflecting left atrial pressure were recorded. Baseline for all pressure measurements was taken as half the chest thickness at the second costal cartilage with the patient supine (Roy, Gadboys, and Dow, 1957). Cardiac output was measured by dye dilution technique at rest and during the 3rd to 5th minutes of a steady leg-raising exercise in supine position, both before and after cardioversion.

Cardioversion was done under transient amnesia induced by a small dose of intravenous thiopentone sodium (50 to 200 mg.) as described earlier from this laboratory (Wasir *et al.*, 1969).

Surface electrocardiogram, oesophageal electrocardiogram, and apex cardiogram were repeated 8 hours after cardioversion when the patient had recovered from the effect of anaesthesia. In five instances it was recorded 1 hour after cardioversion.

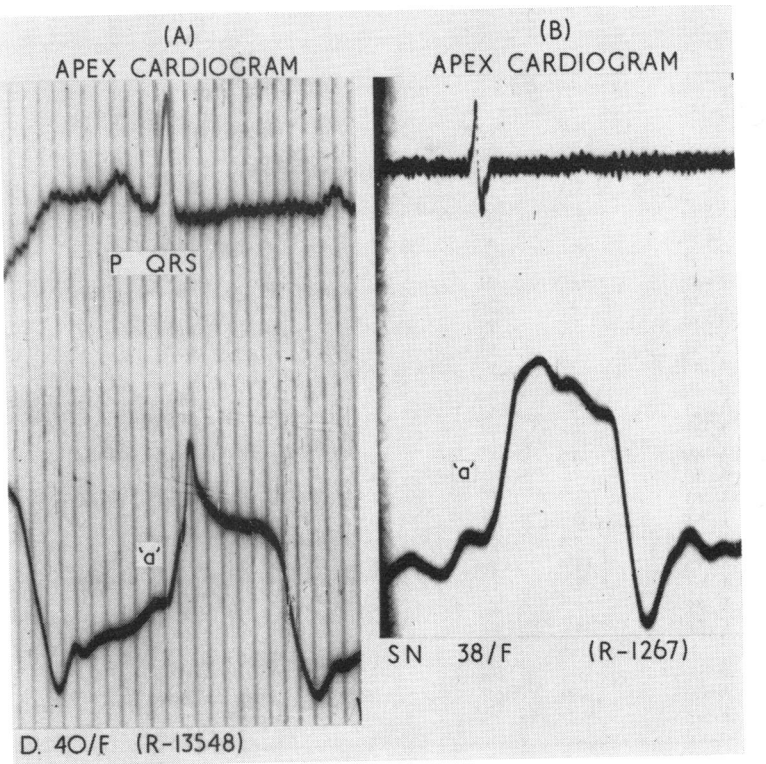



FIG. 1 *Apex cardiograms in two patients immediately after cardioversion showing a distinct 'a' wave representing left atrial contraction.*

## Results

**Apex cardiogram** Out of the 37 instances where apex cardiography was available, 5 were recorded 1 hour and 32 were recorded 8 hours after cardioversion. Of the former, 1 was technically unsatisfactory owing to obesity in a female patient, 2 showed the presence of 'a' wave, and in 2 it was absent. In both it appeared after 24 hours. Of the 32 records taken after 8 hours an 'a' wave was present in 31 and absent in 1, and in this case it appeared after 24 hours. In most cases the 'a' waves were very small.

Two representative tracings are shown in Fig. 1A and 1B.

**Surface electrocardiogram** A P wave was present in the surface electrocardiogram in all cases. In no case were persistent fibrillary waves seen in addition to the P waves.

**Oesophageal electrocardiogram** Oesophageal electrocardiograms were available in 18 cases, and in every case a P wave was present without additional fibrillary waves. Separate components showing right and left electrical atrial activity in the form of notched and bifid P were present in each unipolar tracing, and a large left atrial component could be shown in each bipolar record. Representative oesophageal electrocardiograms are shown in Fig. 2 and 3.

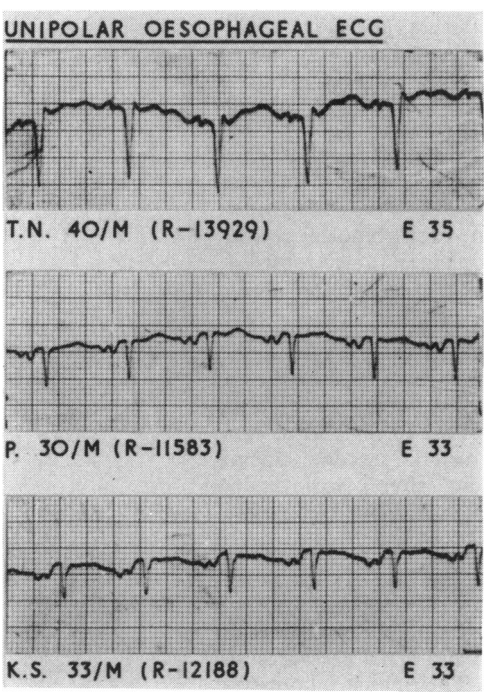
**Atrial pressure tracings** In 19 of the 20 cases where it was recorded an 'a' wave in the right atrial pressure tracing appeared after cardioversion. In 13 of the 16 cases an 'a' wave in the pulmonary arterial wedge tracing appeared immediately after cardioversion (Fig. 4). It was absent or indistinct in 3 cases.

**Haemodynamic data** The cardiac index, stroke index, and stroke work index of all these patients before and after cardioversion along with heart rate and atrial pressures are presented in Table 3. The right atrial pressure

TABLE 3 *Haemodynamic data before and after cardioversion*

| | *RA rest (mm. Hg)* | | | *PAW (mm. Hg)* | | | | | *Heart rate (beats/min.)* | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *AF* | *NSR* | *'a'* | *Rest* | | | *Exercise* | | *Rest* | | *Exercise* | |
| | | | | *AF* | *NSR* | *'a'* | *AF* | *NSR* | *AF* | *NSR* | *AF* | *NSR* |
| C/33 | 20 | 19 | + | 27 | 20 | + | | | 108 | 90 | | |
| C/34 | 5 | 4·5 | + | 8 | 8 | + | | | 70 | 60 | | |
| C/35 | 6 | 5 | + | 18 | 15 | + | 25 | 20 | 75 | 85 | 120 | 100 |
| C/39 | 14 | 7 | + | 28 | 26 | + | | | 150 | 108 | | |
| C/40 | 4 | 3·5 | + | — | — | | | | 80 | 90 | 102 | 120 |
| C/42 | 7·5 | 7 | + | 24 | 21 | ? | 40 | 30 | 135 | 80 | 180 | 90 |
| C/43 | 7 | 5 | + | 12 | 10 | + | 24 | 18 | 110 | 100 | 150 | 135 |
| C/45 | 9 | 7 | + | | | | | | 150 | 72 | | |
| C/46 | 4 | 4 | + | 19 | 14 | + | 40 | 20 | 80 | 90 | 180 | 132 |
| C/47 | 5 | 5 | + | 12 | 11 | + | 24 | 18 | 100 | 72 | 150 | 96 |
| C/48 | 6 | 5 | + | | | | | | 120 | 100 | | |
| C/49 | 3·5 | 3 | + | 15 | 8 | + | | | 90 | 80 | 120 | 85 |
| C/50 | 5 | 5 | + | 15 | 14 | + | 26 | 23 | 75 | 60 | 90 | 80 |
| C/53 | 5 | 5 | + | 17 | 16 | + | 25 | 22 | 60 | 60 | 72 | 80 |
| C/54 | 8 | 8 | + | 19 | 17 | + | 28 | 24 | 70 | 70 | 120 | 100 |
| C/55 | 5·5 | 5 | + | 13 | 10 | + | | | 87 | 78 | | |
| C/63 | 13 | 12 | + | — | | | | | 92 | 75 | | |
| C/65 | 6 | 5 | + | 15 | 11 | 0 | 25 | 18 | 60 | 60 | 90 | 80 |
| C/69 | 7 | 4 | + | 11 | 7 | + | 23 | 19 | 60 | 80 | 75 | 92 |
| C/81 | 6 | 3 | ? | 13 | 10 | ? | 31 | 26 | 100 | 84 | 138 | 120 |
| Average | 7·3 | 6·1 | | 16·7 | 13·7 | | 28·3 | 21·6 | 93·6 | 79·7 | 122·1 | 100·8 |
| SE | 0·9 | 0·8 | | 1·40 | 1·3 | | 1·8 | 1·2 | 6·3 | 3·2 | 10·1 | 5·5 |
| P | <0·005 | | | <0·001 | | | <0·005 | | <0·02 | | <0·02 | |

FIG. 2 *Unipolar oesophageal electrocardiograms of 3 different patients showing right and left atrial components of P wave.*




had fallen from an average of 7·3 mm. Hg to 6·1 mm. Hg. Pulmonary arterial wedge pressure at rest had decreased from an average of 16·7 to 13·7 mm. Hg and during exercise from 28·3 to 21·6 mm. Hg. The cardiac index at rest was 3·77 l./min./m.$^2$ before and 3·83 l./min./m.$^2$ after cardioversion and during exercise 5·58 l./min./m.$^2$ before and 5·20 l./min./m.$^2$ after cardioversion. The resting stroke volume index was 46·1 ml./beat/m.$^2$ before and 51·5 ml./beat/m.$^2$ after cardioversion. The exercise values were 50·9 and 51·6 ml./beat/m.$^2$, respectively. The resting left ventricular stroke work index was 63·0 before and 68·0 g.m./beat/m.$^2$ after cardioversion, and the exercise values were 77·6 and 71·7 g.m./beat/m.$^2$, respectively.

The changes in right atrial and pulmonary arterial wedge pressures were highly significant, but changes in cardiac index, stroke index, and stroke work index were not significant.

## Discussion

The present study shows that after successful cardioversion fibrillary activity ceases in both the atria and electrical activity indicating normal sinus mechanism in both atria returns. Though this has been generally believed to be true, we have not come across any studies

| *Cardiac index (l./min./m.²)* | | | | *Stroke index (ml./beat/m.²)* | | | | *LV stroke work index (g.m./beat/m.²)* | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| *Rest* | | *Exercise* | | *Rest* | | *Exercise* | | *Rest* | | *Exercise* | |
| *AF* | *NSR* | *AF* | *NSR* | *AF* | *NSR* | *AF* | *NSR* | *AF* | *NSR* | *AF* | *NSR* |
| 4·24 | 4·45 | | | 41 | 50 | | | 55 | 72 | | |
| 2·58 | 1·67 | | | 37 | 28 | | | 42 | 39 | | |
| 3·80 | 3·90 | 5·30 | 4·10 | 50 | 44 | 45 | 41 | 68 | 58 | 67 | 66 |
| | | 6·70 | 6·40 | | | 40 | 35 | | | 58 | 40 |
| 3·05 | 3·25 | | | 23 | 41 | | | 27 | 51 | | |
| 3·16 | 2·95 | 4·94 | 3·90 | 29 | 30 | 33 | 29 | 39 | 49 | 45 | 57 |
| 3·25 | 3·45 | 3·77 | 4·27 | 39 | 47 | 25 | 44 | 71 | 79 | 46 | 73 |
| 2·30 | 2·50 | | | 19 | 25 | | | 32 | 41 | | |
| | | 3·90 | 2·96 | | | 33 | 37 | | | 53 | 50 |
| 3·10 | 3·90 | 4·10 | 4·40 | 41 | 65 | 46 | 55 | 52 | 71 | | |
| 4·12 | 5·20 | 5·60 | 6·00 | 68 | 86 | 78 | 75 | 94 | 88 | 102 | 80 |
| 6·80 | 7·70 | 8·05 | 7·00 | 97 | 110 | 72 | 70 | 133 | 141 | 102 | 98 |
| 3·32 | 1·71 | | | 38 | 22 | | | 46 | 33 | | |
| 5·80 | 5·00 | | | 63 | 62 | | | 90 | 89 | | |
| 3·10 | 3·90 | 4·40 | 4·10 | 52 | 65 | 49 | 49 | 74 | 77 | 91 | 63 |
| 2·35 | 2·00 | 5·20 | 3·65 | 39 | 25 | 71 | 46 | 51 | 34 | 108 | 57 |
| 5·60 | 6·14 | 9·40 | 10·40 | 56 | 73 | 68 | 87 | 70 | 97 | 104 | 133 |
| 3·77 | 3·83 | 5·58 | 5·20 | 46·1 | 51·5 | 50·9 | 51·6 | 63·0 | 68·0 | 77·6 | 71·7 |
| 0·34 | 0·43 | 0·54 | 0·65 | 5·5 | 6·6 | 5·5 | 5·6 | 7·2 | 7·5 | 8·3 | 8·53 |
| NS | | NS | | NS | | NS | | NS | | NS | |

where a left atrial electrocardiogram has been specifically recorded after cardioversion. An oesophageal electrocardiogram in the present study was recorded in 18 cases and in each case separate left and right atrial components could be identified. This technique has been employed for several years (Burch and Winsor, 1966; Goldman, 1967) to delineate the atrial activity more clearly than routine surface electrocardiography, and recently Massumi *et al.* (1969) have emphasized its importance in studying left atrial activity. In the bipolar oesophageal electrocardiogram the left atrial component of the P wave is greatly magnified, as shown in Fig. 3, and this component was clearly seen in each case.

We have not come across any reports where one atrium has continued to fibrillate after cardioversion with the routine surface electrocardiogram showing sinus rhythm, neither have we found one in the present series. It is possible that in some of the cases of 'failure' where surface electrocardiography showed persistent atrial fibrillation one of the two atria may be beating regularly. Such dissociation is known in atrial fibrillation, and recently its frequency has been emphasized (Nelson, Jenson, and Davis, 1969), though in most cases the rate of 'regular' electrical activity of one atrium is above 200/min.

Logan *et al.* (1965) reported that after cardioversion there may be disparity between electrical and mechanical functions. In 3 out of 5 cases they did not find a definite 'a' wave in the pressure pulse of the left atrium immediately after cardioversion (though it appeared in 10 minutes in one), and this was interpreted as absence of significant mechanical contraction. It has been reported (Macieira-Coelho *et al.*, 1968) that in dogs an 'a' wave may not return immediately after cardioversion in left atrial pressure pulse, but the authors did not mention how early the data were recorded. It is known that haemodynamics improve in 2 to 3 hours after cardioversion (Rodman, Pastor, and Figueroa, 1966; Scott and Patterson, 1969).

It can be argued that the presence of an 'a' wave in the pressure pulse is not the only reliable evidence of effective left atrial contraction. In a case with a very large left atrium an 'a' wave in the pressure tracing may not be very prominent, though increased flow through the mitral valve may occur during atrial contraction. The best method therefore should be to study flow through the mitral valve continuously or measure the left ventricular diastolic volume continuously. Apex cardiography has been shown to represent diastolic events of the left ventricle very

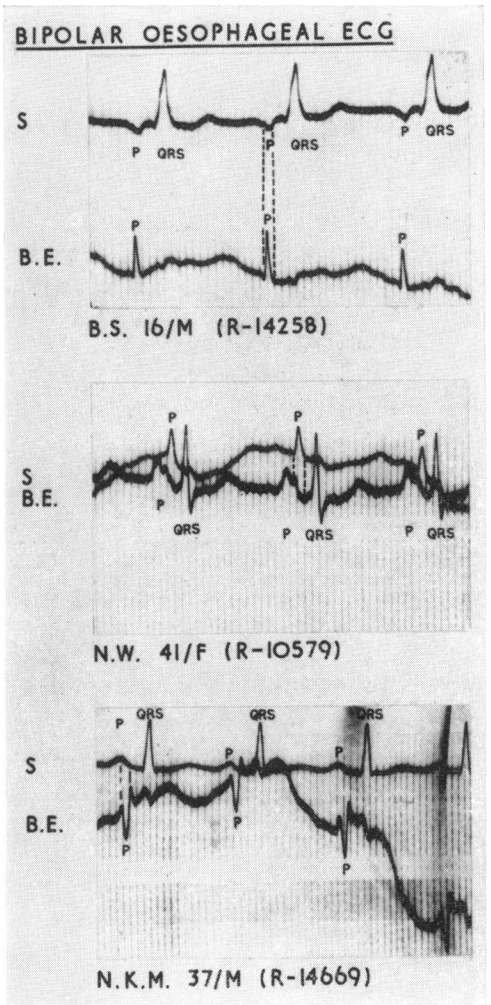


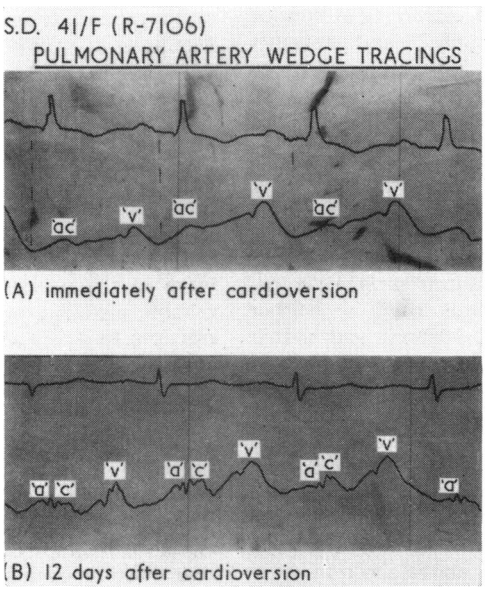

FIG. 3 *Bipolar oesophageal electrocardiograms of 3 patients showing in each case a greatly magnified left atrial component of P wave in the oesophageal electrocardiogram.*

faithfully, and diastolic waves of the apex cardiogram correspond to the ventricular filling pattern (Benchimol and Dimond, 1963; Tafur, Cohen, and Levine, 1964; Epstein *et al.*, 1968). Recently the apex cardiogram has also been shown to have good correlation with left ventricular volume (Sutton, Prewitt, and Craige, 1970). The presence of an 'a' wave in the apex cardiogram is evidence that increased flow has occurred into the left ventricle after atrial contraction, and therefore is a better indicator of mechanical activity of left atrium.

This study shows that in practically every case where surface electrocardiography showed sinus rhythm there was evidence of left atrial contraction in the form of an 'a' wave in the apex cardiogram 8 hours after cardioversion. These findings are different from those of Ikram *et al.* (1968), who found 'a' waves to be absent in 7 of their 12 cases immediately after cardioversion, though they appeared 3 to 6 days later in 6 of the 7 cases. These workers as well as Logan *et al.* (1965) thought that the disparity was greater in rheumatic heart disease. Their results and those of the present series are compared in Table 4.

Correlation of the apex cardiogram with haemodynamic studies was done in 20 cases. Almost uniform appearance of 'a' waves in the right atrial tracing is in agreement with others (Logan *et al.*, 1965; Rowlands, Logan, and Howitt, 1967; Shapiro and Klein, 1968). However, the presence of an 'a' wave in the pulmonary arterial wedge tracing in 13 of 16 cases is higher than that reported by Rowlands *et al.* (1967). Several others (Braunwald, 1964; Graettinger, Carleton, and Muenster, 1964; Corliss *et al.*, 1968) have commented on the diminutive nature of these 'a' waves without specifically mentioning that they were absent in some. The absence of 'a' waves in the pulmonary arterial wedge tracing in 3 cases out of 16 could either be explained by the genuine absence of an 'a' wave in the

FIG. 4 *Pulmonary artery wedge tracing (A) immediately and (B) 12 days after cardioversion showing 'a' wave.*



left atrium or as due to artefact, the pulmonary arterial wedge tracing being only a reflected tracing.

The assessment of haemodynamic status immediately after cardioversion has posed a problem in the present study as it has in most other series. Depending on the dose of anaesthetic used for cardioversion and individual response to it, some patients had completely recovered in 20 to 40 minutes, whereas others had not. Though every patient was awake, the majority were feeling a little sleepy. It is therefore surprising that even in this state 9 of the 15 cases showed an increase in stroke work index of the left ventricle without any increase in mean pulmonary arterial wedge pressure. Three others showed no change in stroke work, but mean pulmonary arterial wedge pressure had decreased from the control period. Increased stroke work without increase in mean left atrial pressure shows the contribution of atrial contraction in these cases (Braunwald and Frahm, 1961). There is therefore strong evidence to suggest that mechanical function of the left atrium had been restored immediately after cardioversion in at least 12 of the 15 cases.

An effort to quantitate the apex cardiogram changes by correlating the height of the 'a' wave to haemodynamic improvement has not been successful, as no linear correlation has been found with either cardiac index, stroke index, or stroke work index. This is not surprising, as the apex cardiogram is more of a qualitative record.

It is difficult to explain the differences in the present study compared with earlier reports (Logan *et al.*, 1965; Ikram *et al.*, 1968). If the suggestion of Ikram *et al.* (1968) that left atrial contraction does not appear till 3 to 6 days after cardioversion in half of all cases is accepted, it is very difficult to explain the almost uniform findings of others (Morris *et al.*, 1965; Reale, 1965; Killip and Baer, 1966; McIntosh, Kong, and Morris, 1964; Shapiro and Klein, 1968) as well as our own indicating immediate haemodynamic improvement after cardioversion in the majority of the cases and the reduction of mean left atrial pressure in every case. A possible explanation for the different findings in the present study may be the duration of atrial fibrillation. It was more than 5 years in all the cases of Ikram *et al.* (1968), but less than 2 years in the majority of our cases and probably less than 5 years in each case.

Another point worth emphasizing is that in many of our cases much time and effort were needed to bring out clear 'a' waves in the apex cardiogram. In many artefactual and unsatisfactory records no 'a' wave was seen, but more carefully recorded tracings invariably showed their presence.

TABLE 4 *Atrial activity after cardioversion*

| | *Logan et al. (1965)* | *Rowlands et al. (1967)* | *Ikram et al. (1968)* | *Present study* |
|---|---|---|---|---|
| a in JVP | — | — | 12/12 | — |
| a in RA | 6/7 | 12/13 | 2/2 | 18/19 |
| a in ACG | — | — | 5/12 | 2/4 at 1 hour<br>31/32 at 8 hours<br>36/36 at 24 hours |
| a in LA or PAW | 2/5 | 5/8 | 1/2 | 13/16 |

It appears, therefore, that immediately after cardioversion left atrial contraction is present in almost every case and the evidence is in the form of a separate left atrial component in the oesophageal electrocardiogram, the presence of an 'a' wave in the apex cardiogram, the presence of an 'a' wave in the pulmonary arterial wedge tracing, though diminutive, and improvement in cardiac performance as judged by higher stroke work index without higher pulmonary arterial wedge pressure. Data from this laboratory (Shrivastava, Mathur, and Roy, 1971) show that when the effect of anaesthesia is over haemodynamic improvement becomes even more obvious, which may indicate that the quality of left atrial contraction further improves. It is therefore fair to suggest that with the passage of time the changes in the pattern of left atrial contraction after cardioversion are quantitative rather than qualitative. There is no true disparity between electrical and mechanical functions of the left atrium at any time, but it is possible that the force of left atrial contraction improves with time.

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