Hemodynamic Effects after Conversion of Arrhythmias

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ABSTRACT Systemic and coronary hemodynamic parameters were determined during an arrhythmia and immediately after a direct current transthoracic shock given in an attempt to convert the arrhythmia to a sinus mechanism. No anesthesia or drugs were administered between the two studies. 16 patients with atrial fibrillation converted to sinus rhythm and five did not. In two patients with atrial flutter and one with supraventricular tachycardia, the arrhythmia was corrected. The arrhythmia persisted in a single patient with ventricular tachycardia. Utilizing each patient as his own control, we compared statistically various hemodynamic parameters before and after the shock. In addition, the group of patients whose atrial fibrillation terminated was compared to the group treated in the same manner but in which the atrial fibrillation persisted. Pressures in the right side of the heart decreased in both groups so that the changes appeared to be caused by factors associated with the transthoracic direct current shock or the catheterization procedure. The differences between those with atrial fibrillation who converted to sinus rhythm as compared to those who did not were a decrease in heart rate, an increase in stroke volume, and an increase in cardiac efficiency. There was no immediate effect on the cardiac output or coronary blood flow.

INTRODUCTION

The effects of arrhythmias on the coronary circulation and myocardial metabolism have not been

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adequately studied in human subjects. The systemic hemodynamic effects of arrhythmias have been investigated but the observed changes have not been uniform (1-22). The cardiac output has been reported to vary from no significant change to more than doubling after conversion of atrial fibrillation to sinus rhythm (16, 9). The pressure responses as well as other indices also have varied, both in direction and degree of change (6, 8, 15).

The various methods used to evaluate these circulatory changes have been criticized (14-16). The most frequent criticisms concern the time interval between the studies performed during the arrhythmia and after its conversion, and the administration of hemodynamically active drugs, such as quinidine or general anesthesia between the two studies. Another unknown is the effect of a transthoracic direct current shock when this method is used for conversion.

The present study was performed to evaluate the effect of arrhythmias on the coronary circulation, myocardial metabolism, and cardiac efficiency. Additional information was also obtained regarding systemic hemodynamic effects of the arrhythmias and of the transthoracic direct current shock used for rhythm conversion. The protocol was devised to eliminate or control the variables mentioned above.

METHODS

Studies were performed on 25 subjects ranging in age from 23 to 59 yr. Further information concerning age, sex, arrhythmia, and diagnosis is presented in Table I, as are the results of the attempt to change their rhythm. All patients were given a maintenance dose of quinidine sulfate for 24-48 hr before the study and, if they had

FIGURE 1 Simultaneous pressures in left atrium, left ventricle, pulmonary artery and femoral artery immediately before and after the electrical conversion of atrial fibrillation in a patient with mitral stenosis. Shaded area, mitral diastolic gradient.

been taking digitalis, it was withheld for at least 24 hr.

Cardiac catheterization was performed in the postabsorptive state using 1% lidocaine hydrochloride for local anesthesia. A Cournand needle was inserted into the femoral artery and cardiac catheters were placed fluoroscopically in the pulmonary artery and coronary sinus (CS). In seven subjects, transseptal catheterizations as well as retrograde arterial (A) catheterizations were performed so, in these subjects, there were catheters in the pulmonary artery, coronary sinus, left ventricle, and left atrium throughout the study. Cardiac output was determined by the Fick principle and coronary blood flow (CBF) by the nitrous oxide saturation method. Analyses on the blood for oxygen and carbon dioxide were done by the Van Slyke-Neill method. The method of Orcutt and Waters was used for nitrous oxide (23).

The systemic arterial, pulmonary arterial, and coronary sinus blood pressures were measured in all patients with Statham P23D or DB strain-gauge pressure transducers and recorded on a Waters photographic recorder. Left atrial, left ventricular, and central aortic pressures were recorded in patients with left-sided catheterizations. Slow-period galvanometers were used for determining mean pressures. An index of cardiac efficiency was calculated in each group by relating external left ventricular work to left ventricular oxygen consumption as follows:

$$\text{Index efficiency} = \frac{\text{left ventricular work index}}{\text{left ventricular oxygen usage}}$$

When the procedure was underway and before any hemodynamic measurements were made, meperidine was

TABLE I
Clinical Data and Systemic Hemodynamic Effects on

Patient No.	Age	Sex	Diagnosis	Rhythm before shock	SVI %Δ	HR %Δ	CI %Δ
	yr				ml/m ²	beats/min	liter/min per m ²
Patients converted to sinus rhythm with DC shock							
1 E. F.	55	M	ASHD p	Atrial flutter 2:1	34.2	126	4.31
			Infarct, 4 yr		+ 19	-25	-10
2 F. T.	38	M	ASHD p	Suprevent. tach.	13.9	169	2.35
			Infarct, 3 wk		+112	-42	+23
3 E. H.	41	F	P. O. ASD	Atrial flutter	35.4	111	3.93
			No shunt		- 6	-14	-19
J. B.	42	F	MS ₄ †	AF			3.53
							+24
4 M. E.	54	F	P. O. MS	AF	17.3	129	2.23
			Res TI ₂		+ 59	-37	0
5 R. H.	48	M	MS ₃	AF	23.4	108	2.53
					+ 29	-31	-11
6 A. K.	35	F	MS ₃ , MI ₂	AF	31.9	111	3.54
					+100	-19	+62
7 H. M.	34	F	P. O. MS ₁	AF	27.9	147	4.10
					+ 44	-39	-11
8 P. K.	42	M	IHSS ₃	AF	21.5	104	2.24
					+ 39	-19	+12
9 L. M.	38	F	P.O. MI, MS	AF	16.3	118	1.93
			GDV-MI ₁ , MS ₀		+120	-47	+17
10 C. H.	38	F	P. O. MI, MS	AF	21.8	95	2.07
			GDV-MI ₁ , MS ₁		+ 57	-22	+23
11 H. G.	47	F	MS ₂	AF	18.7	131	2.45
					+ 44	-31	- 1
12 L. C.	38	F	P. O. MS	AF	46.0	85	3.91
			Res MS ₂		+ 36	- 4	+29
13 M. M.	45	F	P. O. MS ₁ , AI ₃	AF	19.5	74	1.44
					+ 31	+12	+47
14 E. W.	43	F	P. O. MS	AF	36.3	87	3.16
			Res TI ₃ , MS ₃		+ 37	-25	+ 2
15 E. D.	37	M	P. O. MS ₁	AF	29.5	95	2.80
					- 11	- 8	-18
16 R. Z.	60	M	AI ₃	AF	34.3	68	2.33
					+ 20	+22	+48
17 J. B.	44	F	Undiagnosed	AF	23.1	111	2.63
					+ 54	-50	-24
18 G. E.	48	M	AI ₄ , MI ₄ , MS ₁	AF	34.5	69	2.38
					- 10	+26	+14
Averages-AF					26.8	102	2.65
%Δ					+ 40	-22	+11
P value					< 0.001	< 0.01	< 0.2
Patients not converted after DC shock—rhythm unchanged							
19 L. S.	53	F	MS ₁	AF	35.9	73	2.62
					- 11	- 4	-14
20 B. R.	49	M	MS ₁ , MI ₂	AF	32.2	95	3.06
					+ 16	- 1	+15
21 A. B.	59	F	P. O. MS ₁	AF	34.8	99	3.45
			Res AI ₂ , MI ₁		+ 5	+ 2	+ 8
22 S. C.	41	F	MS ₂	AF	33.6	81	2.72
					+ 30	-15	+ 9
23 G. A.	50	M	Myocard.	AF	40.9	106	4.34
					+ 20	- 2	+18
Averages-AF					35.5	91	3.24
%Δ					+ 12	- 4	+ 8
P value					< 0.2	< 0.4	< 0.3
24 D. B.	23	M	Undiagnosed	Vent. tach.	20.3	135	2.74
					+ 18	+ 1	+19

SVI, stroke volume index; HR, heart rate; CI, cardiac index; A-V O₂, arterial venous oxygen difference; O₂ cons, oxygen consumption; CO₂ prod, carbon dioxide production; P_{FA}, mean pressures in pulmonary artery; P_{CS}, mean pressures in coronary sinus; P_{FA}, mean pressures in femoral artery; LVWI, left ventricular work index; TP_{ER}, total peripheral resistance; TP_{UR}, total pulmonary resistance, AF, atrial fibrillation.

Diagnosis: MS, mitral stenosis; MI, mitral insufficiency; AS, aortic stenosis; AI, aortic insufficiency; IHSS, idiopathic hypertrophic subaortic stenosis; TI, tricuspid insufficiency; P. O., postoperative; Res, residual after surgery; GDV, Gott-Daggett prosthetic leaflet valve.

* Recording after conversion not technically acceptable.

† Subscript in diagnosis column indicates severity: 1 = mild; 2 = moderate; 3 = moderately severe; 4 = severe.

Patients Undergoing DC Transthoracic Shock

A-V O ₂ %Δ	O ₂ cons. %Δ	CO ₂ prod. %Δ	P _{PA} %Δ	P _{cs} %Δ	P _{FA} %Δ	LVWI %Δ	TPeR %Δ	TPuR %Δ
ml/100 ml blood	ml/min	ml/min	mm Hg	mm Hg	mm Hg	kg M/ min/m ²	cgS	cgS
3.9	168	155	30	11	95	5.6	785	248
+18	+ 5	+ 3	+ 7	0	- 6	-16	+ 4	+18
7.6	179	117	29	9.8	86	2.7	1741	587
-37	-23	-13	-28	-65	- 3	+22	-21	-41
3.2	126	92	11	3.0	66	3.5	767	128
+16	- 6	+ 1	0	-33	- 6	-23	+16	+20
4.2	148	115						
0	+24	+10						
6.6	147	121	40	17	101	3.1	2347	930
- 3	- 3	- 9	-15	-12	- 3	- 3	- 3	-15
5.8	147	119	56	8.0	100	3.4	1797	1006
+12	0	-11	-20	0	-10	-18	0	-10
4.3	152	126	47	9.3	99	4.8	1388	645
-37	+ 2	-10	- 6	-32	- 9	+46	-44	-41
3.3	135	107	33	10.0	69	3.8	820	392
+15	+ 2	+ 2	-15	0	+ 1	- 8	+15	- 4
6.1	137	108	24	6.5	89	2.7	1533	413
-10	+ 1	+11	-17	-26	0	+11	-10	-25
6.4	124	96	50	9.9	72	1.9	1594	1107
-13	+ 2	- 5	-16	-34	- 7	+11	-21	-28
5.9	122	97	25	5.3	94	2.6	2131	567
-25	- 8	-13	- 4	-19	- 1	+23	-19	-22
6.0	147	105	42	6.8	84	2.8	1814	907
- 7	- 7	- 4	-14	-44	- 2	- 4	- 1	-13
3.8	149	115	43	7.5	102	5.4	1468	619
-13	+10	+13	- 9	-23	+ 3	+33	-21	-30
7.1	102	77	25	13.6	77	1.5	2601	844
-11	+32	+29	+ 8	+10	+ 6	+60	-28	-26
5.1	161	128	44	12.2	61	2.6	1098	792
- 8	- 6	-12	- 9	-38	-10	- 8	-12	-11
4.9	137	97	30	5.3	95	3.6	1324	418
+16	- 4	- 7	-10	-43	+ 1	-17	+23	+10
5.3	123	89	18	5.8	84	2.6	1561	334
-19	+20	+12	0	-40	- 1	+50	-33	-32
5.5	144	101	20	6.8	81	2.9	1521	376
+33	+ 1	- 2	+10	+54	-16	-34	+10	+44
6.5	155	112	32	10.0	81	2.6	1315	519
-11	+ 1	+ 2	- 3	0	+ 4	+19	- 9	-15
5.5	139	107	35	9.0	86	3.1	1621	658
- 5	+ 2	- 2	- 9	-15	- 4	+10	-12	-17
< 0.3	< 0.4	< 0.5	< 0.01	< 0.05	< 0.2	< 0.3	< 0.05	< 0.01
4.7	123	102	22	7.3	89	3.2	1534	379
+ 6	- 9	-25	- 5	-11	+ 2	-13	+20	+12
5.1	156	126	34	10.0	102	4.3	1287	429
- 4	+10	+ 6	- 9	-15	+ 1	+14	-12	-21
4.0	138	104	29	7.3	87	4.1	1294	431
- 5	+ 2	+ 6	- 3	-18	-13	- 7	-19	-10
4.6	125	92	25	6.7	77	2.8	1459	474
0	+10	- 3	-20	0	-13	- 4	-20	-27
3.5	152	111	35	9.4	82	4.9	863	326
-11	+ 5	+ 9	-14	- 9	- 2	+14	-26	-26
4.4	139	107	29	8.0	87	3.9	1287	408
- 2	+ 5	- 1	-10	-10	- 5	+ 3	-10	-15
< 0.5	< 0.5	> 0.8	< 0.05	< 0.05	< 0.3	> 0.8	< 0.4	< 0.2
5.3	145	112	26	10.8	79	2.9	1130	372
-13	+ 3	- 1	- 8	- 7	-11	+ 7	-25	-22

given subcutaneously in a dose of approximately 1 mg/kg of body weight. When the narcotic effect of meperidine was clearly present, the first studies of cardiac output and coronary blood flow were made. Conversion of the arrhythmia was then attempted using an Electrodyne model E-100 M defibrillator (Electrodyne Co., Inc., Norwood, Mass.). No general or barbituate anesthesia was employed, nor were any drugs administered between the studies. A single direct current shock of 200 w-sec was delivered through anterior and posterior paddles, respectively, 3 and 5 inches in diameter. In two patients, a second direct current shock of 250 w-sec was applied when the initial shock was unsuccessful. In one patient, 15, this second shock was successful and in the other patient, 19, it was unsuccessful in converting atrial fibrillation to a sinus rhythm. 20–25 min after the shock, the cardiac output and coronary blood flow was determined and pressures recorded again as in the control study. Data from before and after the shock was analyzed statistically by the *t* test only in the patients with atrial fibrillation. Data from patients with other arrhythmias is presented for reader interest and is not used in statistical analysis. Initially, comparisons were made with each subject as his own control. They also were divided into two groups and the data of those subjects whose atrial fibrillation converted was compared with the group in which it persisted. Thus, not only could the effects of

changing rhythm be evaluated but also those of the electric shock.

RESULTS

16 of 21 patients with atrial fibrillation were converted to sinus rhythm. There was a trend for patients whose atrial fibrillation persisted to have a higher cardiac output, lower pulmonary artery pressure, and a slower ventricular rate at rest before the attempted conversion than the patients who converted, but the differences were not statistically significant. However, the sensitivity of the statistical comparison of the two groups was decreased by the small number of subjects whose atrial fibrillation persisted. Two subjects with atrial flutter and one with an unidentified supraventricular tachycardia were converted to a sinus rhythm. In one patient, ventricular tachycardia persisted after a single shock. Two patients with hinged-flap mitral prosthetic valves were among those converted to sinus rhythm. The changes in the cardiac output after conversion of atrial fibrillation to normal sinus rhythm ranged from a +62 to -24%

TABLE II
Left Heart Pressures after DC Transthoracic Shock

Patient No.	Diagnosis	LAM		LVEP		MS	
		before after	%Δ	before after	%Δ	before after	%Δ

Patients converted to sinus rhythm							
1	MS	36	−28*	NR		NR	
		26					
6	MS, MI	40	−28	12.3	− 7	21.0	−16
		31		11.4		17.5	
10	G. D. mitral valve	13	−15	8.0	+15	5.9	−31
		11		9.2		4.1	
12	P. O. MS, MI	16	−13	11.0	−18	8.2	−10
		14		9.0		7.4	
13	P. O. MS, AI	15	+ 3	15.8	− 8	3.0	+73
		15.5		14.5		5.2	
14	P. O. MS, TI	28.5	−16	10.3	+16	23.0	−26
		24		12.0		17.0	
Patients not converted to sinus rhythm							
22	MS	19	−26	8.8	−11	10.1	−36
		14		7.8		6.5	
24	Myocard.	10.2	0	13.6	−10	No grad.	
		10.2		12.2			

LAM, left atrial mean pressure; LVEP, left ventricular end-diastolic pressure; MS, mitral stenosis, mean diastolic gradient; NR, not recorded.

* Pulmonary artery wedge pressure.

FIGURE 2 Patient J. B. Double dye dilution curve— injection of indocyanine green into the left ventricle with simultaneous sampling from the left atrium and left ventricle. Conclusion: no evidence of mitral insufficiency.

with a mean increase of 11% ($P < 0.2$). There was an increase of 8% ($P < 0.3$) in the cardiac output of patients whose atrial fibrillation persisted. The cardiac output of two patients with atrial flutter was at the upper limits of normal and decreased, but remained within normal limits after the conversion to sinus rhythm. The mean oxygen consumption and carbon dioxide production were normal for this laboratory with no significant difference between the two groups. There was an average decrease in the heart rate of 22% ($P < 0.01$) after conversion of the atrial fibrillation. Associated with this was an increase in stroke volume of 40% ($P < 0.001$). Similar changes were not observed in those whose atrial fibrillation persisted.

There was a decrease in the pulmonary artery pressures of 9 ($P < 0.01$) and 15% ($P < 0.05$) in the coronary sinus pressure after conversion of atrial fibrillation to sinus rhythm. There was a

similar decrease of 10% in the pulmonary artery pressure ($P < 0.05$) and of 10% in the coronary sinus pressure ($P < 0.05$) after similar direct current shocks in the group whose rhythm did not change.

Table II summarizes the left-sided hemodynamic alterations after the shock. In the five patients with atrial fibrillation who had left-sided catheterizations and whose rhythm converted, there was an average decrease of 16% ($P < 0.05$) in the left atrial mean pressure. There was also an average decrease in the mean diastolic gradient across the mitral valve of 16% ($P < 0.05$) as demonstrated in Fig. 1. There was no significant change in the left ventricular end-diastolic pressures after conversion of atrial fibrillation to sinus rhythm. All the patients with left-sided catheterizations had mitral valvular disease except patient 24, who had underlying myocarditis with no demonstrable valvular heart disease.

There was a decrease in the calculated peripheral and pulmonary resistances of 12 ($P < 0.05$) and 17% ($P < 0.01$), respectively, after conversion of atrial fibrillation to sinus rhythm with direct current precordial shocks. Percentage-wise, there was also a similar decrease in the peripheral resistance (10%, $P < 0.3$) and in the pulmonary resistance (15%, $P < 0.1$) of patients with persistent atrial fibrillation after the shock. There was no significant change in the right or left ventricular work indices in either group of subjects.

The degree of mitral insufficiency was evaluated before and after the conversion of atrial fibrillation to sinus rhythm in five subjects (Nos. 10, 12, 13, 14, and J.B.).¹ This evaluation was made by injecting indocyanine green into the left ventricle and by sampling simultaneously from the left atrium and femoral artery (24-26). In subject J.B.,¹ there was no mitral insufficiency detected during atrial fibrillation (Fig. 2). In patients 10 and 14 there was slight to moderate mitral insufficiency during atrial fibrillation, which was less after conversion. Two other patients, 12 and 13, with moderately severe and slight mitral insufficiency, re-

spectively, had no apparent change after the conversion from atrial fibrillation to sinus rhythm. Representative dye curves of patient 10 and 12 are presented in Figs. 3 and 4.

The changes in the coronary hemodynamics are presented in Table III. Changes in the coronary blood flow ranged from -31 to $+24\%$ after conversion of atrial fibrillation to sinus rhythm with nearly an identical range in the group that was shocked but did not convert. There was no significant change in the mean coronary blood flow in either group. The variation in coronary blood flow did not correlate with changes in the cardiac rate. There was a slight decrease in the arterial oxygen content and of the coronary sinus carbon dioxide content in all patients shocked. The changes in the coronary sinus oxygen content, myocardial oxygen utilization, and carbon dioxide production were not of statistical significance. There was an increase of 28% ($P < 0.02$) in the index of efficiency after conversion of atrial fibrillation to sinus rhythm. There was no change in this index of efficiency after direct current shock if atrial fibrillation persisted.

In the two patients with atrial flutter and the

¹ Table I, data not used in statistical analysis.

FIGURE 3 Double dye dilution curve— injection of indocyanine green into the left ventricle with simultaneous sampling from the left atrium and femoral artery. Conclusion: a decrease in the degree of mitral insufficiency after electrical conversion of atrial fibrillation.

FIGURE 4 Patient 12. Double dye dilution curve— injection of indocyanine green into the left ventricle and simultaneous sampling from the left atrium and femoral artery. Conclusion: no change in the degree of mitral insufficiency after electrical conversion of atrial fibrillation.

one with supraventricular tachycardia who were converted to sinus rhythm, there was a decrease in the coronary blood flow, an increase in the coronary sinus oxygen content, and a decrease in the coronary sinus carbon dioxide content (Table III). Thus, there was a decrease in the myocardial oxygen consumption and carbon dioxide production and an increase in the left ventricular index of efficiency.

DISCUSSION

An electrical discharge, which can be delivered transthoracically from a capacitor at a predetermined point in the cardiac cycle, provides an efficient and, with proper precautions, a safe method for the termination of many cardiac arrhythmias (27–37). It also affords the opportunity to evaluate the hemodynamic state before and immediately after the conversion of arrhythmias without the administration of drugs or anesthesia between the studies which may, in themselves, pro-

duce hemodynamic changes and complicate interpretation of the results. The cardiovascular effects resulting from the quinidine, digitalis, and meperidine given before this study were assumed to be constant throughout both studies and not to contribute to any of the observed changes (14, 38–42). Thus, the two variables in the present study were the change in the rhythm and the effect of the direct current transthoracic shock. The hemodynamic changes were analyzed statistically for evaluation of these two variables as described under Methods.

The average increase of 11% in the cardiac output after conversion of atrial fibrillation to sinus rhythm while at rest was similar to that reported by other investigators (12–15). There was also an 8% increase in the cardiac output after the shock in the group of subjects who failed to convert from atrial fibrillation. Comparison of these changes between the two groups indicated no significant difference ($P > 0.9$). This suggests that

TABLE III
Coronary Blood Flow and Myocardial Metabolic Effects of DC Transthoracic Shock

Patient No.	CBF %Δ	Index eff %Δ	A O ₂ %Δ	CS O ₂ %Δ	A CO ₂ %Δ	CS CO ₂ %Δ
	<i>ml/100 g per min</i>		<i>ml/100 ml blood</i>	<i>ml/100 ml blood</i>	<i>ml/100 ml blood</i>	<i>ml/100 ml blood</i>
Patients converted—atrial fibrillation						
6	67	0.53	16.3	2.9	41.2	52.9
	+22	+ 40	-13	+52	+ 3	-3
7	88	0.38	15.5	4.1	44.3	53.1
	-31	+ 55	- 5	+12	- 4	-3
8	66	0.29	19.9	6.2	45.8	54.5
	+14	- 3	+ 1	- 8	-11	-7
9	45	0.30	17.8	3.1	50.1	60.1
	+13	+ 3	- 4	+26	0	-1
11	58	0.29	19.1	3.6	50.8	62.9
	-10	+ 24	+ 1	+11	+ 1	-3
12	90	0.51	15.9	4.1	51.3	61.7
	- 6	+ 49	+ 3	- 5	- 3	-3
13	46	0.25	15.7	3.0	47.1	57.4
	- 2	+ 68	- 1	+10	- 3	-4
14	85	0.29	14.5	3.5	41.1	48.3
	-11	0	- 3	0	- 1	+2
15	44	0.55	19.0	3.9	45.7	55.2
	+ 2	- 13	- 2	+ 5	- 3	-1
16	79	0.24	17.7	4.2	54.0	63.9
	+22	+ 33	- 2	+ 5	- 3	-4
Averages	67	0.36	17.1	3.9	47.1	57.0
%Δ	0	+ 28	- 2	+ 8	- 2	-3
P value	> 0.9	< 0.03	< 0.03	< 0.2	< 0.1	<0.002
Patients converted—atrial flutter and vent. tach.						
1	54	0.80	20.2	7.2	48.8	56.8
	-22	+ 36	- 4	+22	-27	-3
2	118	0.15	17.4	3.6	44.7	54.8
	-37	+147	- 1	+39	- 2	-1
3	90	0.40	14.7	4.5	46.1	53.7
	-42	+ 53	- 3	+22	-26	-2
Patients not converted—atrial fibrillation						
19	52	0.48	16.0	3.5	48.6	58.7
	+33	- 31	- 3	- 3	- 1	-3
20	64	0.58	15.7	4.7	49.1	57.9
	+11	+ 3	+ 1	- 6	0	0
21	54	0.73	16.0	5.4	48.1	55.2
	- 4	- 1	- 5	-11	- 5	-3
22	75	0.32	15.1	3.2	49.3	58.3
	-16	+ 22	- 1	+19	- 3	-4
23	64	0.72	15.0	3.9	48.0	55.3
	+ 2	+ 14	- 1	- 5	- 3	-4
Averages	62	0.57	15.6	4.1	48.6	57.1
%Δ	+ 3	0	- 2	- 2	- 2	-3
P value	< 0.8	> 0.8	< 0.2	< 0.7	< 0.03	<0.001
24	64	0.40	16.5	4.9	49.2	57.1
	- 8	+ 13	- 1	+ 4	- 2	-2

CBF, coronary blood flow; Index eff, index of efficiency, left ventricular work/left ventricular oxygen usage; A O₂, arterial oxygen content; CS O₂, coronary sinus oxygen content; A CO₂, arterial carbon dioxide content; CS CO₂, coronary sinus carbon dioxide content.

the increase in cardiac output after the conversion of atrial fibrillation may not be a result of the return of sinus rhythm but of other factors associated with the shock. The increase in the cardiac output in both groups and the observations by Rodman, Pastor, and Figueroa would indicate that the precordial shock or other factors in this study were not depressing the myocardial function and thus suppressing any hemodynamic changes that otherwise may be evident (22).

Studies in which cardiac outputs are determined hours or days after the conversion of atrial fibrillation to sinus rhythm suggests that there is an increase in the cardiac output after conversion, but it does not always appear to be immediate (15-22). Rodman et al. performed serial determinations of the cardiac output after the electrical conversion of atrial fibrillations and found that the hemodynamic improvements were often delayed for hours or even days (22). The exact mechanism of this delayed benefit is not known, but in this study the most significant change which followed conversion of atrial fibrillation to sinus rhythm was the immediate increase in the left ventricular index of efficiency. It is conceivable that this improvement in myocardial function is cumulative and involves the entire myocardium, both atrial and ventricular, and results in a progressively better mechanical systole. This temporal relationship of hemodynamic improvements after conversion of atrial fibrillation may account for some of the observed changes and at least in part explain some of the controversies arising from other studies on the hemodynamic effects of arrhythmias.

The failure for the cardiac output to increase immediately after the conversion of atrial fibrillation to sinus rhythm with precordial shocks has been noted by other investigators (16, 22). This is not unexpected since such factors as peripheral vascular resistance, the metabolic needs, myocardial function, and valvular lesions are the usual determining factors of the cardiac output and not the cardiac rhythm per se, so long as the heart rate remains within the physiological range. However, one factor that must be considered in the relationship of the conversion of atrial fibrillation to changes in the cardiac output is the contribution that atrial contraction gives to diastolic filling.

In this study, an attempt was made to correlate the height and contour of the left atrial "A" waves

after conversion of atrial fibrillation with the changes in the cardiac output and in the left ventricular end-diastolic pressure. There was no consistent change in the left ventricular end-diastolic pressure, and no correlation was noted between the character of the "A" wave and the cardiac output or left ventricular end-diastolic pressure. In general, the "A" waves were diminutive after conversion. Similar results have been reported by others (43).

The loss of left atrial contraction and its contribution to ventricular diastolic filling that occurs with the onset of atrial fibrillation has been reported to result in the elevation of the mean left atrial pressure and a decrease in the left ventricular end-diastolic pressure (44). Both the left atrial mean pressure and the mitral diastolic gradient decreased in four of five subjects after conversion of atrial fibrillation to a sinus rhythm. The one subject, 13, who increased his left atrial pressure, had a near normal pressure. Furthermore, the absolute change was small after conversion and was accompanied by an increase in oxygen consumption and cardiac output.

Experimental work suggests that the left atrial pressure changes noted in this study are caused by the correction of the arrhythmia (44), but it must be pointed out that in the single patient in this study with mitral valve disease and atrial fibrillation who did not convert, the changes in left atrial pressure were similar to most of the patients who were converted.

Experimental studies indicate that normal atrial contractions are required for effective closure of the atrio-ventricular valves (45, 46). In this study, no evidence of significant mitral insufficiency was found in many subjects with atrial fibrillation when evaluated by left ventricular angiography or by indicator dilution curves as shown in Fig. 2. Although there are many sources of error in the quantitation of mitral insufficiency by indicator dilution curves, evaluation by this method suggests that conversion of atrial fibrillation to sinus rhythm decreases the degree of mitral insufficiency in some but not all patients (Fig. 3). Failure to demonstrate mitral insufficiency consistently in all patients with atrial fibrillation also indicates that atrial contraction may not be essential for adequate closure of the atrio-ventricular valves in human

subjects. Similar observations have been made by others (43, 47, 48).

The decrease in the pulmonary artery and coronary sinus pressures did not appear to be related to conversion of the atrial fibrillation, since comparison between the two groups did not indicate any difference ($P > 0.9$). The changes in calculated ventricular work and vascular resistances also were similar in the two groups ($P > 0.6$). Thus, in the present study, performed at rest and in the supine position, the changes in the cardiac output, arteriovenous oxygen difference, and pressures on the right side of the heart were not specifically related to the changes in cardiac rhythm. There were wide variations in the individual changes in the cardiac output after conversion of the arrhythmia which did not appear to be related specifically to the changes in the heart rate as was found by Graettinger, Carleton, and Muenster (16), although the increase in the stroke volume in the patients who converted is the result of decreasing the heart rate without a significant change in cardiac output. The changes in the cardiac output also did not appear to be related to the severity or type of the underlying heart disease as was found by Killip and Baer, but their studies were performed 1 day after conversion and, as pointed out previously, this is at a time when the hemodynamic changes are more marked (21, 22).

No consistent change in the coronary blood flow was noted after transthoracic direct current shock regardless of whether the arrhythmia was or was not converted. Furthermore, the individual variation in flow did not appear to be related to changes in the cardiac rate. However, the changes in rate were less in the present study than in those studies which demonstrate a clear relation between coronary flow and heart rate (49, 50). The failure to find any significant change in the coronary blood flow after conversion of atrial fibrillation to sinus rhythm is compatible with the results of Wegria who demonstrated in the open-chest dog that electrically induced atrial or ventricular tachycardias usually produced no change or a slight increase in the coronary blood flow (51, 52). Corday, Gold, DeVera, Williams, and Fields, however, believed that a decrease in the systemic blood pressure associated with tachycardias resulted in a decrease in coronary blood flow (53).

Although there was no change in the coronary

blood flow in either group, there was an improvement in the left ventricular function of patients who converted from atrial fibrillation to sinus rhythm, which was not noted when the arrhythmia persisted. This improvement is demonstrated by the index of myocardial efficiency (Table III), which relates the amount of external work performed by the left ventricle to its oxygen consumption. This left ventricular efficiency index is particularly useful in evaluating the effects of atrial fibrillation, because it is influenced by the number of myocardial contractions that are ineffectual in propelling blood to the periphery and also by the amount of mitral insufficiency. Both of these factors may be associated with atrial fibrillation and should make the heart a less efficient pump. Another factor in determining myocardial efficiency is the heart rate and stroke volume. An increase in stroke volume occurred in this study with the conversion of atrial fibrillation. The ventricular end-diastolic pressures suggest that there was no increase in the ventricular end-diastolic volumes after conversions (54). If this is true, then in accordance with the law of Laplace, the myocardial wall tensions during the additional stroke output would be significantly less and so the myocardial energy requirements and oxygen consumption would be reduced (55, 56). Therefore, since the cardiac output is unchanged and the heart rate decreased, the index of myocardial efficiency would improve. In this study, the elimination of the ineffectual ventricular contractions, the possible decrease in mitral insufficiency, and the decreased heart rate and increased stroke volume that occurred after conversion of atrial fibrillation all lead to an increase in the myocardial efficiency. What effect the return to sinus rhythm per se has on the myocardial efficiency cannot be evaluated by this study. This influence of atrial fibrillation on the efficiency of the left ventricle may well be a factor in the association of angina pectoris with the onset of atrial fibrillation and for the improvement in the anginal syndrome or relief of congestive heart failure that follows the conversion of atrial fibrillation to sinus rhythm (12, 57, 58).

It should be reemphasized that, in the present study, immediately after electrical conversion of atrial fibrillation, hemodynamic changes occurred which were statistically significant when each subject served as his own control. However, when

the changes were compared between the group of patients who converted from atrial fibrillation and those who did not, there was little difference. The notable differences between the two groups were a decrease in heart rate, an increase in stroke volume, and an increase in the left ventricular index of efficiency in those patients who reverted to sinus rhythm. The conversion of atrial fibrillation to sinus rhythm by a precordial shock had no immediate effect on the cardiac output or coronary blood flow.

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