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Hemodynamic Effects after Reversion from Atrial Fibrillation to Sinus Rhythm by Precordial Shock *

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Atrial fibrillation is the most common persistent arrhythmia in man. Since it was clinically recognized more than 50 years ago (1, 2), there has been continued interest in the effect of atrial fibrillation on cardiac performance. The increased heart rate and tendency to excessive tachycardia during exercise, characteristic of untreated atrial fibrillation, have frequently clouded interpretation of the dynamic effects of change in rhythm. Many studies in man have reported increases in cardiac output after reversion from fibrillation to sinus rhythm (3–5). Some workers have found no significant changes in a number of subjects (4–6). Interpretation of the data, however, has been tempered by the problem of separating the effects of a change in atrial pacemaker from the influences of therapy. Heart rates have frequently varied greatly. Quinidine or another drug has been administered in various dosages. Time lapse between measurements has been different from one study and from one patient to another. The hemodynamic response to atrial fibrillation may vary in different forms of heart disease, yet most reports have furnished scant functional and diagnostic details.

The application of synchronized, direct current, precordial shock for the reversion of arrhythmias provides a unique tool for the evaluation of hemodynamic changes that occur with change in rhythm (7). The technique appears to avoid the disadvantages associated with previous studies. Since reversion occurs at a predictable moment, studies may be arranged with a constant time lapse before and after application of the technique.

We are reporting the effects of reversion from atrial fibrillation to sinus rhythm on cardiac function in 27 patients with heart disease. Six patients who did not revert form a control group. The data show that in patients with valvular heart disease there is a significant increase in cardiac output after conversion to sinus rhythm. In patients with small hearts who do not have valvular heart disease, there is no significant change in cardiac output after sinus rhythm is induced. Data for subjects at rest and during exercise are presented and the implications discussed.

Methods

Twenty-seven patients with chronic atrial fibrillation were hospitalized for at least 3 days before study (Table I). Seventeen patients had rheumatic heart disease. Six patients had no valvular abnormalities and normal to slightly enlarged hearts and are classified as "benign" fibrillators (8). None of these patients had angina or evidence of old or recent myocardial infarction. The four other patients included a single example of atrial septal defect 48 months after operative closure, a myocardiopathy of unknown cause, a calcific aortic stenosis, and a bacteriologically cured endocarditis.

For purposes of analysis the patients were divided into four groups. Group I comprises six patients who remained in atrial fibrillation despite attempted reversion and serves as a control for the techniques of reversion and repeated hemodynamic study. Group II contains ten patients with mitral valve dysfunction, six patients with predominant mitral stenosis and four with predominant mitral regurgitation. Group III consists of five patients with aortic valve deformity, and Group IV includes the patients with benign fibrillation.

All patients were maintained on digitoxin. Neither quinidine nor procaine amide nor any other antiarrhythmic drugs were administered until after the studies had been completed. No sedation was given before the hemodynamic measurements.

After the patients had fasted overnight, hemodynamic studies were performed. Patients were anesthetized locally with procaine hydrochloride; a polyethylene no. 190 catheter was then placed in the left median arm vein by

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TABLE I

Clinical information on patients undergoing DC reversion

Patient	Sex	Age	Diagnosis*	Functional classification	Duration of atrial fibrillation
		years			
Group I: Controls					
1. RB	M	42	RHD, MS, AS	3	1.5 years
2. VB	F	49	RHD, MS, MI	2	> 5 years
3. DF	M	53	RHD, MI	3	5 years
4. JD	F	31	RHD, AS, AI, TS	2	10 years
5. JC	M	45	ASD	2	>4 years
6. JR	M	50	Myocardiopathy	3	3 years
Group II: Mitral valve disease					
7. AW	F	50	RHD, MS	3	4 months
8. WJ	M	45	RHD, MS	3	1 month
9. EP	M	49	RHD, MS	3	2 months
10. EC	F	40	RHD, MS (AS, AI)	4	3 years
11. AB	M	44	RHD, MS	2	3 years
12. PP	M	40	RHD, MS	2	3 years
13. JT	M	44	RHD, MI (AS, AI)	3	6 months
14. PG	M	33	RHD, MI	2	1 month
15. RC	F	42	RHD, MI	3	8 years
16. WD	M	53	RHD, MI	3	2 months
Group III: Aortic valve disease					
17. FC	M	54	RHD, AI	3	1 month
18. JOB	M	44	RHD, AS, MS	3	1 month
19. PH	M	79	ASHD, AS	4	4 months
20. OE	M	64	BE, AI, MI	4	1 month
21. AC	M	44	RHD, AS	4	1 year
Group IV: "Benign" fibrillation					
22. ST	F	67	?ASHD	2	>10 years
23. CM	M	56	?ASHD	2	6 months
24. WT	M	68	?ASHD	2	>8 years
25. CC	F	53	?ASHD	2	1 year
26. OK	M	73	?ASHD	2	1 year
27. DC	M	65	?ASHD	2	1 year

* Abbreviations: RHD, rheumatic heart disease; MS, mitral stenosis; MI, mitral insufficiency; AS, aortic stenosis; AI, aortic insufficiency; TS, tricuspid stenosis; ASD, atrial septal defect; ASHD, arteriosclerotic heart disease; BE, bacterial endocarditis.

the Seldinger technique, and an indwelling needle inserted into the right brachial artery. The distal end of the venous catheter was advanced into the innominate vein. Catheters of identical length were used for repeat studies in the same patient. Arterial pressure was measured with a Statham transducer. Venous pressure was measured with a saline-filled, calibrated glass tube. Expired air was collected through an open circuit system; oxygen and carbon dioxide content was determined by the method of Scholander (9). PR intervals were measured from the electrocardiogram recorded during the hemodynamic study by averaging ten consecutive intervals. Base-line instability precluded measurements in several patients during exercise.

Cardiac output was measured by the indicator-dilution technique. After injection of approximately 5 mg of indocyanine green from a volumetrically calibrated adapter, arterial blood was withdrawn at a constant rate of 38 ml per minute through a Waters 300 A densitometer. Indicator-dilution curves were inscribed on a Texas Instrument recorder. Calibration of the densitometer was accomplished by passing arterial blood containing known concentrations of green dye through the densitometer at the rate used for arterial sampling. Indicator-dilution measurements that did not include a calibration curve containing four points on a straight line were discarded. Cardiac output and "central" volume were calculated according to the method of Kinsman, Moore, and Hamilton after the curves had been replotted on semilogarithmic paper (10).

Heart rate, cardiac output, and stroke volume were obtained in duplicate on resting patients. In 33 paired determinations in atrial fibrillation the average per cent variations, calculated regardless of sign, from the mean of the pair ± standard deviation were as follows: heart rate, 1.93 ± 1.94; cardiac output, 4.14 ± 3.70; and stroke volume, 4.32 ± 3.92. In 21 paired determinations in sinus rhythm the average per cent variations from the mean of the pair ± standard deviation were as follows: heart rate,

TABLE II

Hemodynamic data obtained in 6 control patients who did not revert and 21 patients who did revert from atrial fibrillation to sinus rhythm after precordial shock

Patient	BSA	Rhythm*	Oxygen consumption		Cardiac index		Heart rate		Stroke volume		Peripheral resistance		Mean arterial pressure	
			Rest	Exercise	Rest	Exercise	Rest	Exercise	Rest	Exercise	Rest	Exercise	Rest	Exercise
	m^2		$ml/min/m^2$		$L/min/m^2$		*beats/min*		*ml*		*dyne-sec-cm*$^{-5}$		*mm Hg*	
Group I: Controls														
RB	2.01	AF	125	232	1.84	2.05	56	96	66	43	1,700	2,200	85	126
		AF	149	358	1.83	2.62	65	126	57	42	1,800	1,600	90	118
VB	1.58	AF	139	249	1.62	2.98	62	84	42	56	2,900	2,200	97	145
		AF	114	232	1.72	2.43	58	69	47	56	2,800	2,400	100	120
DF	1.92	AF	102	338	1.37	2.51	74	150	34	32	3,200	2,200	107	141
		AF	136	396	1.62	3.10	83	156	38	38	2,500	1,900	98	100
JD	1.58	AF	93	206	1.80	2.16	66	111	43	31	2,200	2,400	83	111
		AF	97	238	2.16	2.81	65	102	53	37	1,900	1,800	84	111
JC	1.84	AF	109	344	1.74	3.04	54	93	59	60	2,600	1,900	106	140
		AF	133	385	1.92	3.22	57	84	62	71	2,400	1,700	108	128
JR	1.79	AF	133	310	1.66	2.44	76	102	39	43	2,600	2,200	103	129
		AF	104	294	1.51	2.28	87	108	31	39	2,700	2,500	105	135
Group II: Mitral valve disease														
a) Mitral stenosis														
AW	1.51	AF	112	163	1.47	1.78	42	54	53	50	2,500	2,900	71	102
		SR	123	203	2.24	2.69	50	78	68	52	1,600	2,000	69	104
WJ	1.73	AF	121	234	1.84	2.10	79	108	40	34	2,100	2,100	83	100
		SR	111	255	2.80	3.43	81	100	60	59	1,300	1,300	82	99
EP	1.68	AF	142	259	2.19	3.06	82	124	45	42	1,800	1,500	84	101
		SR	129	300	2.77	3.04	82	111	57	46	1,300	1,600	78	109
EC	1.51	AF	137	242	1.65	1.95	62	122	40	24	2,900	3,000	97	122
		SR	109		2.23	2.21	102	122	33	27	2,100	2,400	89	106
AB	1.80	AF	132	271	2.25	2.96	52	74	78	72	1,700	1,700	92	126
		SR	126	296	2.55	3.23	59	74	79	79	1,300	1,500	87	123
PP	1.84	AF	116	303	2.10	3.40	51	90	76	70	1,800	1,400	87	117
		SR	107	248	2.65	3.97	62	84	79	87	1,500	1,100	86	113
b) Mitral insufficiency														
JT	1.87	AF	204	289	2.24	3.73	66	108	64	65	1,500	1,200	80	107
		SR	168	318	3.16	4.16	72	96	82	81	1,100	1,100	82	110
PG	1.70	AF	109	306	1.66	2.66	46	88	61	51	1,300	1,600	61	105
		SR	113	368	2.17	3.42	69	104	54	56	1,600	1,300	80	108
RC	1.58	AF	102	289	2.20	2.91	56	99	63	46	2,200	1,900	100	115
		SR	111	252	2.81	3.90	59	82	75	75	1,800	1,400	101	114
WD	1.89	AF	144	370	2.25	2.80	81	99	53	54	1,600	1,600	79	105
		SR	139	372	2.93	3.31	89	105	62	69	1,200	1,100	86	111
Group III: Aortic valve disease														
FC	1.75	AF	139	272	1.44	1.95	55	81	47	42	2,200	1,900	70	93
		SR	135	231	2.11	2.15	60	82	62	46	1,500	1,800	69	92

* AF, atrial fibrillation; SR, sinus rhythm.

TABLE II—(*Continued*)

Patient	BSA	Rhythm*	Oxygen consumption		Cardiac index		Heart rate		Stroke volume		Peripheral resistance		Mean arterial pressure	
			Rest	Exercise	Rest	Exercise	Rest	Exercise	Rest	Exercise	Rest	Exercise	Rest	Exercise
	m^2		*ml/min/m²*		*L/min/m²*		*beats/min*		*ml*		*dyne-sec-cm⁻⁵*		*mm Hg*	
JOB	1.77	AF	146	353	2.64	3.12	76	108	61	51	1,500	1,400	92	113
		SR	158	285	3.42	3.76	68	93	90	72	1,100	1,100	92	112
PH	1.83	AF	137	160	1.78	1.86	82	90	40	38	2,700	2,600	115	117
		SR	139	159	2.15	2.06	76	78	52	48	2,100	2,500	106	122
OE	1.57	AF	120		1.17		62		30		3,200		78	
		SR	129		1.96		70		44		2,000		80	
AC	2.08	AF	123	279	1.49	1.88	78	108	40	36	1,900	1,800	82	102
		SR	150	228	1.24	1.45	81	117	32	26	2,500	1,600	87	121
Group IV: "Benign" fibrillation														
ST	1.49	AF	100	204	2.07	2.72	61	90	51	45	2,300	2,200	90	116
		SR	93	220	2.04	2.66	54	76	56	52	1,900	1,900	78	106
CM	2.14	AF	131	407	2.40	3.44	64	81	80	91	1,200	1,000	84	107
		SR	182		2.51	2.66	61	69	88	83	1,100	1,300	85	99
WT	2.11	AF	86	336	1.86	2.95	47	93	85	67	1,600	1,600	79	120
		SR	93	280	2.47	2.64	49	75	106	74	1,200	1,400	84	108
CC	1.90	AF	130	420	2.70	3.23	69	107	72	56	1,500	1,700	97	142
		SR	125	493	2.36	3.30	79	90	55	68	1,800	1,600	103	132
OK	2.01	AF	104	396	2.17	3.33	90	150	49	45	1,900	1,600	109	145
		SR	120	341	2.41	3.07	69	96	70	64	1,400	1,500	92	130
DC	2.14	AF	130	270	1.23	1.62	87	102	30	34	3,100	2,700	110	132
		SR	128	282	1.45	1.78	95	105	33	36	2,900	2,800	118	142
Mean values														
Group I	Mean	AF	117	280	1.67	2.53	65	106	47	44	2,500	2,200	97	132
	±SE		7.4	23.9	0.07	0.17	3.7	9.5	5.1	4.9	200	100	4.3	5.2
	Mean	AF	122	317	1.79	2.74	69	108	48	47	2,400	2,000	98	119
	±SE		8.3	29.7	0.09	0.15	4.4	12.6	4.8	5.5	170	50	3.8	5.0
Group II	Mean	AF	132	273	1.99	2.73	62	97	57	51	1,900	1,900	83	110
	±SE		9.5	17.2	0.10	0.02	4.8	6.8	4.3	4.9	160	200	3.7	2.9
	Mean	SR	124	290	2.63†	3.34†	72‡	96	65‡	63§	1,500§	1,500§	84	110
	±SE		5.9	17.8	0.03	0.02	5.1	5.0	4.8	5.9	100	130	2.6	2.0
Group III¶	Mean	AF	136	262	1.76	2.31	69	93	44	44	2,400	2,000	89	108
	±SE		5.5	55.9	0.28	0.41	6.2	7.9	6.6	3.8	360	350	9.8	7.4
	Mean	SR	140	225	2.41§	2.66	69	84	62‖	55	1,700‖	1,800	87	109
	±SE		6.2	36.4	0.34	0.55	3.3	4.5	10.1	8.3	230	400	8.0	8.8
Group IV	Mean	AF	114	339	2.07	2.88	70	104	62	56	1,900	1,800	95	127
	±SE		12.5	36.4	0.21	0.27	6.7	9.9	8.6	8.3	280	240	5.2	6.2
	Mean	SR	124	323	2.21	2.69	68	85	68	63	1,700	1,800	93	120
	±SE		13.3	42.5	0.16	0.21	7.0	5.7	10.6	6.8	270	230	6.0	7.3

Significance of difference between means in AF and SR: † $p < 0.001$; ‡ $p < 0.05$; § $p < 0.005$; ‖ $p < 0.025$.
¶ Patient AC omitted from totals. See text.

1.73 ± 4.19; cardiac output, 2.70 ± 2.77; and stroke volume, 3.28 ± 3.50.

Measurements were made with subjects at rest and during exercise. The exercise was a standardized bicycling motion of the legs at a rate of 60 to 70 per minute performed while the patient was supine. The rate and the range of leg excursion were kept constant. Venous and arterial pressure, heart rate, oxygen consumption, and cardiac output were obtained between the third and fifth minutes of exercise. The arterial pressure and electrocardiogram were monitored for 10 minutes after exercise, and a final indicator-dilution curve was obtained at that time.

On the second day 24 hours after the control study, reversion to sinus rhythm was attempted with synchronized DC precordial shock on patients under light thiopental anesthesia, according to techniques previously described (11).

On the third day, 24 hours after attempted reversion and 48 hours after the first evaluation, the hemodynamic studies were repeated exactly as on the first day.

Results

Hemodynamic data obtained in 27 patients at rest and during exercise before and after attempted DC reversion to sinus rhythm are presented in Table II.

Rest

Group I. Control. The average age of the six patients in this group was 45 years, and the average duration of atrial fibrillation was more than 5 years (Table I). There were no significant changes in any hemodynamic variables measured before and after attempted reversion. Cardiac output averaged 7% greater during the second study (Figure 1).

Group II. Mitral stenosis and mitral insufficiency. Since the data for the patients with dominant mitral stenosis or insufficiency showed similar trends, they are analyzed as a single group of ten patients. The average age for the group was 44 years, and atrial fibrillation had been present from 1 month to 8 years (Table I).

Heart rate was greater after reversion in all but one patient. The mean heart rate in atrial fibrillation was 62 beats per minute and increased to 72 beats per minute in sinus rhythm ($p < 0.025$). Mean oxygen consumption was not significantly different in the two studies, averaging 132 ml per

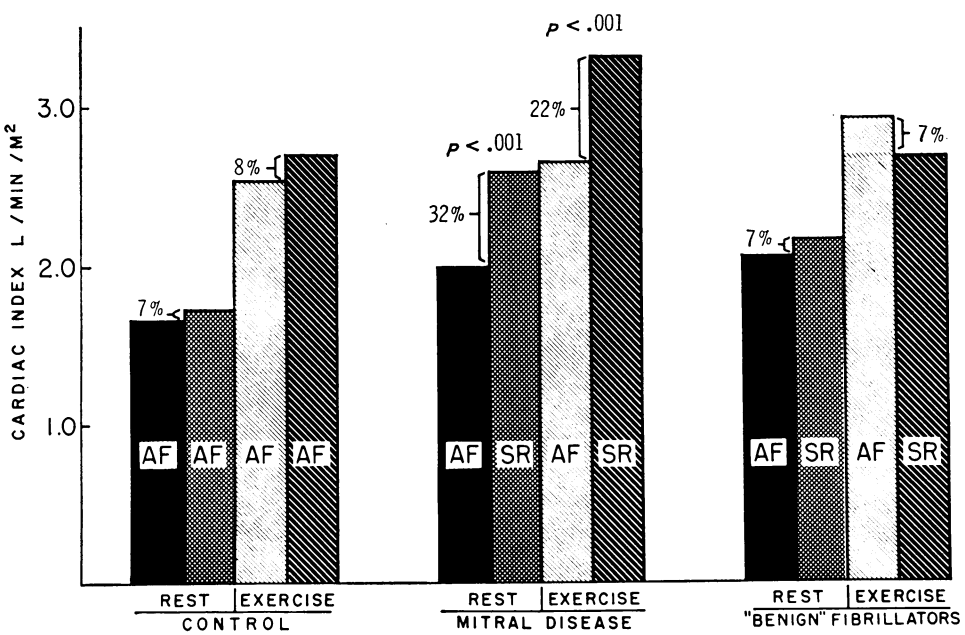

FIG. 1. EFFECT OF PRECORDIAL SHOCK AND REVERSION FROM ATRIAL FIBRILLATION (AF) TO SINUS RHYTHM (SR) ON CARDIAC INDEX AT REST AND DURING EXERCISE. Control patients (left) who did not revert had no significant changes in cardiac index at rest and during exercise after precordial shock. In patients with mitral valve disease (middle) cardiac index increased 32% ($p < 0.001$) at rest and 22% ($p < 0.001$) during exercise after reversion. In "benign" fibrillators (right) cardiac index did not change significantly after reversion.

minute per m² before and 124 ml per minute per m² after reversion.

The resting cardiac index was greater in all patients after reversion. The mean cardiac index during atrial fibrillation for the group was 1.99 L per minute per m² and increased to 2.63 L per minute per m² in sinus rhythm ($p < 0.001$). The increase ranged from 13% to 52% and averaged 32% (Figure 1). The average increase in patients with dominant mitral stenosis was 32%; in those with dominant mitral insufficiency it was 33%.

Mean stroke volume increased from 57 ml per beat to 65 ml per beat ($p < 0.05$) after reversion despite the concomitant increase in heart rate. Arterial pressure remained remarkably constant. Calculated peripheral vascular resistance varied inversely with the cardiac output, falling in each case in sinus rhythm ($p < 0.005$) as cardiac output rose.

Because of the modest increase in heart rate after reversion to sinus rhythm, it is difficult to separate the effect of increased heart rate and the change in rhythm for the group as a whole. Consequently, the data from five patients (WJ, EP, JT, RC, and WD) whose heart rate changed less than 10% after reversion have been analyzed separately (Figure 2). Neither heart rate, which averaged 73 beats per minute in atrial fibrillation and 77 beats per minute in sinus rhythm, nor oxygen consumption, which averaged 143 ml per minute per m² in atrial fibrillation and 132 ml per minute per m² in sinus rhythm, varied significantly. Resting cardiac index increased from 2.14 L per minute per m² before, to 2.89 L per minute per m² after reversion (+ 35%, $p < 0.001$). Stroke volume also increased significantly from 53 to 67 ml per beat (+ 26%, $p < 0.005$). Arterial pressure did not change significantly. Peripheral vascular resistance was lower in sinus rhythm than in atrial fibrillation ($p < 0.005$).

It is clear from this analysis that in the patients with mitral valve disease, reversion from atrial fibrillation to sinus rhythm significantly increased cardiac output as a result of a rise in stroke volume. Since arterial pressure remained remarkably constant, calculated peripheral vascular resistance varied inversely with blood flow.

Group III. Aortic valve disease. One patient, AC, was clinically and hemodynamically worse after DC shock. He complained of dyspnea and fatigue and manifested signs of increased pulmonary congestion for several days after reversion. Similar reactions suggesting that transthoracic electric shock may adversely influence cardiac performance temporarily in an occasional patient have been reported by others (12). His cardiac output and stroke volume were lower in sinus rhythm than in atrial fibrillation both at rest and during exercise. Because of the severe clinical deterioration, the circumstances of the pre- and postreversion study were not considered comparable, and the data for this patient were omitted from the statistical evaluation of group III in Table II.

In the four other patients in group III mean oxygen consumption and heart rate were similar in the two studies. Cardiac index increased from an average of 1.76 L per minute per m² in atrial fibrillation to 2.41 L per minute per m² in sinus

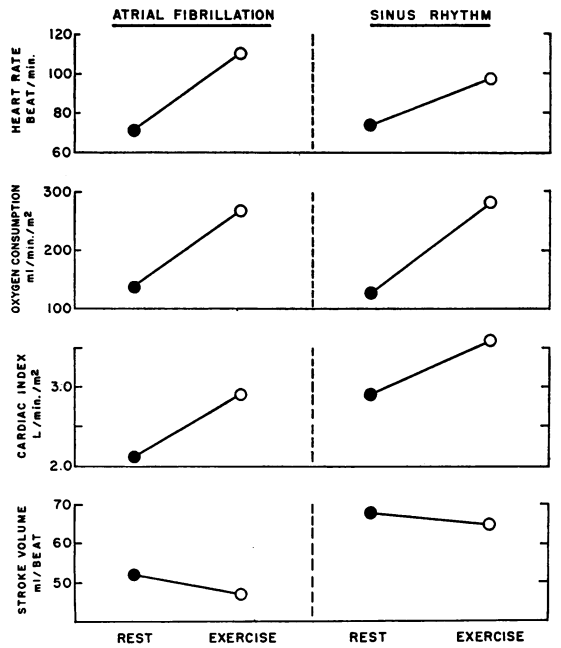

FIG. 2. CARDIODYNAMICS BEFORE AND AFTER REVERSION IN FIVE PATIENTS WITH MITRAL VALVE DISEASE WHOSE RESTING HEART RATES VARIED LESS THAN 10%. See text for details. Note similarity of oxygen consumption in two rhythms. Heart rate was slightly lower in sinus rhythm than in atrial fibrillation during exercise. Average cardiac index increased 35% at rest ($p < 0.001$) and 26% during exercise ($p < 0.05$) after reversion. Stroke volume also increased significantly in sinus rhythm at rest ($p < 0.005$) and during exercise ($p < 0.02$).

rhythm (37%, $p < 0.005$). Mean stroke volume increased from 44 to 62 ml per beat ($p < 0.025$). Mean arterial pressure was essentially unchanged, and the calculated peripheral vascular resistance fell ($p < 0.025$).

We concluded that in patients with symptomatic aortic valve abnormalities, reversion from atrial fibrillation to sinus rhythm is associated with significant increase in cardiac output and stroke volume.

Group IV. Benign atrial fibrillation. The average age in this group was 64 years. The duration of fibrillation varied from 6 months to more than 10 years (and in one patient was possibly as long as 23 years) (Table I).

Average values for oxygen consumption, heart rate, cardiac index, and stroke volume were not significantly different in the two studies. In two patients an increase in cardiac index greater than encountered in group I was observed. In WT cardiac output increased 33% and stroke volume 25%, whereas heart rate and oxygen consumption did not change. In patient DC oxygen consumption was unchanged, but heart rate increased from 87 to 95 beats per minute, and cardiac ouput increased 18%, whereas stroke volume increased 10%. Mean cardiac index for the group was 2.07 L per minute in atrial fibrillation and 2.21 L per minute in sinus rhythm (+ 7%, Figure 1).

Arteriovenous oxygen differences. Calculated arteriovenous blood oxygen content differences (a-v O_2) in resting patients are depicted in Figure 3. These data are derived from a single resting oxygen consumption and the mean of two resting cardiac outputs. Mean a-v O_2 before and after attempted reversion were not significantly different in the control group and in that with benign fibrillation. In the patients with mitral valve disease, a-v O_2 averaged 6.71 ml per 100 ml during fibrillation, but decreased to 4.72 ml per 100 ml after reversion ($p < 0.001$). In the four patients with aortic valve disease in whom the data are tabulated, a-v O_2 fell from 8.29 ml per 100 ml before to 6.02 ml per 100 ml after reversion ($p < 0.05$).

Exercise

Measurements during exercise were obtained in all but one of the patients studied at rest. Mean oxygen consumptions were comparable in each of the four groups studied before and after attempted reversion. Heart rates were also similar before and after reversion in groups I, II, and III. In group IV, benign fibrillation, heart rate during

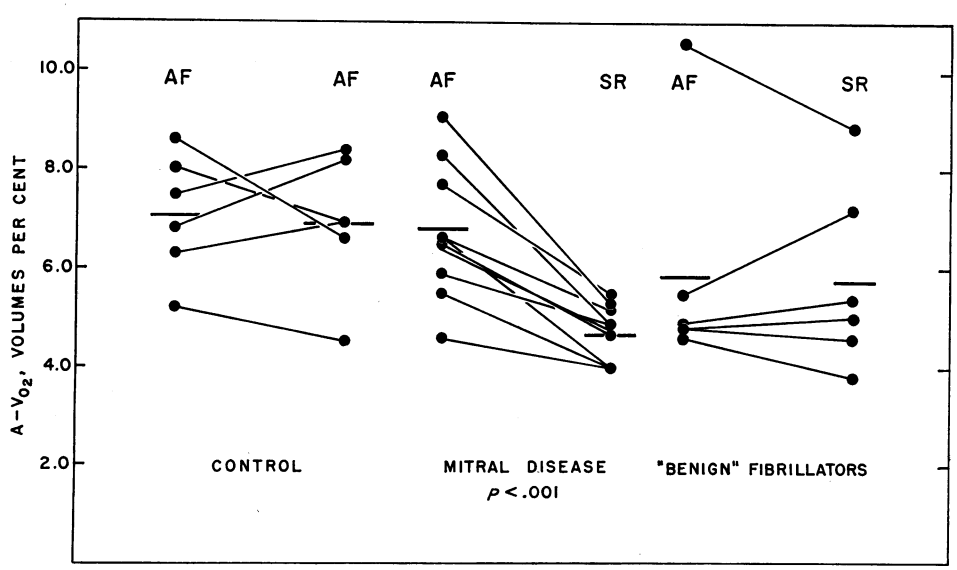

FIG. 3. CALCULATED ARTERIOVENOUS OXYGEN DIFFERENCE BEFORE AND AFTER PRECORDIAL SHOCK. In the control group that remained in fibrillation (left) there was no significant change. a-v O_2 fell significantly after reversion ($p < 0.001$) in the group with mitral valve disease (middle). a-v O_2 was unchanged in benign fibrillators (right) despite reversion.

exercise fell from 104 beats per minute in atrial fibrillation to 85 beats per minute in sinus rhythm (Figure 4).

The increment in heart rate induced by exercise was compared in groups I, II, and IV before and after reversion (Figure 5). The changes were similar in the control group before and after attempted reversion, but were lower in the patients with mitral valve disease ($p < 0.05$) and benign fibrillation ($p < 0.025$) in sinus rhythm. A reduction in the increment in heart rate during exercise, despite a mean oxygen consumption and cardiac output similar to those achieved in atrial fibrillation, was the only measurement that was significantly different in the patients with benign fibrillation after reversion.

Average cardiac output during exercise did not change significantly after precordial shock in groups I, III, and IV (Figure 1). In group II, mean cardiac index increased from 2.74 L per minute per m^2 in atrial fibrillation to 3.34 L per minute per m^2 in sinus rhythm (+ 22%, $p < 0.001$). In the six patients with dominant mitral stenosis the average increase in cardiac output was 22%. In the four patients with mitral regurgitation the average increase in cardiac output was 23%.

Mean a-v O_2 was not significantly different in the two studies in groups I, III, and IV. Mean a-v O_2 fell during exercise in group II from 10.16 before to 8.48 ml per 100 ml blood after reversion ($p < 0.025$).

The increment in cardiac output for each 100-ml increment in oxygen consumption did not change significantly in the patients with mitral disease, averaging 532 ml per 100 ml before and 512 ml per 100 ml after reversion. Hence the increased cardiac output during exercise in group II was not related to change in cardiac function induced by reversion, but rather to a higher resting level upon which the exercise response was added. Although the cardiac output was higher in sinus rhythm than in atrial fibrillation, the changes induced by the exercise were similar in the two rhythms.

Stroke volume during exercise was larger in group II after reversion, increasing from 51 to 63 ml per beat ($p < 0.005$). In the other groups there were no significant differences in stroke volume between the two studies.

Calculated peripheral resistance varied inversely with flow during exercise since arterial pressure

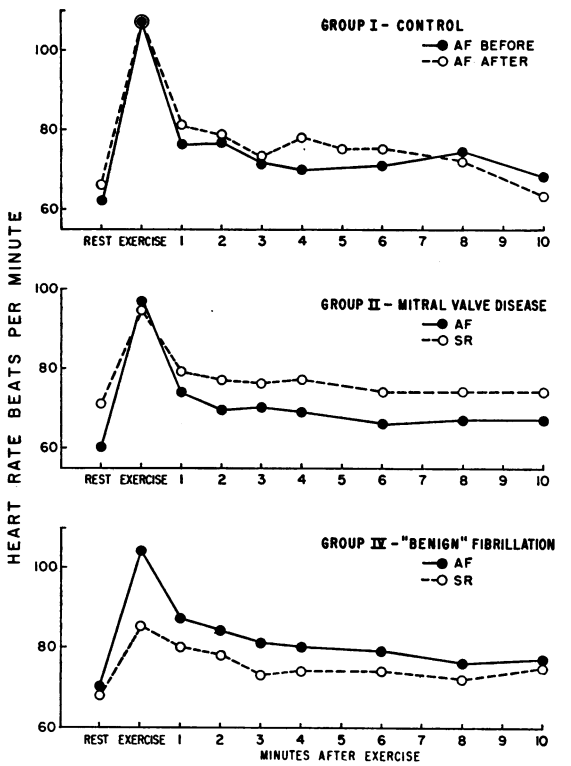

FIG. 4. EFFECT OF EXERCISE ON HEART RATE BEFORE AND AFTER REVERSION. Exercise response was unaffected by attempted reversion in control group (top). In patients with mitral valve disease (middle) exercise heart rates were similar in two rhythms, but pre- and postexercise rates were slightly higher in sinus rhythm. Exercise and postexercise heart rates were lower after reversion in benign fibrillation (bottom).

remained relatively constant. Significant changes were observed only in group II, in which peripheral resistance averaged 1,900 dyne-sec-cm^{-5} in atrial fibrillation and 1,500 dyne-sec-cm^{-5} in sinus rhythm ($p < 0.005$).

Recovery from exercise. Systolic, diastolic, and mean arterial pressures returned to pre-exercise levels by 4 minutes after exercise in all groups in both rhythms. The average cardiac index 10 minutes after cessation of the exercise was not significantly different from the pre-exercise resting value in any group before and after DC shock.

Heart rates during and after exercise were similar in the two studies in group I. In groups II and III, average heart rates during exercise remained slightly higher even at 10 minutes. In group IV, benign fibrillation, the heart rate during

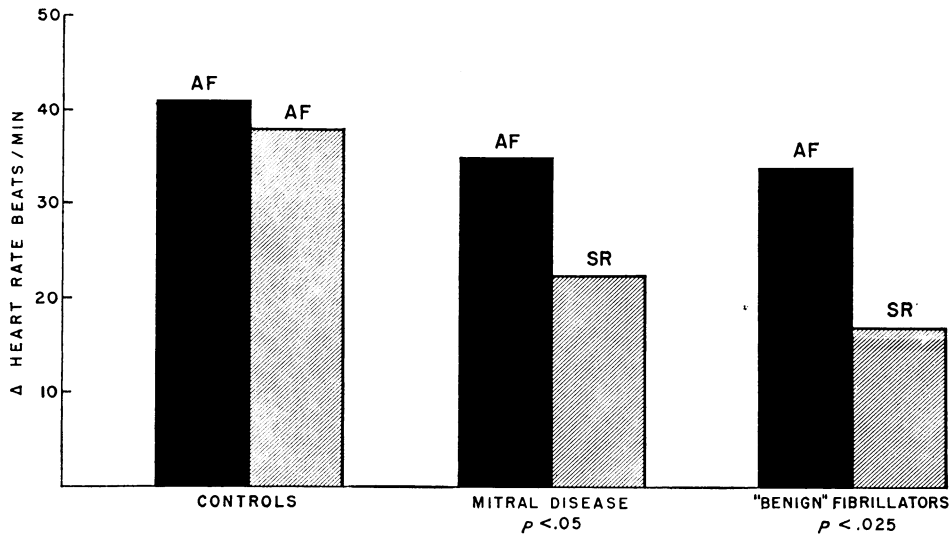

FIG. 5. CHANGE IN HEART RATE DURING EXERCISE AFTER PRECORDIAL SHOCK. Increment in heart rate with exercise was unchanged in controls who did not revert (left), but was significantly lower in patients with mitral valve disease (middle) and benign fibrillation (right) after reversion to sinus rhythm.

exercise was lower after reversion and remained so during the postexercise period (Figure 4).

Other measurements. PR intervals were prolonged at rest in 14 of the 21 patients reverted, averaging 0.26 second in group II, 0.23 second in group III, and 0.21 second in group IV. The PR interval shortened during exercise in all patients in whom reliable measurements were obtained.

Several other variables were analyzed before and after reversion, but are not reported in detail since no significant differences were demonstrated. Factors measured included venous pressure, "central" volume, left ventricular ejection time, and tension-time index. In five patients with mitral stenosis, Q-first sound and aortic second sound-opening snap intervals were not significantly different in the two studies.

Discussion

Since the introduction of medical techniques for the reversion of atrial fibrillation, numerous studies have attempted to determine the effect of the change in rhythm on cardiac function in man and the experimental animal (3–6, 13, 14). In man primary interest has focused on the influence of rhythm on cardiac output (15). Most workers have utilized quinidine for reversion and have continued the drug during the post-treatment study. Few authors have reported or attempted to control oxygen consumption or heart rate. In seven studies in which quinidine was administered and adequate details were recorded, increases in cardiac output averaging 31% were observed in 29 of 43 patients (3). Most of the patients had rheumatic heart disease. Two studies compared carefully matched but separate groups of patients in sinus rhythm and atrial fibrillation and found that cardiac output averaged 18 and 20% higher in sinus rhythm (16, 17).

Despite the reports already available, the hemodynamic consequences upon reversion from atrial fibrillation to sinus rhythm remain controversial (3, 6). Of critical importance in determining the effects of reversion is the design of the study. Unless such variables as the time lapse between studies, metabolic state, heart rate, and concomitant drug therapy are kept constant, it is difficult to be sure that any change or lack of change after reversion is a reflection of rhythm alone. Adequate control of drug administration is especially important when quinidine is utilized, since the drug has myocardial depressant and peripheral vasodilator actions (18, 19). The former might mask improvement after reversion, and the latter

might enhance ventricular function under certain circumstances.

The present study was designed to control as many variables as possible. No antiarrhythmic drugs were administered. Digitalis was maintained at constant dosage, and resting ventricular rates during atrial fibrillation were well controlled. Precordial shock was utilized for conversion and was applied with barbiturate analgesia 24 hours after the first study and 24 hours before the second study. The effects of the reversion and the influences of repeated studies on the hemodynamic functions measured were analyzed in a control group of patients who remained in atrial fibrillation despite precordial shock.

We conclude from the present data that reversion to sinus rhythm from atrial fibrillation enhances stroke volume and therefore cardiac output at rest in patients with mitral or aortic valve disease. Although the outputs are higher during exercise after reversion, the increment induced by exercise remains relatively fixed in the two rhythms.

Our data may be compared with the results reported by Graettinger, Carleton, and Muenster (6) and Morris and co-workers (3) in which DC shock was also used for reversion. Graettinger studied 16 patients of whom 10 had rheumatic heart disease. Premedication consisted of barbiturates and meperidine. The postconversion data were obtained 1 and 2 hours after patients had awakened from thiopental anesthesia. No change in oxygen consumption occurred, but heart rate decreased significantly, falling more than 15 beats per minute in 6 patients at rest. Small increases in cardiac output and decreases in a-v O_2 were observed, but since similar changes occurred in 4 patients who did not revert and also in a control group of 14 patients with chronic heart disease subjected to repeated studies, the authors concluded that reversion had little effect on cardiac function unless the ventricular rate fell sharply. Neither the type of heart disease, the rhythm, nor the sedation, if any, was described for the 14 "control" patients.

Morris studied 12 patients just before and 2 hours after DC shock (3). Six had rheumatic heart disease with mitral valve abnormalities. No medications other than the anesthesia for the shock were employed. An increase in cardiac output, predominantly a reflection of stroke volume, occurred in 7 of 11 patients at rest and in all 5 patients exercised.

The design of the present study is such that it is unlikely that the techniques of cardiac reversion or anesthesia significantly influenced the results. In the study of Graettinger and associates, however, because of the short time interval between study and therapy and the rapid ventricular rates in many of the patients, factors other than the change in rhythm may have played a significant role, including sedation, precordial shock, changes in heart rate, and anesthesia. Thiopental may influence cardiac function, and the effect may last several hours (20). The effects of DC shock on myocardial function in man are insufficiently understood. Ventricular arrhythmias, atrioventricular dissociation, and transient ST segment elevations may be due in part to direct effects of the shock, although digitalis has been implicated. Although serum glutamic oxalacetic transaminase does not increase significantly after DC shock (11), the possibility that some degree of myocardial injury occurs is not ruled out.

Another consideration is the effect of sustained ventricular arrhythmia on cardiac function (15). Recovery from ventricular or supraventricular tachycardias may not be immediate after a precordial shock. Conceivably, long-established atrial fibrillation may adversely affect atrial function with slow recovery after reversion. Atrial waves of small amplitude have been observed immediately after electric reversion (6, 21). They may reflect intrinsic myocardial abnormality or the effect of anesthesia and shock or be a functional consequence of a long-standing arrhythmia. Serial observations on atrial function after DC shock would be of considerable interest.

The atrial pulse pressure and presumably the force of atrial contraction may be greater than normal in patients with left ventricular and mitral valve disease (22, 23). It is probable that the contribution of atrial contraction to the subsequent stroke output will depend to a considerable extent upon the impedance to ventricular filling. If there is little diastolic resistance, inflow will occur predominantly in the first portion of diastole, and atrial contraction may be expected to add but little. If the ventricle or the mitral valve offers significant resistance to ventricular filling

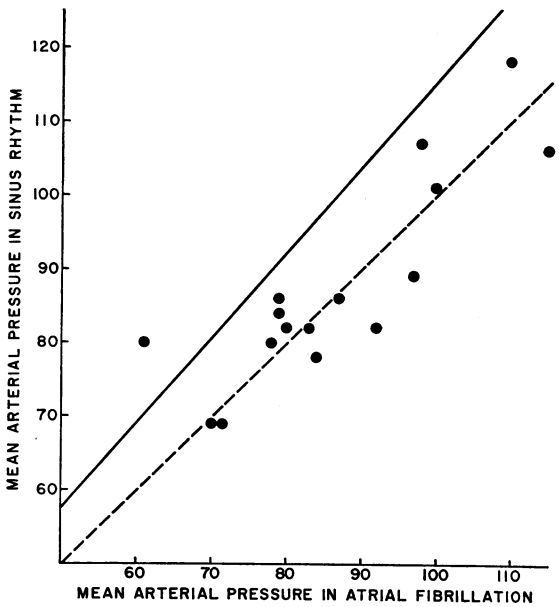

FIG. 6. MEAN ARTERIAL PRESSURE IN ATRIAL FIBRILLATION AND SINUS RHYTHM IN 16 PATIENTS WITH A GREATER THAN 15% INCREASE IN CARDIAC OUTPUT AFTER REVERSION. Dashed line depicts line of identity. Solid line indicates arterial pressure 15% greater in sinus rhythm. Note cluster of values around line of identity indicating maintenance of constant mean arterial pressure despite increase in blood flow.

in early diastole, the contribution of a powerful atrial contraction may be of great importance in increasing end-diastolic volume and fiber length. When atrial contraction is absent or in improper temporal sequence, mean left atrial pressure rises, and the force of ventricular contraction declines (22, 24).

It is apparent, therefore, that the influence of sinus rhythm on cardiac output after reversion from atrial fibrillation at constant heart rate and metabolic demand may not be the same in all forms of heart disease. Moreover, in situations characterized by increased resistance to ventricular filling, a sinus mechanism may not be especially advantageous if the atrium is functionally unable to generate a contraction of sufficient force. In seriously ill patients under these circumstances a sinus tachycardia largely uninfluenced by digitalis may develop consequent to the reversion. We have had this experience in several patients, and it has been reported by others (12).

In the present study the patients with benign fibrillation, selected by virtue of their small hearts, lack of murmurs, and absence of heart failure, failed to show hemodynamic improvement after conversion aside from a reduction in the increase in heart rate induced by exercise. Patients with lone or benign fibrillation have a good prognosis (8), but systematic observations on the effects of reversion have not been previously available. Whether the failure to improve cardiac output reflects an intrinsically weak atrium, or is a manifestation of such low resistance to ventricular filling that atrial contraction adds but little to ventricular performance, cannot be determined at present. That these patients had myocardial abnormality is attested by their low resting cardiac outputs and poor response to exercise (Table II).

Comparison of arterial pressures measured at rest with similar heart rates during atrial fibrillation and sinus rhythm reveals a remarkable constancy of mean, systolic, and diastolic pressures despite marked increases in stroke volume and cardiac output. In Figure 6 are plotted the arterial mean pressures from 16 patients in whom cardiac output increased 15% or more after reversion. Mean arterial pressure remained essentially constant despite the increased blood flow. It is generally accepted that baroreceptor function is predominantly responsible for reflex control of blood pressure (25–27). Most of the information relevant to baroreceptor function and the reflex control of blood pressure has been obtained from anesthetized animals (25). The present study has provided an unusual opportunity to observe the effects of significant changes in cardiac output on arterial pressure under basal conditions and comparable heart rates in unanesthetized humans. Under these circumstances the constancy of arterial pressure is truly impressive and attests to the precision of the control mechanism.

Excessive tachycardia during exercise and recovery from exercise is characteristic of untreated fibrillation (28), and palpitations may be a troublesome problem. Digitalis reduces the heart rate achieved during exercise and permits more rapid return to resting levels (29). If sufficiently large doses are administered, these effects cannot be overcome by atropine (29). Heart rates during exercise in the present study were similar in the patients with valvular abnormalities before and after reversion. Resting and postexercise heart rates were increased after shock, however, indi-

cating that the high degree of atrioventricular block produced by doses of digitalis sufficient to control the exercise tachycardia with fibrillation was largely overcome when the normal sinus mechanism was restored.

In the patients with benign fibrillation, resting heart rates were similar after reversion, but the exercise and postexercise rates were lower in sinus rhythm. The increment in heart rate induced by exercise was significantly reduced by reversion. An explanation for the difference in the heart rate response in groups II and III compared with group IV is not readily apparent. Patients in group IV were older and less symptomatic than those in other groups. It has been suggested that cardiac acceleration during exercise is largely due to release of vagal influence and that sympathetic stimulation participates only in time of great need (30). Slower heart rates during stress in the benign fibrillators after reversion may be a manifestation of decline in pacemaker function with age. The possibility that age, the type and severity of heart disease, and the size of the heart influence the balance of reflex factors controlling heart rate must also be considered.

It may be concluded that reversion from atrial fibrillation in adequately digitalized patients with valvular heart disease will probably have little effect on the heart rate achieved during moderate exertion. Resting heart rates may be higher in sinus rhythm because of the high degree of a-v block required to achieve satisfactory control of the exercise heart rate during atrial fibrillation. In patients with benign fibrillation, the heart rate during and after moderate exercise may be significantly lower after reversion.

The possibility that reversion will improve cardiac function in selected patients with atrial fibrillation is only one of several factors that should be taken into consideration in evaluating the choice of an elective change in rhythm. Other factors include the likelihood that reversion will persist, the incidence of complications as a result of the reversion technique, the tolerance of the patient to the necessary long-term administration of suppressive drugs, and the occasional untoward event that may occur during spontaneous relapse from sinus rhythm. Each consideration must be given due weight in the management of the individual patient.

Summary

Direct current precordial shock for the reversion of atrial fibrillation to sinus rhythm was applied to 27 patients with heart disease. Cardiodynamic studies were performed 24 hours before and 24 hours after precordial shock. Six patients who failed to revert constituted a control group and showed no significant changes in cardiac function after precordial shock.

In ten patients with mitral valve disease, mean cardiac index increased from 1.99 L per minute per m^2 in atrial fibrillation to 2.63 L per minute per m^2 in sinus rhythm ($p < 0.001 + 32\%$). Resting mean oxygen consumptions were similar, but heart rate was slightly higher in sinus rhythm. Resting stroke volume increased from 57 ml per beat to 65 ml per beat ($p < 0.05$). Peripheral vascular resistance varied inversely with cardiac output. Arterial pressure remained remarkably constant.

In four patients with dominant aortic stenosis or insufficiency, mean oxygen consumption and heart rate were similar in the two rhythms. Cardiac index increased after reversion from 1.76 L per minute per m^2 before to 2.41 L per minute per m^2 after reversion ($p < 0.005$, $+ 37\%$). Stroke volume was greater ($p < 0.025$), and peripheral vascular resistance fell as blood flow increased ($p < 0.025$) in sinus rhythm.

Six patients with chronic atrial fibrillation, an average age of 64 years, small hearts, no murmurs, and no evidence of heart failure or ischemic heart disease were termed "benign" fibrillators. Average values for oxygen consumption, heart rate, cardiac index, and stroke volume for this group were not significantly different before or after reversion.

Calculated arteriovenous blood oxygen differences (a-v O_2) were unchanged before and after precordial shock in the control group and after reversion to sinus rhythm in the patients with benign fibrillation. a-v O_2 fell from 6.71 ml per 100 ml during fibrillation to 4.72 ml per 100 ml in sinus rhythm ($p < 0.001$) in the patients with mitral valve disease. In the group with aortic stenosis and insufficiency a-v O_2 fell from 8.29 ml per 100 ml to 6.02 ml per 100 ml in sinus rhythm ($p < 0.05$).

Data obtained during exercise were essentially

similar in the control patients before and after precordial shock. The increment in heart rate during exercise was decreased after reversion in patients with mitral stenosis ($p < 0.05$) and benign fibrillation ($p < 0.025$). In the group with mitral valve disease mean cardiac index during exercise increased from 2.74 L per minute per m^2 in atrial fibrillation to 3.34 L per minute per m^2 in sinus rhythm ($p < 0.001$, + 27%), whereas mean a-v O_2 fell from 10.16 to 8.48 ml per 100 ml. The increased cardiac output during exercise reflected the higher resting level, since the changes induced by the exercise were similar in the two rhythms.

It may be concluded that reversion from atrial fibrillation to sinus rhythm with precordial shock in the absence of myocardial depressants or changes in drug therapy produces a significant improvement in cardiac output due to an increased stroke volume in patients with valvular heart disease. In patients with benign fibrillation reversion from atrial fibrillation is not associated with improvements in cardiac output or stroke volume. Exercise heart rates are similar before and after reversion in valvular heart disease, but lower in benign fibrillation. After reversion, the increment in heart rate induced by exercise is significantly reduced in patients with valvular heart disease and benign fibrillation.

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