

Hæmodynamic Studies Before and After Electrical Conversion of Atrial Fibrillation and Flutter to Sinus Rhythm

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The clinical use of direct current shock in the management of cardiac arrhythmias (Lown, Amarasingham, and Neuman, 1962) has been amply confirmed by many other workers (O'Brien, Resnekov, and McDonald, 1964; Oram *et al.*, 1964; Morris *et al.*, 1964b). The length of time that sinus rhythm persists after the electrical conversion of chronic arrhythmias is often disappointingly short, even when quinidine is prescribed (Resnekov, 1965), and complications following direct current shock are by no means infrequent (McDonald and Resnekov, 1964; Resnekov and McDonald, 1964, 1965). It seemed important therefore to determine what hæmodynamic benefit might be expected following the electrical conversion of atrial fibrillation and flutter to sinus rhythm.

SUBJECTS AND METHODS

Fifteen patients were studied (Table I). Atrial fibrillation was present in 12 and atrial flutter in 3 patients. Of the patients with atrial fibrillation, the underlying heart disease was rheumatic mitral or aortic valvar disease in 3, and cardiomyopathy in 2. Chronic ischæmic heart disease was present in 2 and treated thyrotoxicosis in one. In 4 patients, atrial fibrillation was unassociated with clinical evidence of underlying heart disease, and these patients have been labelled as "lone" (Evans and Swann, 1954). No underlying heart disease could be detected in the 3 patients with atrial flutter and these patients were also labelled as "lone". The age range of the 15 patients varied from 31 to 65 years (mean 49.3). There were 12 men and 3 women. The duration of the arrhythmias before conversion, which ranged from 3 weeks to 12 years, and the radiographic size of the heart are given in Table I.

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Digitalis, when used, was stopped for at least 24 hours before the study. None of the 15 patients was taking quinidine before or after the electrical conversion of the arrhythmia to sinus rhythm. Hæmodynamic studies before and after electrical conversion were made in all the patients according to methods previously described (Resnekov, 1965). In summary, Portex tubing, size FG3, 130 cm. long (external diameter 1.02 mm., internal diameter 0.80 mm.) was passed percutaneously into an arm vein through a no. 18-gauge thin-walled Yale needle, and advanced slowly to the pulmonary artery. Pressure measurements were taken to confirm that the tubing lay in the pulmonary artery. Teflon tubing, TF10 (external diameter 1.0 mm., internal diameter 0.60 mm.), was passed by the technique described by Seldinger (1953) over a nylon guide wire introduced into the brachial artery on the same side as the venous catheter, through a 19-gauge thin-walled Riley needle and advanced for about 30 cm. The patient was asked to sit on a bicycle ergometer (Elema Corporation) with the pulmonary and brachial arterial catheters in position, and pressure recordings were made with reference to the sternal angle using two strain gauges and a multi-channel pen recorder. The electrocardiogram (lead CR5) was recorded simultaneously with the pressures from the brachial and pulmonary arteries. Once a steady state had been achieved, expired air was collected for 4 minutes in a Douglas bag, and simultaneous blood samples were obtained from the pulmonary and brachial arteries during the last two minutes. Exercise was then started and performed for six minutes at each of three work loads, or four work loads if the condition of the patient allowed. The approximate physical activity and oxygen uptake at the work loads used is shown in Table II. Pressure measurements and the electrocardiogram were recorded each 30 seconds; during the last two minutes of each work load expired air was collected simultaneously with blood samples from the pulmonary and brachial arteries. Expired air was analysed by a micro-method for oxygen and carbon dioxide content (Scholander, 1947) and the oxygen content of mixed

TABLE I
CLINICAL DETAILS, RADIOGRAPHIC SIZE OF HEART, AND DC ENERGY SETTINGS REQUIRED FOR THE ESTABLISHMENT OF SINUS RHYTHM IN 15 PATIENTS

Patient No.	Age and sex	Underlying heart disease	Arrhythmia		Radiographic size		DC energy setting for version (joules†)
			Type	Duration (yr.)	Cardio-thoracic ratio (%)	Left atrium* N to +3	
1	62 M	Ischæmia	Atrial fibrillation	4 mth.	53	+1	200
2	49 M	Aortic valve disease post-op.	" "	1	49	+1	300
3	65 M	Ischæmia	" "	1 mth.	50	+1	100
4	51 F	Mitral + aortic valve disease	" "	3 mth.	56	+2	100
5	50 M	Post thyrotoxicosis	" "	6	50	N	300
6	54 M	Alc. cardiomyopathy	" "	1	50	N	300
7	55 M	Lone	" "	1	47	N	250
8	36 M	Cardiomyopathy	" "	1	52	+1	50
9	34 F	Mitral stenosis post-operatively	" "	12	55	+2	200
10	60 M	Lone	" "	3 mth.	50	+1	100
11	42 M	Lone	" "	9 mth.	48	N	300
12	44 M	Lone	" "	5	48	N	300
13	60 M	Lone	Atrial flutter	3 mth.	48	N	200
14	31 M	Lone	" "	3 wk.	42	N	50
15	47 F	Lone	" "	1	42	N	50

N = Normal.
* Size of left atrium graded from a grid film at 40 in. (Resnekov, 1965).
† Delivered using antero-posterior paddles.

venous and arterial blood was determined by the manometric method of Van Slyke and Neill (1924); the cardiac output was calculated (Fick, 1872) and the stroke volume derived. The physical work capacity at a heart rate of 170 beats/min. (PWC₁₇₀) was determined by extrapolation of the linear relationship between heart rate and work load.

The day following the hæmodynamic studies phased direct current shock was used to restore sinus rhythm, the energy setting required being shown in Table I. The hæmodynamic study both at rest and on effort was repeated in an identical fashion 24 or 48 hours after the establishment of sinus rhythm. Stroke volume and heart rate were plotted against the work load before and after the electrical conversion to compare the values during the arrhythmia and in sinus rhythm at equivalent heart rates.

RESULTS

The mean and standard deviations of the results obtained in the 15 patients studied are shown in Table III. A paired "t" test was obtained for the results during the arrhythmia and in sinus rhythm, at rest and at each work load, and p values are referred to in the text and in Fig. 1-5.

Heart Rate. The heart rate was related in a linear fashion to the work load both during the arrhythmia and in sinus rhythm. The average heart rate at rest during the arrhythmia was 91.4 a minute and varied from 50 to 133 a minute (SD 24.8). Following the establishment of sinus rhythm the average heart rate at rest was 79.9 a minute and varied from 63 to 105 a minute (SD 14.2); the difference is statistically significant (p < 0.001). The average heart rate on maximal effort (600

kpm*/minute) during the arrhythmia in the patients studied was 170.0 a minute and varied from 130 to 200 a minute (SD 22.7). Once sinus rhythm had been established the heart rate at 600 kpm/minute averaged 139.7 a minute and varied from 110 to 171 a minute (SD 21.2), and the difference is highly significant (p < 0.001). The difference between the heart rates during the arrhythmia and in sinus rhythm was exaggerated as the work load increased (Fig. 1).

Physical Work Capacity. The PWC₁₇₀ averaged 624.2 kpm during the arrhythmia and varied from 200 to 1170 kpm (SD 212.6). In contrast, once sinus rhythm had been established the average PWC₁₇₀ was 858.9 kpm and varied from 590 to 1050 kpm (SD 153.0), p < 0.001.

Arteriovenous Oxygen Difference. The arteriovenous oxygen difference at rest during the arrhythmia averaged 70.2 ml./l. and ranged from 56.2 to 97.5 ml./l. (SD 10.8). Following the establishment of sinus rhythm the average value of the arterio-

TABLE II
WORK LOAD SETTINGS, EQUIVALENT PHYSICAL ACTIVITY, AND APPROXIMATE EXPECTED OXYGEN CONSUMPTION

Work load (kpm/min.)	Equivalent activity	Oxygen uptake (ml./min.)
200	Walking at moderate pace	900
400	Walking briskly	1350
600	Running moderately	1800
800	Running hard	2200

100 kpm = 16 watts.
kpm/min. = kilopond-metre per minute.

TABLE III
MEAN HAEMODYNAMIC RESULTS IN 15 PATIENTS BEFORE AND AFTER ELECTRICAL CONVERSION
OF ATRIAL FIBRILLATION OR FLUTTER TO SINUS RHYTHM

Exercise (kpm/min.)	Before conversion						After conversion					
	Heart rate/min.	Oxygen uptake* (ml./min.)	A-V oxygen diff. (ml.)	Cardiac output (l./min.)	Stroke volume (ml.)	PWC ₁₇₀ † (kpm)	Heart rate/min.	Oxygen uptake* (ml./min.)	A-V oxygen diff. (ml.)	Cardiac output (l./min.)	Stroke volume (ml.)	PWC ₁₇₀ † (kpm)
Control	91.4 (24.8)	333.0 (33.9)	70.2 (10.8)	5.2 (0.7)	60.7 (18.1)		79.9 (14.2)	356.0 (28.5)	68.7 (4.3)	5.3 (0.8)	67.1 (11.1)	858.9 (153.0)
200	124.0 (21.8)	757.7 (86.8)	102.8 (9.6)	7.4 (1.0)	63.7 (12.4)	624.2 (212.6)	100.2 (13.7)	762.5 (31.5)	98.3 (10.1)	8.3 (2.1)	82.2 (13.7)	
400	147.0 (21.1)	1151.9 (123.7)	118.4 (14.5)	9.8 (1.5)	68.1 (13.5)		120.0 (11.3)	1177.0 (136.7)	109.3 (7.1)	11.4 (1.9)	94.9 (18.4)	
600	170.0 (22.7)	1542.7 (179.7)	129.6 (17.3)	12.1 (2.2)	71.0 (16.4)		139.7 (21.2)	1572.0 (204.0)	113.6 (17.5)	14.2 (2.7)	106.7 (24.4)	

* Expressed as STPD.

† PWC₁₇₀ = Physical work capacity at heart rate of 170/min. (See text.) Standard deviations are in parentheses.

venous oxygen difference at rest was reduced to 68.7 ml./l. (range 55.0 to 96.5, SD 4.3), but the difference is not statistically significant, $p < 0.05$. The mean arteriovenous oxygen difference at 200 kpm/minute was 102.8 ml./l. (range 88.5 to 116.2, SD 9.6) during the arrhythmia and 98.3 ml./l. (range 66.6 to 110.8, SD 10.1) in sinus rhythm, and is statistically significant ($p < 0.001$). At 400 kpm/minute, the corresponding figures were 118.4 ml./l. (range 98.8 to 140.8, SD 14.5) during the arrhythmia and 109.3 ml./l. (range 72.7 to 123.2, SD 7.1) in sinus rhythm ($p < 0.001$). At 600 kpm the mean value was 129.6 ml./l. (range 104.2 to 170.1, SD 17.3) during the arrhythmia and 113.6 ml./l. (range 74.5 to 138.5, SD 17.5) in sinus rhythm ($p < 0.001$)

(Fig. 2). The reduction in the arteriovenous oxygen difference was due to the higher oxygen content of mixed venous blood at rest and on effort in sinus rhythm, and desaturation of arterial blood did not occur during exercise in any of the patients studied.

Oxygen Uptake. The investigations were performed under strictly comparable conditions during the arrhythmia and in sinus rhythm, and great care was taken to ensure that all the expired air was collected in the Douglas bag. The difference in the volume of oxygen uptake at rest and on effort measured during the arrhythmia and in sinus rhythm was not statistically significant at rest or on effort (Fig. 3).

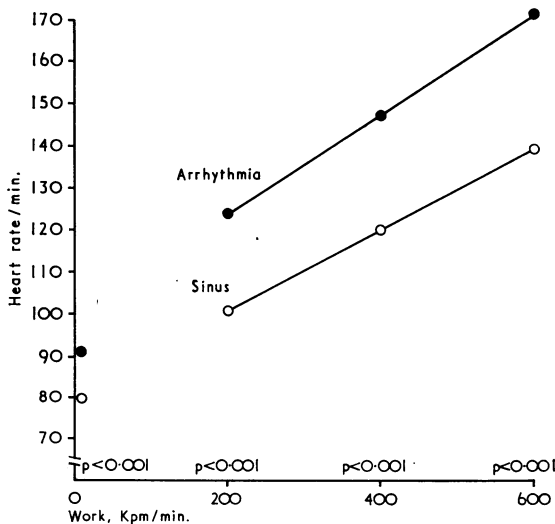


FIG. 1.—Mean of the heart rates of 15 patients at rest and exercising at increasing work loads during arrhythmia and in sinus rhythm.

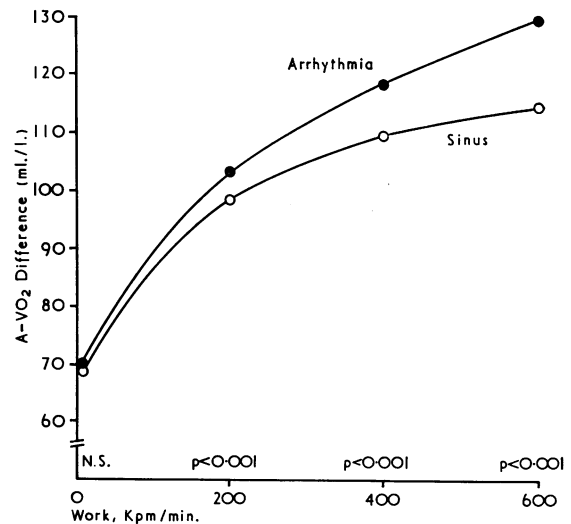


FIG. 2.—Mean arteriovenous oxygen difference in 15 patients at rest and on exercise plotted against work load. N.S. = difference not significant.

Cardiac Output. The mean cardiac output at rest during the arrhythmia was 5.2 l./min. (range 3.3 to 6.3, SD 0.73) and following the establishment of sinus rhythm 5.3 l./min. (range 3.3 to 7.7, SD 0.89). The difference is not statistically significant ($t=0.29$, $p<0.05$). A significant difference occurred, however, with the stress of exercise, and was accentuated as the work load increased. The mean cardiac output measured at 200 kpm/min. was 7.4 l./min. (range 4.5 to 8.7, SD 1.01) during the arrhythmia and 8.3 l./min. (range 4.7 to 10.7, SD 2.05) in sinus rhythm, a difference of 0.9 l./min., $p<0.001$. Comparable values at 400 kpm/min., however, were 12.1 l./min. (range 8.0 to 15.2, SD 2.2) during the arrhythmia and 14.2 l./min. (range 9.1 to 17.3, SD 2.7), a difference of 2.1 l./min. ($p<0.001$). These changes are shown graphically in Fig. 4.

Stroke Volume. The stroke volume averaged 60.7 ml. (range 37 to 104, SD 18.1) at rest during the arrhythmia and was significantly higher during sinus rhythm, being 68.7 ml. (range 48 to 79, SD 11.1, $p<0.01$). This difference increased with the stress of exercise. The stroke volume at 200 kpm/min. during the arrhythmia was 63.7 ml. (range 44 to 87, SD 12.4) and 82.2 ml. (range 55 to 107, SD 13.7) in sinus rhythm, a difference of 18.5 ml. ($p<0.001$): comparable figures at 600 kpm/min. were 71.0 ml. (range 54 to 104, SD 16.4) during the arrhythmia, and 106.7 ml. (range 66 to 139, SD

24.4) in sinus rhythm, a difference of 35.7 ml. ($p<0.001$) (see Fig. 5). The mean of the stroke volumes at increasing heart rates is shown in Fig. 6. During the arrhythmia there is little change in stroke volume with increasing heart rates, but in sinus rhythm the stroke volume rises up to a heart rate of 130-140 per minute whereafter it remains constant.

Pressure Measurements. There was no consistent change between the mean of the pulmonary and brachial arterial pressures at rest and on exercise during the arrhythmia and in sinus rhythm measured with reference to the sternal angle. Typical pressure tracings are shown in Fig. 7. Systolic pulmonary arterial pressure rose from normal values to 40 mm. Hg or more, on the highest exercise loads in four patients. Two patients developed marked systolic and diastolic systemic hypertension during exercise but neither had evidence of hypertension in the past.

DISCUSSION

These results show that there is a significant increase in the cardiac output in sinus rhythm as compared with measurements made during atrial fibrillation or flutter. The difference was not always present at rest, but could invariably be provoked by exercise, and was accentuated as the exercise level was increased. The findings differ from those of Graettinger, Carleton, and Muenster (1964) on supine exercise but are in agreement as regards the

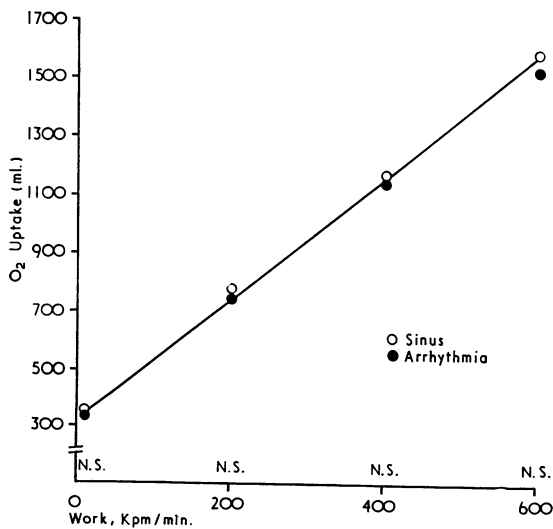


FIG. 3.—Mean oxygen uptake in 15 patients at rest and on exercise plotted against work load. N.S.=difference not significant

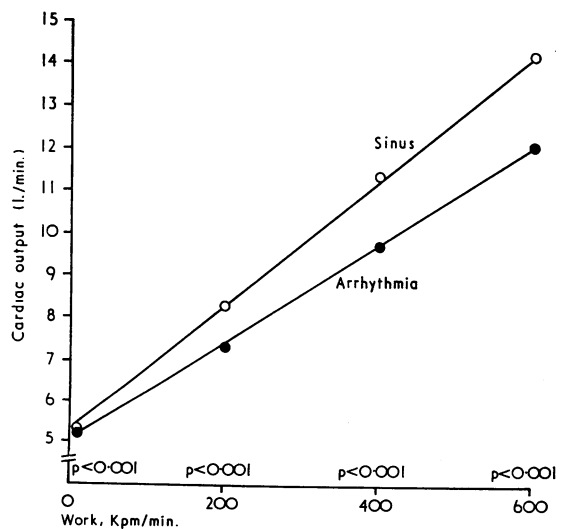


FIG. 4.—Mean cardiac output in 15 patients at rest and on exercise plotted against work load.

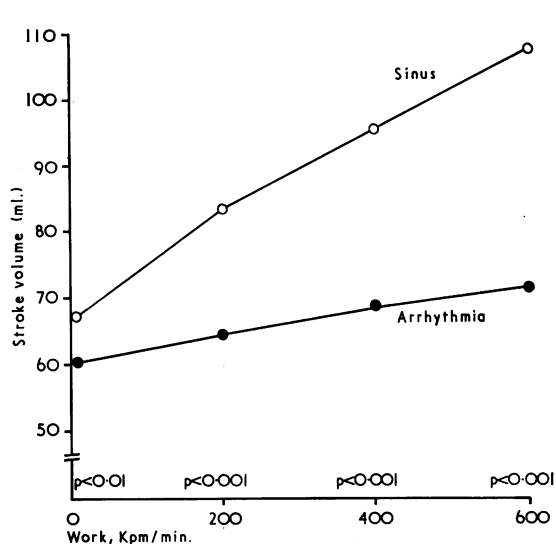


FIG. 5.—Mean stroke volume in 15 patients at rest and on exercise plotted against work load.

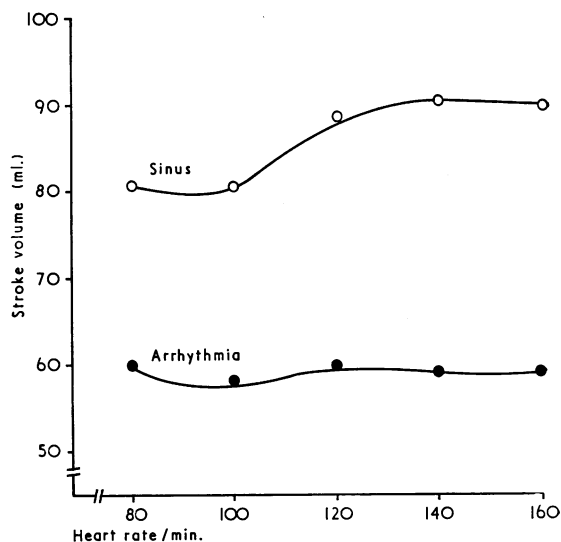


FIG. 6.—Mean stroke volume in 15 patients at rest and on exercise plotted against heart rate.

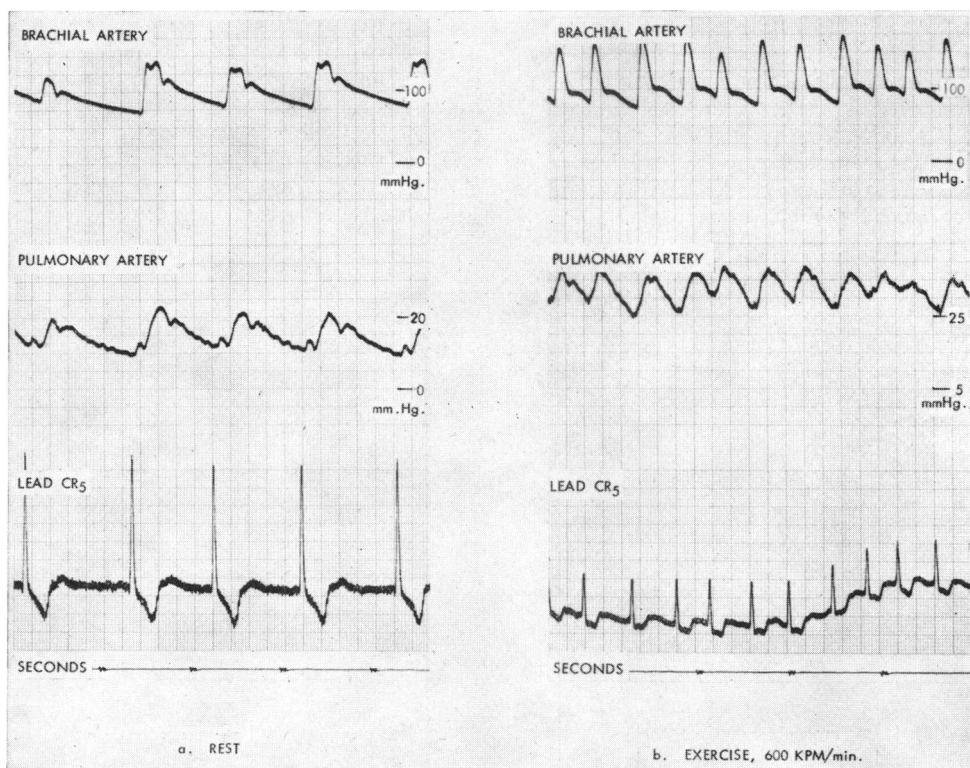


FIG. 7.—Pressures recorded simultaneously from the pulmonary and brachial arteries and lead CR₅ during atrial fibrillation.

(a) At rest, sitting on the bicycle.

(b) During the sixth minute of exercise at 600 kpm/min.

Note: the sensitivity of the cardiogram has been reduced during exercise to lessen the effect of respiratory swing.

cardiac output at rest in atrial fibrillation and sinus rhythm. The levels of oxygen consumption on effort, however, are considerably less in the investigations done by these authors and rarely exceeded 300 ml./min./m.² body surface area, whereas oxygen consumption during the maximum exercise load in the present series was usually three times higher. It may well be, therefore, that more severe exercise in their investigation would have demonstrated an increased cardiac output in sinus rhythm.

The series reported by Oram *et al.* (1963) was unusual in that a mean increase of 70 per cent in the cardiac output measured at rest was found in patients studied in sinus rhythm up to 16 days after electrical conversion. The preconversion levels of the cardiac output were excessively low in some of these patients, however. Large increases in cardiac output following the establishment of sinus rhythm are also reported by Halmos and Patterson (1965) and by Kahn *et al.* (1964). Benchimol, Lowe, and Akre (1965) who studied eight patients before and after the conversion of atrial fibrillation to sinus rhythm also demonstrated that the improvement in cardiac function when in sinus rhythm was more evident during exercise. Morris *et al.* (1964a) showed an average increase in cardiac output at rest of 34 per cent in 7 of 11 patients converted to sinus rhythm, while all five patients studied on exercise had an average increase in cardiac output of 17 per cent.

Three patients with atrial fibrillation had no detectable underlying heart disease responsible for the arrhythmia. All three patients showed hæmodynamic improvement in sinus rhythm (Table IV). Indeed, in one patient an extra work load of 200 kpm/min. to a total of 800 kpm/min. was possible in sinus rhythm, and was achieved with a heart rate in sinus rhythm similar to the heart rate at 600 kpm/min. during the arrhythmia. Similar beneficial results in sinus rhythm were found when the cardiac output and therefore the stroke volume measurements were compared. In contrast, Baer,

Weglarz, and Killip (1964) and Killip and Baer (1966) found no improvement after sinus rhythm had been achieved in the lone fibrillation group.

There was a most satisfactory improvement in the circulation following the establishment of sinus rhythm in patient No. 2 whose aortic valve had been replaced six weeks before study. In atrial fibrillation, the increase in cardiac output on exercise was entirely rate dependent, and the stroke volume fell progressively as the exercise load increased (Fig. 8); once sinus rhythm had been achieved, however, a more normal response to exercise occurred, and the stroke volume was maintained even at a heart rate of 150 beats a minute.

The occurrence of hæmodynamic benefit in sinus rhythm on exercise, was not dependent on the nature of the underlying heart disease in the 15 patients, in this series, which is in agreement with the findings of McIntosh, Kong, and Morris (1964) and of McIntosh and Morris (1966) who reviewed the subject. Efficient atrial systole, however, is particularly important in conditions associated with valvar or subvalvar obstruction of the left ventricle and in diseases causing a diminished compliance of that chamber (Braunwald and Frahm, 1961). The patients who had a virtually normal heart once sinus rhythm had been achieved benefited more than those with underlying heart disease.

The increase in the cardiac output in sinus rhythm resulted in a decrease in the arteriovenous oxygen difference since oxygen consumption during the arrhythmia and in sinus rhythm was not significantly altered, as was also found by Morris *et al.* (1963) and Graettinger *et al.* (1964).

Body posture has a profound effect on the circulation both at rest and on exercise (Bevegård, Holmgren, and Jonsson, 1960). Cardiac output and stroke volume are both less in the sitting position than in the supine. These changes occur at rest (McMichael and Sharpey-Schafer, 1944; Donald *et al.*, 1953) and are probably due to a redistribution

TABLE IV
HEART RATE AND CARDIAC OUTPUT IN 3 PATIENTS WITH LONE ATRIAL FIBRILLATION BEFORE AND AFTER CONVERSION TO SINUS RHYTHM AT REST AND ON EFFORT

Patient No., age, sex		Before conversion							In sinus rhythm						
		Rest	Exercise (kpm/min.)				PWC ₁₇₀ (kpm)	Rest	Exercise (kpm/min.)				PWC ₁₇₀ (kpm)		
			200	400	600	800			200	400	600	800			
7 55 M	{Heart rate/min. {Cardiac output (l./min.)	80 5.2	100 8.7	122 11.9	145 15.2	—	810	68 5.3	100 9.4	118 13.7	134 17.8	—	1020		
11 42 M	{Heart rate/min. {Cardiac output (l./min.)	95 4.9	115 7.8	132 10.9	150 14.0	—	820	90 4.6	102 8.6	116 12.8	132 16.7	150 20.6	1050		
12 44 M	{Heart rate/min. {Cardiac output (l./min.)	105 4.7	130 7.4	160 9.8	190 12.3	—	468	78 6.0	100 8.0	123 10.0	140 12.8	—	965		

PWC₁₇₀ = Physical work capacity at a heart rate of 170/min.

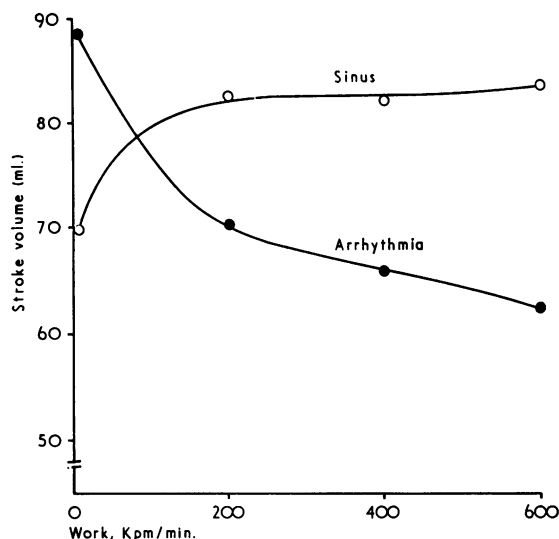


Fig. 8.—Stroke volume plotted against work load in Patient No. 2 studied in atrial fibrillation and in sinus rhythm six weeks after aortic valve replacement. Note the progressive fall in stroke volume with exercise during the arrhythmia.

of blood from the thorax to the legs. The effect of position on the circulation is therefore important and could be responsible for some of the conflicting reports on hæmodynamic changes before and after the conversion of arrhythmias to sinus rhythm. All measurements in this study whether at rest or on effort were made sitting on the bicycle ergometer.

The concept of the physical work capacity as a measure of the work performed per heart beat was put forward by Sjöstrand (1947) and by Wahlund (1948). The physical work performed by a patient pedalling a bicycle ergometer at a heart rate of 170 per minute was obtained in the present investigation from the linear relation between heart rate and work performed. The PWC_{170} is reduced in the presence of heart disease (Holmgren *et al.*, 1957), but in addition the present investigation has shown that the combined effects of the loss of atrial systole and a more rapid ventricular rate on exercise during the arrhythmia significantly lowers the work capacity.

There have been several reports on the changes in cardiac output before and after the conversion by quinidine of atrial fibrillation to sinus rhythm. The results are often conflicting. Storstein and Tveten (1955) found an increase in cardiac output when in sinus rhythm but attributed this in part to the effect of quinidine on the peripheral circulation and on the myocardium; this concept was later challenged by Broch and Müller (1957) who found no evidence

that quinidine was beneficial in the way suggested. Kory and Meneely (1951) failed to demonstrate an improvement in the cardiac output in sinus rhythm, but Hansen, McClendon, and Kinsman (1952) succeeded in doing so.

Numerous investigators have paid attention to the circulatory function of the atrium. Harvey (1628) described the action of the atrium as an act of contraction, throwing blood into the ventricle. Henderson (1906), however, concluded that in dogs the atrium was more important as an elastic reservoir, and recently Grant, Bunnell, and Greene (1964) concluded the same of man. Lewis (1912) showed that at a given rapid ventricular rate a regular or irregular ventricular contraction would lead to the same decrease in blood pressure, and he thought that the changes in the circulation recorded during atrial fibrillation were entirely rate dependent. Gesell (1911), however, had already shown that an appropriately timed atrial systole would augment blood pressure, and later (Gesell, 1916) was able to show that the immediate effect of atrial systole was to amplify the cardiac output by 30 per cent. Ventricular plethysmography was used by Straub (1910), Hirschfelder (1908), and Wiggers and Katz (1922) who concluded that ventricular filling was augmented by atrial systole and that this contribution no longer occurred in atrial fibrillation. Stewart, Crawford, and Hastings (1926) demonstrated that the cardiac output in dogs decreased by as much as 60 per cent during atrial fibrillation. It was suggested by Stewart, Crawford, and Gilchrist (1928) that this resulted from a decrease in the oxygen saturation of mixed venous blood. A disproportionate increase in the ventricular rate on exercise is found in patients with atrial fibrillation, as shown by Blumgart (1924). This is confirmed in the present series which shows, moreover, that even when digitalized to therapeutic levels the rate response to the stress of exercise is excessive in atrial fibrillation; some rate benefit does occur in the digitalized heart, however, as suggested by Modell, Gold, and Rothendler (1941).

It could be argued that the circulatory benefit in sinus rhythm is entirely rate dependent. Knox (1949) and Wetherbee, Brown, and Holzman (1952) agreed with earlier workers, that the major disability of atrial fibrillation was the rapid ventricular rate, especially on exercise. With the establishment of sinus rhythm and consequent slowing of the heart an increased diastolic filling time would result in a larger stroke volume and cardiac output. Stroke volumes were larger in sinus rhythm than in atrial fibrillation and atrial flutter at equivalent heart rates (Fig. 6), and increased more in sinus rhythm as the heart rate rose to 120 a minute. With a further

increase in heart rate, the stroke volume remained constant in sinus rhythm but began to fall during the arrhythmia. These figures are deduced from studies in which it is known that the oxygen uptake is similar and the work load is identical; as the heart rate is also similar the beneficial results in sinus rhythm cannot be explained only on the basis of an increased diastolic filling time, but must also be due to the benefit of atrial systole in sinus rhythm. This is in agreement with the conclusions of Skinner *et al.* (1964) who demonstrated that a fall in stroke volume and cardiac output occurred in anæsthetized dogs, with the onset of atrial fibrillation even when the ventricular rate remained constant.

The results in this series suggest that when the heart is healthy, the atrium functions as a "booster pump" to improve ventricular filling on exercise; when the heart is abnormal, however, the atrial contribution to ventricular filling is important even at rest, as shown by Braunwald and Frahm (1961). There is little confirmatory evidence in man that an efficient atrial systole prevents atrio-ventricular regurgitation, as suggested by Little (1951) and by Sarnoff, Gilmore, and Mitchell (1962), and Braunwald *et al.* (1966) failed to demonstrate mitral regurgitation on cine-angiography in patients lacking atrial systole.

SUMMARY

The hæmodynamic changes in 15 patients were studied at rest and during graded exertion before and after the electrical conversion of atrial fibrillation or flutter to sinus rhythm. There was no significant change at rest when the heart was normal apart from the arrhythmia, but atrial systole appeared important even at rest when the ventricle was diseased. On exercise, the beneficial effects of sinus rhythm were demonstrated with normal and abnormal hearts and the larger the exercise load the greater the benefit. A disproportionate increase in heart rate occurred when patients were exercised during atrial fibrillation and flutter even when digitalized, and the physical work capacity was reduced. The cardiac output and stroke volume were less for equivalent work loads during the arrhythmia. Equivalent exercise loads were performed in sinus rhythm at a slower heart rate, the physical work capacity was higher, and cardiac output and stroke volume were increased, resulting in a narrower arteriovenous oxygen difference. A paired "t" test demonstrated that the changes were highly significant. The stroke volumes were compared during the arrhythmia and in sinus rhythm at equivalent heart rates at rest and on exercise; in sinus rhythm, the stroke volume was larger and was held more constant as the heart rate increased

under the stress of exercise. The hæmodynamic benefit of atrial systole actively filling the ventricle during exercise or in the presence of a diseased ventricle was deduced.

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