

# THE CARDIAC OUTPUT IN ACUTE RHEUMATIC CARDITIS

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In the treatment of rheumatic fever by rest in bed, one of the most important clinical decisions needed is the stage in the recovery process when graded exercise may be safely initiated. Since the principle underlying this is the avoidance of extra work by the heart until it is fit again to undertake it, and since no direct information is available, we thought it advisable to investigate how the heart dealt with the mild degree of extra work entailed in a short period of exercise at various stages during the recovery process. The ultimate object was to ascertain the point at which the heart showed a normal response to effort.

The present observations deal with the relation between cardiac output, pulse rate, and oxygen consumption, using data obtained by cardiac catheterization. We felt justified in employing this method since we had used it earlier to establish the presence or absence of pericardial effusion in active cases and had at no time encountered ill-effects, discomfort, or distress. We were first concerned with the meaning of the tachycardia that has been traditionally accepted as a common sign of activity. This earlier work seemed to establish two facts. (1) In active rheumatic carditis uncomplicated by gross valve damage, pericardial effusion, or congestive heart failure, tachycardia did not appear to be disproportionate to fever and anxiety. (2) Tachycardia induced by means of atropine led to an increase of cardiac output which was proportional to the rate, and did not differ in degree from controls; the critical rate, beyond which any further increase led to a fall in output, was not established, but it could hardly have been less than in controls, for no decrease was observed with rates up to 160 (Hollman, 1950). While these investigations were in progress, parallel observations were being made on heart size. Using Wood's method of demonstrating pericardial effusion by means of cardiac catheterization, it was found that enlargement of the heart shadow in active rheumatic carditis nearly always depended on some severe valve lesion, congestive heart failure, or pericardial effusion (Wood, 1950; Besterman and Thomas, 1953), and rarely occurred otherwise. Since the venous pressure and blood pressure were also normal in uncomplicated cases, it looked as if the circulation as a whole was not disturbed. It was decided to check this conclusion by measuring the effect of effort on the cardiac output itself, and seeing whether cases of rheumatic carditis behaved similarly to cases in whom recovery had occurred.

## MATERIAL

Seventeen patients were investigated, ten with acute rheumatic carditis and seven without. The diagnosis of active rheumatic carditis depended on a raised sedimentation rate and failure to gain weight in patients who had recently exhibited polyarthritis, fever, and changing murmurs or pericarditis (Bywaters, 1950). Six of the ten active cases were suffering from their first attack of rheumatic fever and, apart from one with pericardial effusion, none had a raised venous pressure, cardiac enlargement, or severe valve lesions. Cortisone or A.C.T.H. had been given to four, and salicylates to one, and all had been treated with rest in bed.

Six of the seven controls had recently recovered from their first attack of acute rheumatic carditis and were believed to be inactive at the time of catheterization. Four of them had mild residual valve lesions but the other two seemed entirely normal. Three of these patients had been catheterized previously during the

active phase of the disease and thus appear in both groups. In order to eliminate possible differences of behaviour due to prolonged rest in bed, the inactive patients (with one exception) were catheterized before mobilization. The seventh patient differed from the others in that she had had active rheumatism for five weeks without demonstrable carditis, and served as a control against the possibility that acute rheumatic fever itself might influence the behaviour of the cardiac output at rest or on exercise.

#### METHOD

Routine premedication included quinidine, nembital or amytal, and penicillin. A number 7, 8, or 9 nylon catheter, connected to a saline manometer, was used. Mixed venous samples were obtained from the main pulmonary artery and were analyzed in a Haldane blood gas apparatus. The oxygen consumption was measured by means of a Benedict-Roth spirometer. Since, in several trial cases, the oxygen uptake obtained by means of the usual nose-clip and mouthpiece was found to be the same as that obtained with the use of an anaesthetic mask, precautions against leakage were considered adequate. In preliminary tests the use of air instead of oxygen for filling the spirometer had no effect on either the oxygen uptake or cardiac output; oxygen was used throughout this study.

It was feared that continuous spirometric tracings might prove too irregular during effort to allow accurate estimations of oxygen consumption to be made at precise moments when blood samples were withdrawn, but in practice little difficulty was encountered in this respect. The patient exercised with a bicycling movement of the legs, the feet driving pedals against an artificial load applied by an adjustable friction resistance. The actual load was not calculated, but was adjusted for each patient according to his work capacity. The ergometer clipped on to the radiological couch, thus allowing the subject to exercise while lying horizontally, and without disturbing the position of the catheter. In the hope of obtaining a more or less basal resting cardiac output, initial samples were not taken until at least twenty minutes after the pulmonary artery had been catheterized. The patient was then exercised and 5-ml. samples were withdrawn after one minute and again after two or three minutes pedalling. Another sample was taken three minutes after ceasing exercise, and usually a final one about fifteen minutes later. Serial arterial samples before, during, and after exercise were analysed in one typical case of active rheumatic carditis, and were all normally saturated with oxygen. In view of this, only a single arterial sample was obtained in the others, to make sure there was no unexpected unsaturation. As previously stated, none of these patients had heart failure, and none had any other reason to have a lowered arterial oxygen saturation; in fact all arterial samples were normal.

#### RESULTS

The behaviour of the cardiac output on effort in each of the seventeen patients is shown individually in Fig. 1, the actual values being presented in the Table. To simplify these results, a single composite graph has been constructed for each group in Fig. 2, which also shows composite curves for pulse rate and stroke volume. In plotting mean cardiac outputs and pulse rates some discretion had to be allowed in respect of the precise time relationship between the samples, for these were not identical in each case. Thus mean values plotted at -30 to -11 minutes represent all observations made between 30 and 11 minutes before the start of exercise. Subsequent points on the graph both before and after exercise similarly represent mean figures computed from observations made during the time intervals stated. During effort, mean values for cardiac output and pulse rate have been calculated from data obtained after one, two, and three minutes pedalling.

The pulse rate immediately before exercise was similar in both groups. The mean value was 87 in those with active carditis and 93 in the controls. The resulting cardiac output was raised in all cases, the arterio-venous oxygen difference being low and the oxygen consumption rather high. The mean output in those with carditis was lower (9.6 l./min) than in the controls (13.0 l./min.). The systemic peripheral resistance at rest ranged between 358 and 1160 dynes sec./cm.<sup>5</sup> in those with carditis and between 500 and 770 dynes sec./cm.<sup>5</sup> in the controls.

During exertion the mean rise of pulse rate was 24 beats a minute in the cases with active carditis and 28 in those without carditis. The mean increase of arterio-venous difference was similar in both groups (22.2 ml./l. and 26.5 ml./l. respectively), whereas the mean rise of oxygen uptake during

TABLE  
DATA OBTAINED BEFORE AND DURING EXERCISE IN ACTIVE CARDITIS AND IN THE CONTROLS

Initial and age	Pulse rate		Venous unsaturation ml./l		Oxygen uptake ml./min.		Cardiac output l./min.		Peripheral resistance dynes sec./cm. <sup>5</sup>	Arterial unsaturation ml./l.	
	R.	E.	R.	E.	R.	E.	R.	E.	R.	R.	
					<i>Cases with Active Carditis</i>						
O.W. 12	100	130	35.5	56.0	220	330	8.8	7.25	682	10.5	
P.D. 16	58	72	37.5	58.0	225	390	8.0	7.9	980	9.5	
A.S. 13	79	124	38.0	76.0	230	500	7.7	7.35	935	8.0	
T.S. 13	100	—	36.5	44.0	240	295	9.1	8.7	810	10.0	
T.E. 12	116	124	41.5	52.0	240	340	8.4	8.75	790	13.0	
J.B.* 12	90	112	27.0	50.0	260	565	14.5	13.8	358	9.0	
M.W.* 11	64	104	31.0	73.5	186	525	8.1	8.0	720	8.0	
J.E.* 12	96	112	22.0	48.0	210	560	15.0	14.0	415	8.0	
P.D. 12	128	152	49.5	69.5	213	350	5.7	6.1	1160	12.0	
F.T. 12	116	132	30.0	43.0	195	310	10.8	10.0	555	12.0	
					<i>Control Cases</i>						
D.G. 15	90	111	25.5	38.5	230	575	13.1	18.85	672	8.0	
P.B. 11	110	132	31.0	62.5	250	770	13.5	15.4	516	12.5	
M.W.* 11	96	—	26.5	38.5	210	660	12.7	23.0	535	10.0	
J.B.* 12	79	111	45.0	70.5	280	1625	8.1	27.0	770	10.5	
J.E.* 12	100	—	32.0	71.75	285	1100	13.6	18.1	500	11.0	
N.P. 14	100	136	31.5	72.0	290	1125	14.3	18.5	615	11.0	
J.H. 20	98	128	33.0	55.5	330	860	13.2	18.3	605	8.0	

\* Indicates three cases catheterized twice, during rheumatic activity and during convalescence.  
R=data obtained at rest. E=data obtained during exercise.

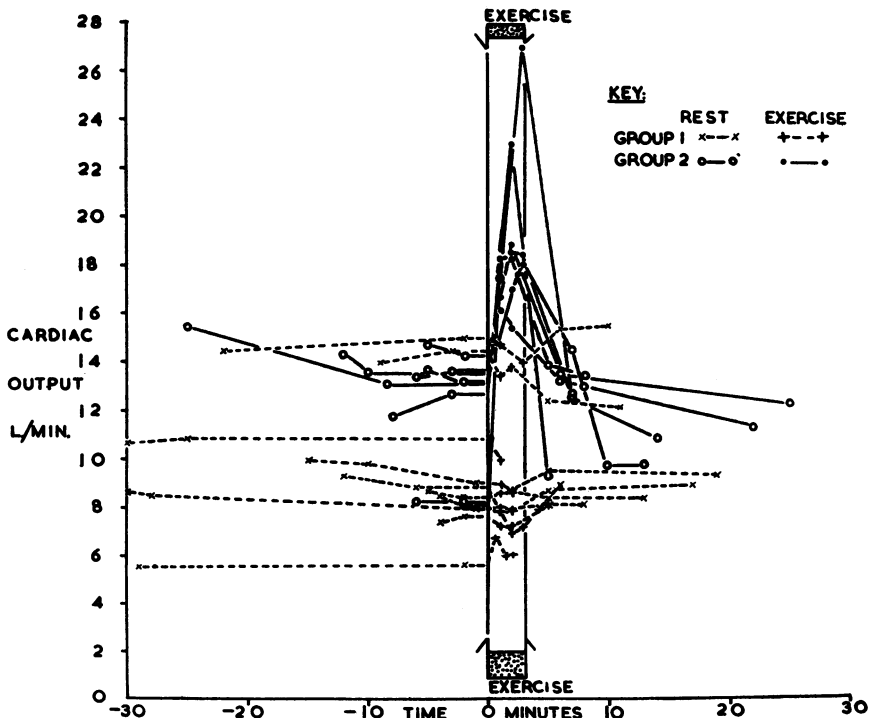


FIG. 1.—Response of cardiac output to exercise in ten cases of active carditis (group 1) and seven control cases (group 2).

exercise was far greater in the controls (Fig. 3). The mean oxygen consumption rose by 195 ml./min. in the cases with active carditis as opposed to a mean increase of 690 ml. in those without carditis. The behaviour of the cardiac output was also very different in the two groups. The output changed but little in the cases with active carditis, but increased significantly during exercise in all the controls, including the one patient with acute rheumatic fever without carditis. The most severely ill patient in the active group, suffering from pericardial effusion, had the lowest cardiac output. The mean change of output on exertion was  $-0.4$  l./min. in the active cases and  $+6.9$  l./min. in the controls. Thus the stroke volume diminished considerably during exercise in the first group, and increased in the second (Fig. 2).

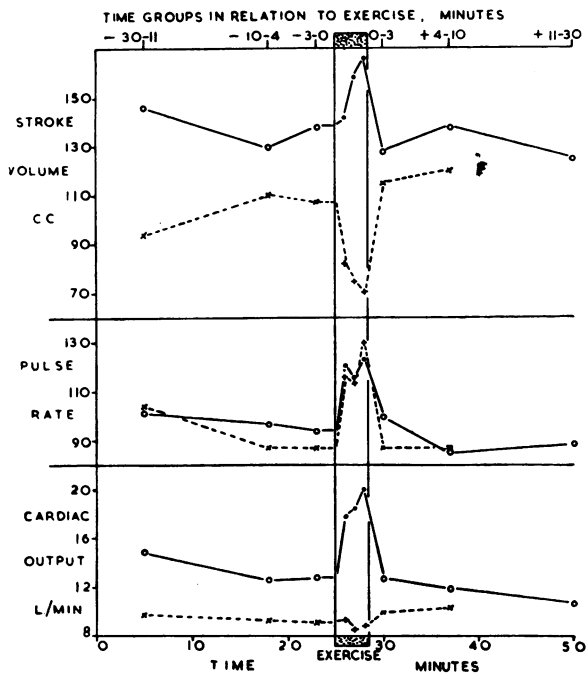


FIG. 2.—Mean values of cardiac output, pulse rate, and stroke volume before, during, and after exercise in the cases of active carditis (interrupted line) and in the control group (continuous line).

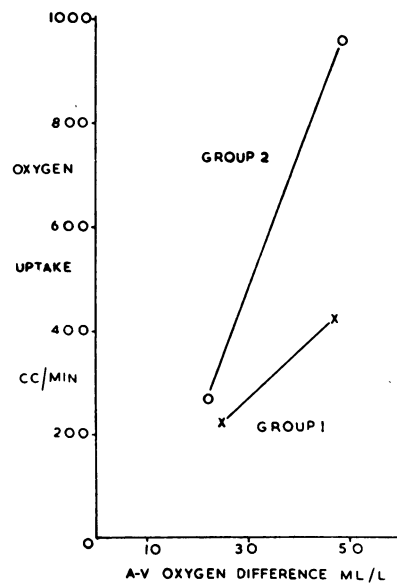


FIG. 3.—Effect of exercise on mean oxygen consumption and mean arterio-venous oxygen difference in active cases (group 1) and controls (group 2).

## DISCUSSION

The increase in oxygen utilization by the tissues during exercise is reflected in a rise of arterio-venous oxygen difference, cardiac output, and oxygen uptake from the lungs. Strenuous exertion may raise the cardiac output above 30 l./min. in a normal subject. The presence of mild cardiac lesions, without failure, does not prevent a rise of output (Dexter, 1951). Anxiety usually increases the cardiac output and was probably responsible for the wide range of normal adult resting values (4.2 l./min. to 14.8 l./min.), observed by Stead and Warren (1945). In the same study, the resting systemic peripheral resistance ranged from 475 to 1580 dynes sec./cm.<sup>5</sup> If the normal rise of output cannot occur, as in "low output" failure, exercise induces an extreme degree of venous unsaturation with only a small rise of oxygen uptake. Failure to raise the cardiac output with exertion has also been reported in cases of anxiety without cardiac lesions (Hickam and Cargill, 1948).

In the present series, the cardiac output at rest was similar in both active and inactive cases; whether the output falls to normal or remains raised in the active cases under truly basal conditions

is not known, and the findings described above throw no light on this question. In the cases of active carditis, the heart responded to the demands of exercise by an increase of rate. However, the stroke volume simultaneously diminished, and no rise of minute output occurred. Thus the cardiac reserve must have been reduced, and the level of output in these patients at rest was the maximum attainable by them. Had further exertion been allowed it might have induced cardiac decompensation. Right atrial pressures were not recorded during exercise, but mean pulmonary artery pressures showed no change in those cases in which it was measured. It has not been possible to correlate the histological findings in rheumatic carditis with this impairment of function, but it may be presumed that active rheumatism alters some biochemical or biophysical property of heart muscle in a manner that causes the heart to be a less efficient pump.

The actual amount of work done during exercise was not known, but since the A-V difference did not increase more in the cases of active carditis than in the controls, whereas the oxygen consumption rose far less, it is obvious that the active cases performed less work. It is of interest that despite prolonged rest in bed and a recent severe illness, all the control cases showed a normal cardiac response to exertion.

#### SUMMARY

The cardiac output at rest, during exercise, and afterwards was measured in ten patients with acute rheumatic carditis without heart failure or advanced valve disease, and in seven patients after recovery. The output at rest was high in both groups, but was higher in the latter.

On exertion, the output did not change significantly in the active cases, but rose conspicuously in those who had recovered. The active cases performed less work than the others, but sufficient to have produced a rise in output if that had been possible. It is concluded that active rheumatic carditis impairs the functional efficiency of the myocardium.

It should *not* be concluded that the cardiac output at rest in cases of active rheumatic carditis is necessarily set at its maximum level, for truly basal resting conditions could not be attained during cardiac catheterization in the patients studied.

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