Disparities in ventilatory and circulatory responses to bicycle and treadmill exercise¹

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Haemodynamic and ventilatory responses, during multilevel bicycle exercise and during multilevel symptom-limited treadmill exercise, were compared in 8 patients with coronary heart disease and in one sedentary middle-aged man, at known percentages of each subject's maximal oxygen uptake ($\dot{V}O_{2max}$), determined on a treadmill.

When comparisons were made at the same percentages of $\dot{V}O_{2max}$ on treadmill or bicycle, we found higher arterial mean pressure, heart rate, pressure-rate product, peripheral vascular resistance, and pulmonary ventilation during bicycle exercise. Cardiac output was the same and stroke volume was lower on the bicycle.

We conclude that in terms of arterial blood pressure and pressure-rate product, all conditions being as mentioned above, bicycle exercise constitutes a greater stress on the cardiovascular system at any given oxygen uptake than does treadmill exercise.

The mechanism of the observed differences in the responses to the two types of exercise may reflect potentiation of normal reflexes initiated by working muscles when arterial inflow is impeded by more vigorous contractions, especially in individuals not conditioned for bicycling. The clinical importance of these findings in relation to the evaluation of exercise performance of patients with cardiovascular disease is that slightly higher values of $\dot{V}O_{2max}$ are achieved with slightly less haemodynamic stress on the diseased left ventricle with treadmill exercise.

Inasmuch as normally there are greater circulatory and ventilatory responses to bicycle exercise in terms of heart rate and pulmonary ventilation at submaximal exercise and in terms of blood pressure at maximal exertion (Hermansen, Ekblom, and Saltin (1970), yet lower limits of oxygen uptake ($\dot{V}O_2$) and cardiac output ($\dot{Q}$) than are observed with treadmill exercise (Åstrand and Saltin, 1961; Hermansen and Saltin, 1969; Hermansen *et al.*, 1970; Miyamura and Honda, 1972), there is a legitimate question as to which method imposes the greater stress on cardiovascular patients. This problem is particularly relevant to those patients who develop myocardial ischaemia on exertion, whether in the context of exercise stress testing or of exercise conditioning for cardiac rehabilitation.

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This study was designed to compare the haemodynamic responses, primarily of patients with coronary heart disease, to bicycle and treadmill exercise performed within one to two hours. In order to obtain valid comparisons, data were selected from a series of observations to represent responses to exercise on both bicycle and treadmill with similar relative aerobic requirements, or percentages of highest possible oxygen uptake ($\dot{V}O_{2max}$) for each person.

Subjects and methods

Physical and clinical characteristics of 1 normal middle-aged man and 8 patients with coronary heart disease are shown in Table 1. None of the subjects had valvular heart disease, was anaemic, or had evidence of pulmonary disease by chest x-ray and pulmonary functional screening tests. None of them suffered from intermittent claudication or from skeletal muscular disease limiting exercise performance. None was treated with digitalis or beta-blocking agents for at least two weeks before the study. Physical activity status was defined as active if the

TABLE I Physical and clinical characteristics

Subjects (men)	Diagnosis	NYHA class	Activity status	Age (yr)	Height (cm)	Weight (kg)	Relative weight* (%)	Blood pressure at rest (mmHg)	
								Systolic	Diastolic
1	Normal	I	Sedentary	59	182	83.2	100	128	96
2	ASHD, post SVG	I	Active	63	170	65.9	90	160	105
3	ASHD, HMI, post SVG	III	Active	60	174	69.1	90	130	72
4	ASHD, HMI, AP	I	Sedentary	41	183	79.5	95	126	86
5	ASHD, HMI, AP	III	Sedentary	50	178	70.4	80	130	100
6	ASHD, HMI, essential hypertension	IV	Active	50	156	66.4	106	170	100
7	ASHD, post SVG	I	Active	40	180	88.2	108	132	80
8	ASHD, 2 HMI, post SVG	II	Active	58	170	71.8	98	188	90
9	ASHD, HMI, AP	I	Sedentary	49	170	62.3	85	130	80
				52 ± 8	174 ± 8	73.0 ± 8.7	95 ± 8	144 ± 23	90 ± 11

* Relative weight = $\frac{\text{Observed weight}}{\text{Predicted weight for height by regression}} \times 100$

where Predicted weight in kg for men = $60.7 + 0.79 (\text{height in cm})$.

Abbreviations: AP angina pectoris; ASHD atherosclerotic heart disease; HMI healed myocardial infarction; SVG saphenous vein grafting.

subjects performed exercise strenuous enough and long enough to cause sweating regularly at least once a week (McDonough, Kusumi, and Bruce, 1970). All subjects performed a control treadmill test (Bruce, Kusumi, and Hosmer, 1973), usually on the day before the haemodynamic studies. Functional aerobic impairment was derived from the duration of this control test by use of nomograms, taking age, sex, and physical activity status into account. After explanation of the test and signed informed consent, a Swan-Ganz catheter (French size 7) was placed through a left cubital vein into a pulmonary artery and a 'teflon' catheter was inserted into the left radial or brachial artery by the Seldinger technique. Pressures were recorded by use of the same Statham pressure transducers (P23Gb for systemic and P23V for pulmonary arterial pressures), and mean pressures were derived by electrical averaging. The zero pressure reference level was adjusted at the height of the anterior fourth intercostal space in the upright position; as the gradient of the treadmill increased, it was moved upward with the subject.

The tracing of a bipolar transthoracic electrocardiographic lead (Bruce, 1971) was continuously displayed on a scope, together with both systemic and pulmonary arterial pulsatile pressures. Heart rate was obtained from an electrocardiographic tracing recorded at 25 mm/sec paper speed. ST segment depression was also defined visually from this tracing; depression of more than 0.1 mV for 0.06 sec of the segment was considered abnormal. Cardiac output (Q) was estimated by the direct Fick method. $\dot{V}O_2$ was measured from 180 sec samples of expired air breathed into evacuated neoprene bags at rest and from 60 sec samples during exercise. Arterial mixed venous oxygen difference ($AV O_2 D$) was calculated from the oxygen contents, measured by use of a LEX- O_2 -CON (Kusumi, Butts, and Ruff, 1973), of arterial and mixed venous blood (sampled from the

brachial or radial and from the pulmonary artery). After placement of the catheters, the subjects (except one, who performed the treadmill test first) were seated on a Monark bicycle ergometer. Saddle height was adjusted as chosen by the subject. The subjects were allowed to hold the handlebars with both hands, but were cautioned against a strong handgrip.

Lowest workload and increases in workload were adjusted to the circulatory and physical capacity of each subject, as evaluated during the control treadmill test, in order to obtain at least four measurements within the subject's functional limits. Thus, the rates of increase in workload ranged from 12.5 to 50 watts.

Each workload was kept constant for 4 minutes, except the highest workload which was usually terminated after 3 minutes. Measurements were taken at rest, while pedalling against 0 watts, in the last minute of each workload and in the last 2 to 3 minutes of exercise. After an hour of supine bed rest, the subjects performed a multilevel symptom-limited treadmill test. Six subjects started at stage 1/2 (1.7 miles/hr, 5% gradient) and 3 started at stage 1 (1.7 miles/hr, 10% gradient). Thereafter the test followed the standardized procedure as described (Bruce *et al.*, 1973). Measurements were taken while standing at rest, in the last minute of each stage, and in the last 2 to 3 minutes of exercise. Durations of these treadmill tests are not comparable with those of the control treadmill tests because the patients could rest some weight on an arm support, necessary to keep the catheterized arm in a steady position. After completion of the treadmill test, the catheters were removed and the subjects were kept under medical supervision for at least two hours and were then discharged from the hospital. No complications occurred.

The data were analysed with reference to per cent $\dot{V}O_{2\max}$, which expresses absolute $\dot{V}O_2$ as a percentage of the highest $\dot{V}O_2$ observed during maximal treadmill

TABLE 2 Circulatory and electrocardiographic responses to maximal exercise (treadmill)

Subjects	Dura- tion (sec)	Func- tional aerobic impair- ment(%)	$\dot{V}O_2$ max (ml/min)	$\dot{V}O_2$ max ml/ (kg·min)	Max. heart rate/ min (beats)	Change in heart rate/ min (beats)	Max. systolic blood pressure (mmHg)	Change in systolic blood pressure (mmHg)	Max. pressure rate product/ 100	ST depres- sion
2	420	14	2420	29.1	160	65	212	84	339	—
3	555	—5	2001	30.4	175	93	166	6	290	+
4	413	18	1999	28.9	131	51	175	45	229	+
5	416	36	2128	26.8	175	99	162	40	283	+
6	401	30	1616	23.0	180	84	188	58	338	+
7	450	25	1971	29.7	175	103	210	40	367	+
8	628	15	3093	35.1	190	120	204	72	387	+
9	380	24	1713	23.9	160	102	180	—8	287	+
	428	34	1802	28.9	148	76	180	50	266	+
	454 ± 82	21 ± 13	2083 ± 447	28.4 ± 3.6	166 ± 18	88 ± 21	186 ± 19	43 ± 29	310 ± 51	

exercise. Statistical analysis used Students' paired t test. P values larger than 0.05 were considered not significant.

Results

1) Responses to maximal exercise on treadmill (control test)

Maximal exercise elicited $\dot{V}O_2$ max of 2083 ± 447 ml/min, 28.4 ± 3.6 ml/kg·min. Functional aerobic impairment of 14 per cent for the normal subject was well within the normal range, while the mean was 21 per cent for the entire group (Table 2).

Similarly, circulatory responses were typical of individuals with only mild functional aerobic impairment. All 8 patients exhibited horizontal or downsloping ST segment depression (< -0.1 mV) after this exertion; the normal subject did not.

2) Comparison of haemodynamic responses

The major findings were that at all equivalent levels of $\dot{V}O_2$ and percentages of $\dot{V}O_2$ max, mean systemic arterial blood pressure ($\bar{P}_{SA}$) and pressure

TABLE 3 Haemodynamic responses to upright exercise, bicycle vs. treadmill, mean values $\pm$ SD

Type of exercise	Rest		Pedalling without resistance 0 watt	Workloads requiring 40–60 per cent $\dot{V}O_2$ max		Workloads requiring 61–85 per cent $\dot{V}O_2$ max	
	(sitting)	(standing)		Bicycle	Treadmill	Bicycle	Treadmill
No.	9	9	9	9	9	9	9
Watts or stage	—	—	0	56 ± 17	1–1' ± 1.6'*	96 ± 27	11–2' ± 2.0'
$\dot{V}_E$ (l/min)	8.9 ± 3.1	10.8 ± 4.0	10.6 ± 2.1	25.8 ± 7.0	22.2 ± 4.3	46.0 ± 14	37.2 ± 12.4†
$\dot{V}O_2$ (ml/min)	309 ± 43	348 ± 63	428 ± 64	1069 ± 236	1090 ± 149	1553 ± 402	1559 ± 443
Per cent $\dot{V}O_2$ max	15.2 ± 2.9	16.9 ± 3.1	21.1 ± 4.6	51.5 ± 4.5	52.8 ± 3.5	74.1 ± 8.4	74.1 ± 8.2
AV O ₂ D (ml/l.)	70.9 ± 5.9	77.6 ± 13.6	77.9 ± 6.2	112.6 ± 9.1	113.7 ± 12.8	133.9 ± 11.0	132.3 ± 14.1
Q (l/min)	4.4 ± 0.8	4.6 ± 1.0	5.5 ± 0.8	9.5 ± 2.0	9.7 ± 2.1	11.6 ± 2.8	11.8 ± 3.0
HR (beats/min)	83 ± 17	93 ± 16‡	87 ± 12	122 ± 19	113 ± 12	149 ± 16	137 ± 19‡
SV (ml)	54 ± 13	50 ± 15	63 ± 7	78 ± 11	86 ± 17	78 ± 18	85 ± 16†
$\bar{P}_{SA}$ (mmHg)	100 ± 12	102 ± 10	116 ± 16	128 ± 21	111 ± 14†	147 ± 19	118 ± 15§
$\bar{P}_{PA}$ (mmHg)	12 ± 2	14 ± 2‡	16 ± 3	22 ± 4	23 ± 3	28 ± 5	25 ± 4
PR/100	87 ± 19	94 ± 18	101 ± 19	157 ± 43	125 ± 21‡	220 ± 43	162 ± 33§
Systemic resistance	2004 ± 509	1863 ± 465	1736 ± 474	1102 ± 244	958 ± 263‡	1078 ± 334	846 ± 244§

* Explanation of the — SD is that some patients start with stage '1/2', i.e. — 3 minutes.

† P < 0.01. ‡ P < 0.05. § P < 0.001 (paired t test).

Abbreviations: AV O₂ D = arterial mixed venous oxygen difference; HR = heart rate; $\bar{P}_{PA}$ = mean pulmonary arterial pressure; PR = pressure rate product; Q = cardiac output; $\bar{P}_{SA}$ = mean systemic arterial pressure; SV = stroke volume; $\dot{V}_E$ = total ventilation; $\dot{V}O_2$ = oxygen uptake; $\dot{V}O_2$ max = maximal oxygen uptake.

FIG. 1 Measurements during bicycle and treadmill exercise of mean systemic ($\bar{P}_{SA}$) and mean pulmonary arterial pressures ($\bar{P}_{PA}$) plotted against per cent $\dot{V}O_2$ max. During bicycle exercise, all subjects had higher $\bar{P}_{SA}$ than at comparable $\dot{V}O_2$ on the treadmill.

Regression equations are $\bar{P}_{SA}$: bicycle: $y = 107 + 0.0479x$; treadmill: $y = 95 + 0.0310x$;
 $\bar{P}_{PA}$: bicycle: $y = 12 + 0.0202x$; treadmill: $y = 12 + 0.0198x$.

rate product were higher during exercise on the bicycle than on the treadmill (Fig. 1 and 2).

Pertinent baseline haemodynamic data from sitting at rest on the bicycle and standing at rest on the treadmill before exercise are given in Table 3. Pedalling against zero resistance increased $\dot{V}O_2$ by 38 per cent and reduced pooling of blood in the legs. This lowest level of bicycle exercise caused a 25 per cent increase in cardiac output ($\dot{Q}$), accounted for by increases of 16 per cent in stroke volume (SV) and of 5 per cent in heart rate, accompanied by a rise in mean pulmonary arterial pressure ($\bar{P}_{PA}$) from 12 to 16 mmHg.

Exercise of moderate intensity with relative aerobic requirements of around 50 per cent $\dot{V}O_2$ max was associated with a tendency towards larger pulmonary ventilatory volumes on the bicycle (an average difference of 16%). There were no differences in $\dot{Q}$ and AV O_2 difference, but heart rate tended to be faster (8%) and stroke volume was accordingly smaller on the bicycle. $\bar{P}_{SA}$, because of greater systemic resistance, and pressure rate product were 15 per cent and 26 per cent higher, respectively.

More strenuous exercise, eliciting around 75 per cent $\dot{V}O_2$ max, caused even greater differences, all of them statistically significant. Measurements of ventilatory volumes during bicycle exercise exceeded those during treadmill exercise by 24 per cent, of $\bar{P}_{SA}$ and systemic resistance by 25 per cent and 27 per cent respectively, and of heart rate by 9 per cent, while $\dot{Q}$ and AV O_2 difference were essentially the same, and stroke volume was 8 per cent smaller during bicycle exercise; pressure rate product, however, was 39 per cent greater during bicycle exercise.

Discussion

At all comparable submaximal workloads, requiring similar percentages of $\dot{V}O_2$ max, bicycle exercise caused greater rise in $\bar{P}_{SA}$ than did treadmill exercise, accompanied by faster heart rate and by larger ventilatory volumes/minute. A sedentary middle-aged man with functional aerobic impairment of 14 per cent showed similar responses. The highest workloads on the bicycle were subjectively felt by the patients to approach their working capacity, but the test protocol was not designed to

FIG. 2 Values for PR/100 versus per cent $\dot{V}O_2$ max. Regression equations are: bicycle: $y = 60 + 0.200x$; treadmill: $y = 48 + 0.151x$.

reach symptomatic limits on the bicycle. In some patients, however, haemodynamic measurements, such as $\bar{P}_{SA}$ and pressure rate product, obtained during such bicycle exercise, reached or slightly exceeded values obtained during maximal symptom-limited treadmill exercise.

The observed differences in $\bar{P}_{SA}$ measurements were not due to variations in adjustments of the zero pressure reference level, since similar pulmonary arterial pressures which were recorded during both forms of exercise remained the same. With one exception, the bicycle test was performed first, but repetition of such bicycle exercise in coronary patients does not result in different haemodynamic responses (Malmberg, 1965). Similarly, in a different group of coronary patients, we found satisfactory reproducibility of circulatory measurements when a multistage symptom-limited treadmill test was repeated after bed rest of 1–1.5 hours (Niederberger *et al.*, 1974).

Strenuous isometric muscle contraction, such as during handgrip, is associated with increased $\bar{P}_{SA}$ and other cardiovascular responses (Lind *et al.*, 1964; Bruce *et al.*, 1968; Siegel *et al.*, 1972). For our subjects, this should not have been an important contributory factor, because we cautioned them to avoid any gripping of the handlebar, which may occur during strenuous bicycle exercise. If present

at all, these factors should not have been more important than in the investigation of Hermansen *et al.* (1970), who found in 13 young normal men significant differences in $\bar{P}_{SA}$ between bicycle and treadmill exercise at maximal effort, but not at submaximal exercise levels. At submaximal levels, however, they also found a trend towards higher heart rate and lower stroke volume for the same $\dot{Q}$ on the bicycle, together with greater pulmonary ventilation.

Thus, there are similar qualitative differences between the two forms of exercise in normal young men and middle-aged men with coronary heart disease, but the magnitude of difference, at least in $\bar{P}_{SA}$, is greater in the latter. Explanations may lie in the pathophysiology of cardiovascular function in patients with ischaemic heart disease and with hypertension, but no direct attempt was made to answer specific questions in this regard. However, similar findings in one healthy, middle-aged man with only slightly raised diastolic blood pressure suggest normal circulatory regulation in our subjects. If the muscular pain in the muscles of the thighs, which usually occurred during bicycle exercise in our subjects, indicated high muscle tension and muscle ischaemia, then a possible explanation of their hypertensive and hyperventilatory responses is that these factors caused

potentiation of normal reflex mechanisms originating from working musculature when arterial inflow is impeded (Asmussen and Nielsen, 1964; Lind *et al.*, 1964).

However, greater $\dot{V}_{SA}$ responses on the bicycle were also measured in the absence of leg or thigh pain or before such pain developed. A possible explanation for differences between these measurements and the findings in young men (Hermansen *et al.*, 1970) is that in our middle-aged men the leg musculature was not conditioned for bicycling, but was relatively well conditioned for walking. Therefore, during bicycling there was a greater strain on muscle groups or individual muscle fibres in the legs and this evoked increased stimuli for reflex mechanisms (Paterson, 1928; Coote, Hilton, and Perez-Gonzales, 1971). In contrast, on the treadmill the strain may have been more uniformly distributed over a greater mass of working muscles.

The data, in conclusion, indicate that in our subjects bicycle exercise caused greater ventilatory responses and constituted a greater stress in terms of arterial blood pressure and pressure-rate product than did comparable treadmill exercise.

These findings are of clinical importance, as they indicate that in some patients with ischaemic heart disease treadmill exercise may allow achievement of appreciably higher $\dot{V}O_2$ than does bicycle exercise. This also applies to patients who are limited by angina pectoris, of which the onset is often closely related to a given pressure rate product (Robinson, 1967).

Even at maximal exertion on the treadmill, $\dot{V}_{SA}$ may not exceed values measured on the bicycle at relatively low workloads. This can have significant implications with respect to relative hazards in the type of stress testing or exercise conditioning used for older healthy persons or for patients with cardiovascular disease, such as angina pectoris or arterial hypertension. Also a lower arterial pressure for a given aerobic requirement may diminish the frequency and severity of exertional arrhythmias (Gey *et al.*, 1973).

References

- Asmussen, E., and Nielsen, M. (1964). Experiments on nervous factors controlling respiration and circulation during exercise employing blocking of the blood flow. *Acta Physiologica Scandinavica*, **60**, 103.
- Åstrand, P.-O., and Saltin, B. (1961). Maximal oxygen uptake and heart rate in various types of muscular activity. *Journal of Applied Physiology*, **16**, 977.
- Bruce, R. A. (1971). Exercise testing of patients with coronary heart disease. Principles and normal standards for evaluation. *Annals of Clinical Research*, **3**, 323.
- Bruce, R. A., Kusumi, F., and Hosmer, D. (1973). Maximal oxygen intake and nomographic assessment of functional aerobic impairment in cardiovascular disease. *American Heart Journal*, **85**, 546.
- Bruce, R. A., Lind, A. R., Franklin, D., Muir, A. L., MacDonald, H. R., McNicol, G. W., and Donald, K. W. (1968). The effects of digoxin on fatiguing static and dynamic exercise in man. *Clinical Science*, **34**, 29.
- Coote, J. H., Hilton, S. M., and Perez-Gonzales, J. F. (1971). The reflex nature of the pressure response to muscular exercise. *Journal of Physiology*, **215**, 789.
- Gey, G. E., Fisher, L. D., Pettet, G. E. M., and Bruce, R. A. (1973). Exertional arrhythmia and nitroglycerin. *Journal of the American Medical Association*, **226**, 287.
- Hermansen, L., Ekblom, B., and Saltin, B. (1970). Cardiac output during submaximal and maximal treadmill and bicycle exercise. *Journal of Applied Physiology*, **29**, 82.
- Hermansen, L., and Saltin, B. (1969). Oxygen uptake during maximal treadmill and bicycle exercise. *Journal of Applied Physiology*, **26**, 31.
- Kusumi, F., Butts, W. C., and Ruff, W. L. (1973). Superior analytical performance by electrolytic cell analysis of blood oxygen content. *Journal of Applied Physiology*, **35**, 299.
- Lind, A. R., Taylor, S. H., Humphreys, P. W., Kenelly, B. M., and Donald, K. W. (1964). The circulatory effects of sustained voluntary muscle contraction. *Clinical Science*, **27**, 229.
- McDonough, J. R., Kusumi, F., and Bruce, R. A. (1970). Variations in maximal oxygen intake with physical activity in middle-aged men. *Circulation*, **41**, 743.
- Malmberg, R. O. (1965). A clinical and hemodynamic analysis of factors limiting the cardiac performance in patients with coronary heart disease. *Acta Medica Scandinavica*, **177**, Suppl. 426, 1.
- Miyamura, M., and Honda, Y. (1972). Oxygen intake and cardiac output during maximal treadmill and bicycle exercise. *Journal of Applied Physiology*, **32**, 185.
- Niederberger, M., Bruce, R. A., Frederick, R., Kusumi, F., and Marriott, A. (1974). Reproduction of maximal exercise performance in patients with angina pectoris despite ouabain treatment. *Circulation*, **49**, 309.
- Paterson, W. D. (1928). Circulatory and respiratory changes in response to muscular exercise in man. *Journal of Physiology*, **66**, 323.
- Robinson, B. F. (1967). Relation of heart rate and systolic blood pressure to the onset of pain in angina pectoris. *Circulation*, **35**, 1073.
- Siegel, W., Gilbert, C. A., Nutter, D. O., Schlant, R. C., and Hurst, J. W. (1972). Use of isometric handgrip for the indirect assessment of left ventricular function in patients with coronary atherosclerotic heart disease. *American Journal of Cardiology*, **30**, 48.

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