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Pulmonary Artery Pressure Changes During Exercise and Daily Activities in Chronic Heart Failure

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Long-term continuous pulmonary artery pressure monitoring was used to investigate pressure changes during different types of exercise and normal daily activities in patients with chronic heart failure. Nine men (mean age 55 years) with treated chronic heart failure underwent continuous pulmonary artery pressure measurement with use of a micromanometer-tipped catheter with in vivo calibration and frequency-modulated recording.

The mean (± SD) maximal systolic pulmonary artery pressure (in mm Hg) was 59.4 ± 26.1 on treadmill exercise, 54.9 ± 30.6 on bicycle exercise, 52.5 ± 26.1 walking up and down stairs and 43.5 ± 23.9 walking on a flat surface. The mean maximal diastolic pressure (in mm Hg) was 27.8 ± 14.6 on treadmill exercise, 25.5 ± 14.9 on bicycle exercise, 24.9 ± 14.8 walking up and down stairs and 20.4 ± 12.5 walking on a flat surface. The increase in pulmonary artery pressure did not correlate with the severity of the limiting symptoms except during walking on a flat surface.

All patients had marked postural changes in pressure, with the systolic pressure difference from lying to standing ranging from 8 to 25 mm Hg and the diastolic pressure difference ranging from 3 to 13 mm Hg. Eating meals caused an increase in pressure in three patients, but less than that when lying flat. There was an increase in pressure during urination in four patients equal to that when walking on a flat surface. None of these activities was associated with symptoms.

Neither symptoms nor pulmonary artery pressure during maximal exercise is the same as during daily activities. This may restrict the value of maximal exercise tests in assessing patients with chronic heart failure.

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The similarity between changes in pulmonary artery pressure during exercise tests and normal daily activities in patients with chronic heart failure is unknown. Exercise testing is an effective method of assessing the severity of functional impairment (1–4) and prognosis (5–7) of patients with chronic heart failure. Traditionally, pulmonary capillary wedge and pulmonary artery pressures at peak exercise have been used to investigate the pathophysiology and outcome of treatment in chronic heart failure. The demonstration of juxtapulmonary receptors that could be stimulated by an increase in pulmonary artery pressure and, thus, cause the sensation of breathlessness (8) supported the hypothesis that exercise pulmonary artery pressure and breathlessness may be related. The role of pulmonary artery pressure changes in causing the symptoms that limit exercise has been challenged (2,9–12). The pulmonary capillary wedge pressure at peak exercise does not correlate with the maximal rate of oxygen consumption or symptom-limiting exercise (13), and the excessive ventilatory response to exercise is not a result of acute changes in intrapulmonary pressure (14).

It has been suggested (2,15,16) that symptom-limited maximal exercise tests may differ in their hemodynamic consequences from those in the type of activities undertaken by patients during daily life, which mainly involve submaximal exercise. The purpose of this study was to compare pulmonary artery pressure at peak exercise with that during daily activities in patients with chronic heart failure. This problem was addressed by comparing pulmonary artery pressure during two conventional maximal exercise tests with that during two submaximal tests and with the effects of posture, lying in bed at night, meals and urination.

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Table 1. Clinical Features of Nine Patients

Patient No.	Age (yr)	NYHA Functional Class	VO_2 Max (ml O_2/kg per min)	LVEF (%)		Medication
				Rest	Exercise	
1	58	III	15.3	18	15	Bumetanide, 1 mg once daily
2	65	III	9.5	21	23	Furosemide, 80 mg, once daily, captopril, 25 mg, three times daily, nicardipine, 40 mg, three times daily, isosorbide mononitrate, 30 mg three times daily
3	53	II	15.9	21	30	Aspirin, 75 mg once daily
4	59	II	19.1	15	18	Bumetanide, 0.5 mg, once daily, amiodarone, 200 mg, once daily, lorazepam, 1 mg at night
5	38	III	11.2	14	—	Furosemide, 80 mg, once daily, amiloride, 10 mg, once daily, captopril, 6.25 mg, three times daily, amiodarone, 200 mg, once daily, isosorbide mononitrate, 10 mg twice daily
6	53	III	21.9	35	48	Furosemide, 40 mg, once daily, amiloride, 5 mg, once daily, temazepam at night
7	67	III	10.1	20	—	Furosemide, 120 mg, twice daily, captopril 50 mg, twice daily, insulin twice daily
8	55	III	23.0	14	—	Furosemide, 40 mg, once daily, amiloride, 5 mg, once daily, digoxin 0.25 mg, once daily, aspirin, 75 mg, once daily, allopurinol, 300 mg once daily
9	44	II	18.4	26	34	Furosemide 40 mg, once daily, prazosin, 5 mg, three times daily, methyldopa, 250 mg, twice daily, glibenclamide, 15 mg once daily

LVEF = radionuclide left ventricular ejection fraction; NYHA = New York Heart Association; VO_2 Max = maximal rate of oxygen consumption during exercise.

Methods

Study patients (Table 1). Nine men (mean age 55 years) with chronic heart failure caused by either ischemic heart disease (seven patients) or dilated cardiomyopathy (two patients) underwent 24 h ambulatory pulmonary artery pressure measurement. They had been admitted to the hospital for treatment of heart failure. All patients had previously undergone cardiac catheterization with coronary angiography and had previously performed at least two treadmill exercise tests.

Their main complaints were breathlessness and tiredness. No patient had peripheral edema, angina or evidence of myocardial ischemia at rest or during exercise. Eight patients had sinus rhythm and one (Patient 8) had chronic atrial fibrillation. Patients 2 and 5 had, respectively, undergone coronary artery surgery 1 and 2 years previously. All patients had been symptomatic for ≥3 months and had not sustained a myocardial infarction within the previous year. Patient 7 had type I and Patient 9 had type II diabetes mellitus.

All of the patients had a cardiothoracic ratio >0.55 and no pulmonary edema on a posteroanterior chest radiograph. They had a normal forced expiratory volume and vital capacity. The mean (±SD) radionuclide left ventricular ejection fraction was 20.4 ± 6.7% at rest and 28.0 ± 11.6% during exercise. The mean radionuclide right ventricular ejection fraction was 24.5 ± 6.8% at rest (no data for Patient 8) and 32.3 ± 8.9% during exercise (no data for Patients 5, 7, and 8). The mean maximal rate of oxygen (O_2) consumption on treadmill exercise was 16.1 ± 5.0 ml O_2/kg per min.

The choice of protocol for treadmill exercise was determined by the previous performance of patients on the treadmill. Those patients who were able to exercise for ≥4 min on a modified Bruce protocol were exercised using this protocol (3 min stages, starting at 1 mph and 5% gradient, then 1.7 mph and 10% gradient, 2.4 mph and 12% gradient, 3.4 mph and 14% gradient and 4.2 mph and 16% gradient). Patients with more limited exercise tolerance were exercised using the National Heart Hospital heart failure protocol (3 min stages with no gradient starting at 1 mph, then 1.5, 2 and 2.5 mph).

Pulmonary artery pressure measurements. Pulmonary artery pressure was measured with use of a 7F micromanometer-tipped catheter, incorporating an in vivo calibration system (Gaeltec Ltd., U.K.) that eliminates the error of zero drift (17). The pressure transducer signal was amplified by a portable battery-driven amplifier worn on a belt around the waist of the patient. A frequency-modulated signal was recorded using a Hewlett Packard 3964A instrumentation recorder to which the amplifier was connected by a 7 m coaxial cable. The frequency response of the amplifier was 15 Hz (−3 dB), and that of the other components of the system was in excess of this.

Before use, the catheter was sterilized in Cidex (Surgikos

Ltd., U.K.) overnight, after which it was washed and soaked in normal saline solution and temperature-stabilized for 6 min in saline solution between 37° and 39.5°C. Immediately before the catheter was inserted into a patient, it was calibrated by sealing its tip in a sterile plastic cylinder that was attached to a 239P air-operated dead weight pressure balance (Budenberg Gauge Company Ltd., U.K.) by means of a sterile tube. Static pressure calibrations were recorded on tape and read off a Fluke 75 digital voltmeter (RS Components Ltd., U.K.) to the nearest millivolt at 0, 1.5, 2.5, 5.0, 7.5 and 10 kPa (1 kPa = 7.5006 mm Hg). This calibration procedure was repeated immediately after the catheter was removed from a patient, and calibration lines were used to confirm the absence of a change in transducer sensitivity.

After catheterization, the catheter was calibrated at 20 min intervals for the first 8 h and hourly during the day thereafter. During the night, calibrations were performed every 2 to 4 h. The catheter was calibrated at rest before and immediately at the end of exercise.

An ambulatory electrocardiogram (ECG) using two bipolar leads, an anterior lead CM_5 and an inferior lead was performed simultaneously. The sites and method of application of the electrodes have been described elsewhere (18). Recordings onto magnetic tape were made with use of a frequency-modulated dual channel recorder (Oxford Medilog II, frequency response 0.05 to 40 Hz) to detect ST segment changes and arrhythmias.

Activity protocol. Patients gave written, informed consent to the procedure, which was approved by the National Heart and Chest Hospitals Ethics Committee on December 5, 1986.

For the day before catheterization and during the study, patients kept a diary of their activities. The investigators kept a separate diary of the patients' activities to ensure that adequate details were recorded and to check the accuracy of the patient's diary. The types of activity recorded were standing, sitting (with the legs down), sitting upright on a bed (with the legs horizontal), lying on a bed, eating a meal, standing while urinating, walking on a flat surface, walking up and down stairs, treadmill exercise, bicycle exercise and lying in bed at night. When recording posture during the day, no distinction was made between different activities such as talking, reading, watching television or sleeping.

On the day of catheterization, patients were fasted from 02.00 h. No premedication was administered. Catheterization was performed at 09.00 h and patients returned to their room by 09.45 h. In the catheterization laboratory, an 8F introducer was inserted into a subclavian vein under local anesthesia. The catheter was inserted through this and located in either the proximal right or left pulmonary artery under X-ray screening. The introducer was then withdrawn and the catheter stitched in place to the skin.

On return to their room, patients had breakfast, after which they went about normal daily activities. On the day of catheterization, three exercise tests were undertaken: walking up and down a flight of 18 steps at 14.30 h, walking on a flat surface in their room at 15.30 h and treadmill exercise at 16.30 h. To separate the two maximal exercise tests, bicycle exercise was performed at 09.45 h on the second day. The treadmill exercise protocol was the same as that used for determining the maximal rate of oxygen consumption already described. The bicycle exercise protocol was the same for all patients: stages were 3 min each, starting at 25 W and increasing by a 25 W increment per stage.

The treadmill and bicycle tests were maximal symptom-limited tests during which the patients were pushed to their limit. Walking on a flat surface and walking up and down stairs were not maximal tests, and the patients were instructed to exercise to the point at which they thought they would normally stop. After all exercise tests had been completed, patients were asked to rank the tests in order of the severity of their limiting symptoms, regardless of whether these were breathlessness or fatigue.

Mealtimes were determined by the hospital routine: lunch was served at 12.15 h, supper at 17.15 h and breakfast on the second and subsequent mornings at 08.00 h. All patients urinated at 13.30 h on the first day and measurements during this activity were made.

Patients received their usual medication during the study (Table 1). Once daily drugs and the first dose of other drugs were given in the morning (on the day of catheterization at 11.00 h and the second morning at 08.00 h). Patients taking diuretic drugs and nitrates twice daily received their second dose at 14.00 h. The second dose of other twice daily drugs was administered at 22.00 h. The second and third doses of three times daily drugs were administered at 14.00 h and 22.00 h.

Data analysis. Tape recordings of pulmonary artery pressure were replayed on an ink jet chart recorder (Siemens-Elema Mingograf 82). Systolic and diastolic pressures were sampled manually every 7.5 min by measuring the mean systolic and diastolic pressures over a 20 s period (10 to 15 s during exercise). Each 7.5 min pressure sample was annotated with the patient's activity as recorded in the patient's or the investigator's diary. The pressures were corrected for zero drift by linear interpolation between the in vivo calibrations.

Pressures were also sampled before exercise and at peak exercise. Before upright exercise, the control pressure reading was taken by asking the patient to stand quietly for 1 min. Bicycle exercise was preceded by 1 min of sitting on the bicycle. Heart rate was determined from the frequency of the pressure waves at the same time as the pressure was sampled.

Statistics. Systolic and diastolic pulmonary artery pressure and heart rate data were analyzed separately for individuals as well as for the whole group. For individual

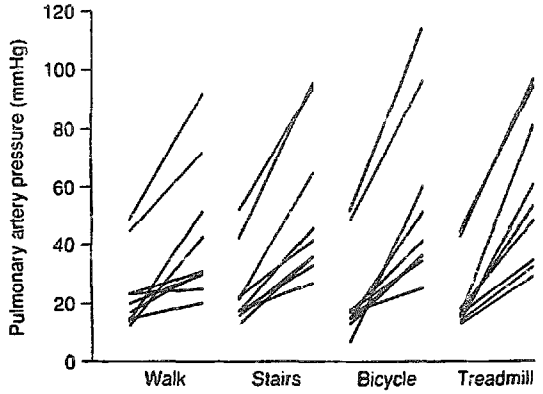


Figure 1. Systolic pulmonary artery pressure before and at peak exercise walking on a flat surface, walking up and down stairs, on the bicycle and on the treadmill in nine patients.

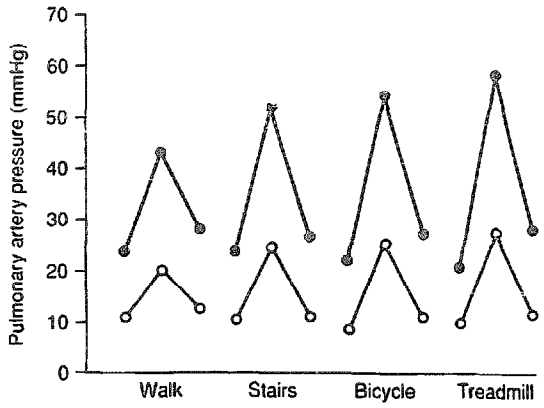


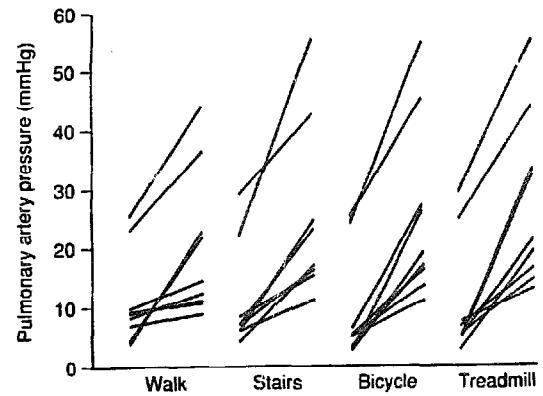
Figure 3. Mean systolic (**solid circle**) and diastolic (**open circle**) pulmonary artery pressure of all nine patients before exercise, at peak exercise and on recovery 5 min later walking on a flat surface, walking up and down stairs, on the bicycle and on the treadmill.

patients, a one way analysis of variance was used to compare the pressure values corresponding to each activity with those of all other activities. Corrections for multiple comparisons were made using Bonferroni's procedure. Pressure changes for meals were averaged for breakfast, lunch and supper. Values for each activity were also compared with nighttime, daytime and 24 h values. The daytime and 24 h values corresponded to all data points, excluding those associated with the activity under examination. For the entire study group, the maximal systolic and diastolic pulmonary artery pressures and heart rate during different exercise tests were compared using paired *t* tests and corrected by Bonferroni's procedure. Data are reported as mean values ± standard deviation.

Results

Successful continuous recording over 24 h was achieved in seven of the nine patients. Because of an electrical fault, no recording was made overnight in Patient 6. Patient 9 asked to have the catheter removed after 10 h and performed his bicycle exercise test just before this was done. There were no complications caused by the catheter or the exercise tests. Some patients had a mild ache at the site of catheter insertion, but this was always relieved by paracetamol and did not restrict their activities. Continuous recording of the ECG revealed no ST segment changes and no sustained rhythm disturbance during pulmonary artery pressure recording.

Figure 2. Diastolic pulmonary artery pressure before and at peak exercise walking on a flat surface, walking up and down stairs, on the bicycle and on the treadmill in nine patients.



Exercise tests. Patients exercised for an average of 10.3 ± 4.1 min walking on a flat surface, 5.6 ± 2.8 min on the stairs, 8.9 ± 4.5 min on the bicycle and 8.0 ± 2.1 min on the treadmill (8.1 min for Patients 1, 3, 4, 6, 8 and 9 using the modified Bruce protocol, and 7.5 min for Patients 2, 5 and 7 using the heart failure protocol). Exercise time did not correlate with the maximal absolute or percent systolic or diastolic pulmonary artery pressure increase.

Pulmonary artery pressure. There was a significant increase in the pulmonary artery pressure compared with the control measurement for all exercise tests. There was no significant difference between the mean pulmonary artery pressures at rest before each type of exercise for the entire group. The maximal pulmonary artery pressure achieved on different exercise tests varied. The increase in systolic pressure correlated with the increase in diastolic pressure ($r = 0.89$, $p < 0.001$).

The values for the systolic and diastolic pulmonary artery pressures before and at peak exercise are shown in Figures 1 and 2. There was wide variation among individuals in the preexercise and maximal exercise readings. The mean maximal pulmonary artery pressure for each of the exercise tests for all of the patients is shown in Figure 3. The mean maximal systolic pulmonary artery pressure (in mm Hg) was 59.4 ± 26.1 on treadmill exercise, 54.9 ± 30.6 on bicycle exercise, 52.5 ± 26.1 on walking up and down stairs and 43.5 ± 23.9 on walking on a flat surface. The mean maximal

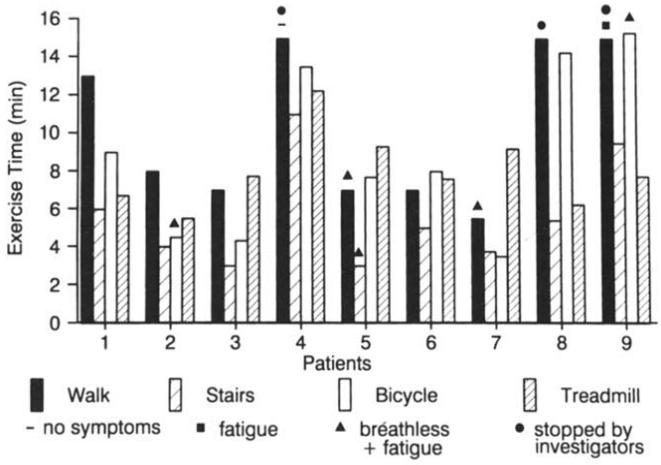
Table 2. Comparison of the Pulmonary Artery Pressure Increase During Exercise Tests Averaged for Nine Patients

Exercise Tests Compared	Systolic Pressure (mm Hg)			Diastolic Pressure (mm Hg)		
	Difference Between Mean Values of Exercise Tests	SD	p Value	Difference Between Mean Values of Exercise Tests	SD	p Value
Treadmill/bicycle	5.4	8.7	NS	1.1	5.2	NS
Treadmill/stairs	9.6	9.9	NS	3.7	6.9	NS
Treadmill/walk	18.5	9.1	0.006	8.3	3.4	0.006
Bicycle/stairs	4.3	10.2	NS	2.6	4.1	NS
Bicycle/walk	13.1	9.4	NS	7.2	3.4	0.006
Stairs/walk	8.9	7.6	NS	4.6	6.3	NS

diastolic pressure (in mm Hg) was 27.8 ± 14.6 on treadmill exercise, 25.5 ± 14.9 on bicycle exercise, 24.9 ± 14.8 on walking up and down stairs and 20.4 ± 12.5 on walking on a flat surface. The most striking feature is that walking on a flat surface caused less pressure increase than did other forms of exercise.

A comparison of the differences between the mean increase in pulmonary artery pressure for the exercise tests is shown in Table 2. All patients had a greater systolic and diastolic pressure increase on the treadmill and bicycle than when walking, although statistical significance for the group was achieved only between the systolic and diastolic pressures on the treadmill and when walking ($p < 0.006$) and between the diastolic pressure on the bicycle and when walking ($p < 0.006$). In eight patients, walking up and down stairs caused a greater increase in pressure than did walking on a flat surface. The pressure increase on the treadmill exceeded that on the bicycle in six patients and on the stairs in seven patients. In five patients, the pressure increase on the bicycle exceeded that on the stairs.

Figure 4. Exercise duration for the nine patients for each of the four exercise tests: walking on a flat surface, walking up and down stairs, on the bicycle and on the treadmill. Shortness of breath was the primary reason for stopping exercise in all of the tests except those with a symbol above the bar.



The principal symptom that limited treadmill, bicycle and stairs exercise in all patients was breathlessness (Fig. 4). However, patients complained that the most fatigue they experienced occurred during bicycle exercise, and Patients 2 and 9 considered it to be as severe as their breathlessness. On the stairs, Patient 5 rated his breathlessness equal to fatigue. Walking on a flat surface, Patient 4 was stopped by the investigators without symptoms after 15 min; Patient 8 was stopped by the investigators after 15 min and was also breathless; Patient 9 was stopped by the investigators after 15 min and was also tired; Patients 5 and 7 were stopped by breathlessness and fatigue equally and Patients 1, 2, 3, and 6 were stopped by breathlessness more than fatigue. The symptom of breathlessness did not correlate with the increase in pulmonary artery pressure or the maximal pressure during exercise.

Patients ranked the four exercise tests according to the severity of the symptoms that stopped them. Six patients ranked the bicycle and two ranked the treadmill as causing the most severe symptoms. All patients ranked walking on a flat surface as causing the mildest symptoms. Because three patients (Patients 4, 8 and 9) were stopped by the investigators after walking for 15 min on a flat surface, they might not have ranked this test as the easiest had they gone on until they would normally stop. Five patients considered the treadmill as the second easiest and six patients considered the stairs as the third easiest. Comparison of the symptom rankings with the change in pulmonary artery pressure (Fig. 5) revealed that the smallest pressure increases were associated with the mildest symptoms (rank 4) in all but Patient 5.

Neither systolic nor diastolic pressure, absolute pressure increase or percent pressure increase during treadmill exercise correlated with either the maximal rate of oxygen consumption measured on a previous treadmill exercise test using the same exercise protocol or the radionuclide ventricular ejection fraction.

Heart rate (Fig. 6). There was a significant increase in heart rate during all types of exercise. The mean heart rate (in beats/min) was 118 ± 28.2 at maximal treadmill exercise, 114 ± 24.1 at maximal bicycle exercise, 112 ± 19.4 at

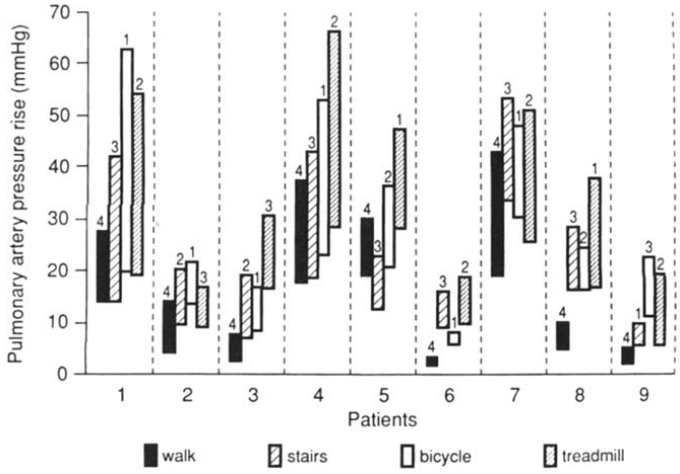


Figure 5. Maximal systolic and diastolic pulmonary artery pressure increase achieved walking on a flat surface, walking up and down stairs, on the bicycle and on the treadmill. The results are shown for the nine patients. The scale of symptoms, shown above each bar, was from 1 = worst to 4 = mildest.

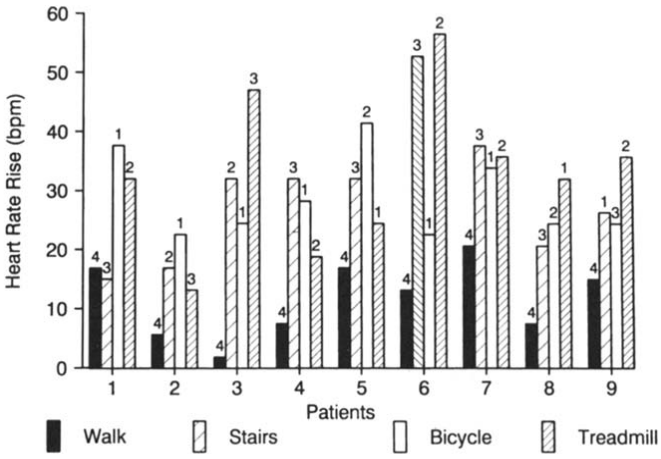


Figure 6. Heart rate response to walking on a flat surface, walking up and down stairs, on the bicycle and on the treadmill. The results are shown for the nine patients. The scale of symptoms, shown above each bar, was from 1 = worst to 4 = mildest.

maximal stairs exercise and 100 ± 21 when walking on a flat surface. A comparison of the differences between the mean heart rates during the exercise tests showed that walking on a flat surface caused significantly less increase in heart rate than did the other exercise tests ($p < 0.006$), between which there was no difference. For the entire group, there was no correlation between the change in heart rate and either systolic or diastolic pulmonary artery pressure or their absolute or percent increase during exercise.

Role of posture (Table 3). Pulmonary artery pressure altered with changes in posture in all patients. Pressure was lowest when the patient was standing still and increased with the degree of recumbency until the patient was lying flat on one pillow. The pressure difference between lying and standing ranged from 8 to 25 mm Hg for systolic pressure and from 3 to 13 mm Hg for diastolic pressure. The pressure when lying in bed was not significantly different from that achieved during upright exercise walking up and down stairs.

Nighttime versus daytime measurements (Table 4). In six of the seven patients with nocturnal recordings, there was an increase in systolic and diastolic pulmonary artery pressures at night. Statistical significance by analysis of variance ($p < 0.02$) was achieved in all but Patient 7. In two of the patients (Patients 3 and 5), there was no difference between the nocturnal pressure and that during lying during the day. In Patient 2, the nocturnal pressure was the same as that during sitting on the bed during the day. In Patient 4, the pressure at night exceeded that during lying down during the day. When the patients got out of bed in the morning, pulmonary artery pressure immediately decreased. The lack of pressure difference in Patient 7 may have been caused by the patient's

Table 3. Effect of Postural Changes on Systolic and Diastolic Pulmonary Artery Pressure (mm Hg) in Nine Patients

Patient No.	Standing				Sitting				Sitting on Bed				Lying			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	38.9	11.5	20.8	6.8	45.0	5.4	24.7	3.2	53.8	2.7	30.7	1.1	56.7	3.2	32.0	3.1
2	14.8	2.2	6.1	1.8	16.3	2.5	6.9	1.7	18.5	1.1	8.8	1.1	25.6	2.2	13.5	2.2
3	17.3	3.7	5.1	1.9	18.9	2.7	7.0	2.2	27.9	4.1	13.2	2.7	27.2	1.8	14.3	0.9
4	13.9	6.4	3.5	2.4	25.7	8.4	8.9	4.6	30.2	6.3	12.7	3.6	38.6	7.9	16.7	3.8
5	15.7	4.6	6.3	3.1	20.8	3.2	9.4	2.4	30.2	4.6	16.9	3.7	36.4	9.2	20.4	7.3
6	17.6	2.5	6.9	1.8	19.4	2.7	8.1	2.0	22.6	3.1	9.7	2.6	29.2	2.9	12.3	1.4
7	43.8	9.0	24.1	5.5	48.2	9.1	25.4	5.1					51.8	7.0	27.3	3.2
8	13.3	4.0	3.8	2.9	19.0	3.6	6.2	2.3	28.3	2.1	13.3	1.6	31.5	2.3	16.4	1.9
9	15.2	1.9	5.9	1.3	15.0	1.9	6.1	1.0					26.6	3.6	13.0	1.9

Lying = lying down with one pillow; Sitting = sitting in a chair with legs dependent; Sitting on bed = sitting up on a bed with the legs horizontal; Standing = standing still. Patients 7 and 9 did not sit on their bed during the day.

Table 4. Mean Systolic and Diastolic Pulmonary Artery Pressure (mm Hg) During the Day and at Night in Nine Patients

Patient No.	Systolic Pulmonary Artery Pressure				Diastolic Pulmonary Artery Pressure			
	Day		Night		Day		Night	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	47.2	11.3	44.8	4.4	25.5	5.4	24.8	3.3
2	20.0	4.7	21.9	3.5	8.6	3.0	11.2	2.8
3	24.5	6.4	27.1	4.7	10.8	4.4	13.6	2.9
4	29.5	11.4	43.0	7.0	11.5	6.0	21.9	4.1
5	28.5	10.3	37.2	4.2	14.9	7.1	21.3	3.0
6	23.1	5.1			9.7	2.9		
7	51.2	11.7	53.2	5.9	27.1	6.8	29.3	2.9
8	20.9	7.0	36.0	4.1	7.9	4.4	18.9	3.3
9	21.2	6.6			9.6	2.9		

Patients 2 and 10 did not have nocturnal pressure measurements.

spending more than half of the night sitting on the edge of his bed.

Effects of meals. Pulmonary artery pressure increased significantly in three of nine patients (Patients 1, 3 and 6) by 4.5 mm Hg systolic and 2.0 mm Hg diastolic during meals compared with that when sitting (the posture during eating). The pressure increase was never as much as that achieved by lying flat. Heart rate during meals did not increase significantly.

Urination. The increase in pulmonary artery pressure during urination was not significantly different from that when walking on a flat surface in eight of the nine patients. It was always greater than that when standing. The increase in pressure could be accounted for by the short walk that patients had to make to the toilet. In Patient 5, pressure increased but was less than when walking on a flat surface. A significant increase in heart rate during urination over that when walking on a flat surface of 18 beats/min occurred only in Patient 9.

Discussion

The micromanometer-tipped intrapulmonary catheter. We used a micromanometer-tipped catheter to measure pressure in the pulmonary arteries accurately and continuously. As used in this study, the catheter overcomes the problems of fluid-filled catheters, especially the errors in leveling an external transducer to a zero reference point, temperature sensitivity and electrical zero drift. The tip of the catheter itself is the zero reference for pulmonary artery pressure. When there are changes in body position or when respiratory changes in intrathoracic pressure become large, as occurs during exercise, leveling an external transducer to the level of the left atrium by an arbitrary reference point on the body can lead to gross inaccuracies in the estimation of left atrial and pulmonary artery pressure. Fluid-filled catheters may also generate artifacts such as catheter impact and end pressure (19,20), in addition to those caused by threading the catheter through the beating heart (21).

Our results show wide variation in pulmonary artery pressure among patients during the day and at night and with different postures and different exercise tests. These differences may be explained by the heterogeneous nature of the disease under scrutiny, interindividual variation and the effects of drug treatment. For these reasons, the analysis of data was concentrated on modeling the behavior of individual patients, as well as summarizing the data for the group as a whole.

Effects of exercise on pulmonary artery pressure in the normal subject. The order of the exercise tests was not randomized because it was necessary to perform the two maximal exercise tests on different days to circumvent excessive exhaustion. For the same reason, the treadmill test was performed at the end of the day after the two submaximal exercise tests.

In normal subjects, the magnitude of the increase in pulmonary artery pressure during exercise shows wide variations and depends partly on the severity of exercise. The behavior of the normal pulmonary circulation may be mainly passive, the elevation of pressure during exercise being attributable to increased cardiac output (22). Moderate exercise may not be associated with an increase in pulmonary artery pressure (23,24). Severe upright bicycle exercise in normal subjects caused the pulmonary artery pressure to double from 13.8/5.2 to 26.8/10.3 mm Hg (25), whereas in athletes, it increased from 19.1/9.1 to 46.3/13.1 mm Hg, possibly because their central blood volume was larger. Pulmonary wedge pressures during seated bicycle exercise have been shown to increase from 13 to 22 mm Hg (26) and pressures up to 25 (27) and 30 mm Hg (28) during exercise have been recorded.

Effects of different types of exercise in patients with heart failure. In our patients, different exercise tests caused varying increases in pressure. In part, this variation may

have been caused by a relative hypovolemia due to diuretic drugs or vasodilators. Before exercise, seven of the nine patients had normal pulmonary artery pressure at rest. Their incremental increase in pulmonary artery diastolic pressure at peak treadmill exercise was similar to previous observations (4) in patients with heart failure and was normal for intense exercise. The two patients whose pulmonary artery pressure exceeded normal exercise values had a higher pressure at rest compared with normal subjects. Although on average the treadmill generated the greatest increase in pressure, in three of the nine patients the bicycle exercise test and in one of the nine the stairs caused the greatest pressure increase.

Walking on a flat surface and up and down stairs simulated everyday exercise. The patients set their own pace and stopped without encouragement to go further. During the treadmill and bicycle tests, patients were strongly encouraged to exercise for as long as possible. In eight of the nine patients, there was less of an increase in pulmonary artery pressure and milder symptoms walking on a flat surface than during more severe exercise. During exercise, pulmonary artery pressure was not a function of either the duration of exercise or the heart rate. There may be several explanations for the difference between the pressure increase during different types of exercise. First, the patients may have put a different amount of effort into the various exercise tests. Second, the types of exercise undertaken were different. Stair climbing has an isometric component and has been shown (29) to constitute strenuous anaerobic exercise in moderate to moderately severe heart failure. Third, it has been proposed that diurnal variation in pulmonary artery pressure may occur (30), and we (31) have previously observed this in some patients with chronic heart failure. It was characterized by a significant increase in pulmonary artery pressure at night, although our limited data did not show diurnal variation over short periods of time during the day.

Relation between symptoms during exercise and pulmonary pressures. Patient rankings of the severity of their limiting symptoms during exercise were not related to the pressure increase except for walking on a flat surface. Such a method of symptom assessment has shortcomings because the limiting symptoms for different types of exercise were not exactly the same. If symptoms that limited exercise were related to the maximal pulmonary artery pressure during exercise, it would be expected that in individual patients, the increase in pressure would correlate with the symptom rank. We only observed this for the walk on a flat surface.

The association between the mildest symptoms and the least increase in pressure does not necessarily mean that the pulmonary artery pressure increase is the cause of symptoms. There is accumulating evidence that the main cause of exertional breathlessness limiting exercise in chronic heart failure is an increase in pulmonary physiologic dead space (9,32) caused by ventilation-perfusion mismatch (12). The reason for the ventilation-perfusion mismatch has not been identified. Nevertheless, pulmonary artery pressure is an important determinant of perfusion (33) and it may be one of the factors contributing to the increase in physiologic dead space. Alternative mechanisms for breathlessness on exertion include increased muscle lactate production causing a disproportionate increase in ventilation (4,32,34) and skeletal muscle acidosis (10).

Role of posture and urination. Although pulmonary blood volume decreases by 33% from lying down to standing up in normal subjects (35), postural changes in pulmonary artery pressure are small (36,37) in contrast with that in our patients with heart failure. The changes in pulmonary artery pressure at night are consistent with alterations in pressure caused by posture, although other mechanisms may have been responsible. Urination was chosen as representative of a mild daily activity and, in accord with the small amount of exercise required, caused a small increase in pulmonary artery pressure and heart rate.

Role of food ingestion. During ingestion of food, three of the nine patients showed a small increase in pressure but no increase in heart rate. Food ingestion causes generalized sympathomimetic stimulation (38), in the absence of which systemic hypotension may occur (39,40). Gastric distension itself may elevate left ventricular end-diastolic pressure (41). The findings in six of our patients are in agreement with the lack of change in pulmonary wedge pressure and mean pulmonary artery pressure during food ingestion and digestion in patients with heart failure reported by others (30,42). The small difference in the hemodynamic response of the three patients whose pressure increased may reflect different changes in their cardiac output or splanchnic blood flow during meals from those of the other patients. In turn, these may be related to the reduced hunger, premature fullness and satiety, reduced gastric capacity and retarded gastric emptying, which have been observed in patients with heart failure (43). There was no consistent postprandial change in pulmonary artery pressure within individuals. This may be explained by variations in their postprandial posture and activities, emphasizing the unimportance of the effect of meals on pulmonary artery pressure during normal life.

Conclusions. Continuous ambulatory pulmonary artery pressure recording in patients with chronic heart failure is safe and well tolerated. Exercise on a bicycle, treadmill and stairs caused large and varied pulmonary artery pressure increases that were greater than those during other activities. The pressure increase and limiting symptoms when walking on a flat surface were less than with other forms of exercise. Eating meals and urination caused relatively minor alterations in the pressure. The symptom of breathlessness did not correlate with the changes in pulmonary artery pressure and the latter was unrelated to heart rate during exercise.

Thus, symptoms and changes in pulmonary artery pressure during maximal exercise are not the same as those during everyday activities. These differences may limit the value of maximal exercise tests in assessing patients with heart failure and the effects of therapy.

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