fusion was present on both sides. Pleuracentesis yielded 2200 ml of clear fluid without tumor cells. Transthoracic echocardiography could exclude a large pericardial effusion and showed hypertrophied ventricles but was otherwise nondiagnostic because of the patient's obesity and respirator therapy. Transesophageal echocardiography with a 5 MHz monoplane probe revealed severe thickening of both left and right ventricular wall measuring up to 48 mm. The myocardial structure appeared very heterogeneous, with irregular areas of low and high echodensity (Fig. 1, *A*). Less echogenic intramyocardial nodules bulged the epicardial and the endocardial surface and protruded from the interventricular septum into the left ventricular outflow tract without causing hemodynamically significant obstruction. The interatrial septum was replaced by a large tumor mass obstructing the coronary sinus and the right upper pulmonary vein and extending into the surrounding tissue (Fig. 1, *B*). The valves appeared normal, and there was only a small pericardial effusion. Transesophageal echocardiography was interpreted as showing extensive myocardial tumor infiltration. For histologic confirmation a transvenous biopsy of the interatrial mass was considered, but it was believed to be less invasive to excise a small inguinal lymph node. Histologic analysis showed metastatic malignant melanoma. The patient developed protrusion of the right eye and generalized seizures. Cerebral computed tomography disclosed a retrobulbar mass consistent with a tumor metastasis and a left cerebral infarction. The patient died 10 days after admission with intractable heart failure. At autopsy there was 50 ml of pericardial fluid, and the heart with its metastases weighed 2450 gm. Black tumor nodules extended through the entire wall of each cardiac chamber, almost completely replacing the myocardium (Fig. 2). Massive tumor infiltration, especially of the atrial and ventricular septum, extended into the atrioventricular sulcus. In addition, small pigmented metastases were present in the lungs, lymph nodes, kidneys, adrenal glands, pancreas, gastrointestinal tract, subcutaneous tissue, and the right retrobulbar region. The tumor burden in these organs, however, appeared small compared to the excessive tumor infiltration of the heart.

Most malignant melanoma metastases are located in the myocardium as a result of hematogenous tumor spread.[1, 2] Despite the great propensity for heart involvement, cardiac dysfunction is rarely the cause of death. Although there have been reports of extensive cardiac infiltration by melanoma with a heart weight of up to 880 gm,[2] this case is remarkable for its excessive tumor infiltration almost completely replacing the myocardium of each cardiac chamber, resulting in death from intractable heart failure. The heart weight of 2450 gm is the highest figure reported so far in literature. Transthoracic echocardiography has been the technique of choice for the diagnosis of intracavitary tumor growth. For evaluation of an extracavitary origin or extension of the tumor, however, magnetic resonance imaging proved to be highly superior.[4-6] In this patient transesophageal echocardiography was diagnostic of metastatic heart disease. The use of a higher transducer frequency with this method greatly increased image resolution and allowed differentiation of myocardial tumor infiltration within the severely thickened ventricular walls as a result of different acoustic properties of the tumor tissue. The superior diagnostic potential of transesophageal compared to transthoracic echocardiography for intracavitary and paracardiac mass lesions[7] in this case also holds true for mainly myocardial tumor involvement. Because it is a relatively inexpensive technique that can rapidly be performed at the patient's bedside, transesophageal echocardiography is recommended as the primary diagnostic tool in patients with suspected myocardial metastases.

REFERENCES

1. Young JM, Goldman IR. Tumor metastasis to the heart. Circulation 1954;9:220-9.
2. Glancy DL, Roberts WC. The heart in malignant melanoma. A study of 70 autopsy cases. Am J Cardiol 1968;21:555-71.
3. Kutalek SP, Panidis IP, Kotler MN, Mintz GS, Carver J, Ross JJ. Metastatic tumors of the heart detected by two-dimensional echocardiography. AM HEART J 1985;109:343-8.
4. Emmot WW, Vacek JL, Agee K, Moran J, Dunn MI. Metastatic malignant melanoma presenting as obstruction of the right ventricular inflow and outflow tracts. Characterization by magnetic resonance imaging. Chest 1987;92:362-4.
5. Freedberg RS, Kronzon I, Rumancik WM, Liebeskind D. The contribution of magnetic resonance imaging to the evaluation of intracardiac tumors diagnosed by echocardiography. Circulation 1988;77:96-103.
6. Vetto JT, Heelan RT, Burt M. Malignant melanoma metastatic to the right atrium: an asymptomatic solitary metastasis diagnosed incidentally by magnetic resonance imaging. J Thorac Cardiovasc Surg 1992;104:853-4.
7. Mügge A, Daniel WG, Haverich A, Lichtlen PR. Diagnosis of noninfective cardiac mass lesions by two-dimensional echocardiography. Comparison of the transthoracic and transesophageal approaches. Circulation 1991;83:70-8.

Comparison of submaximal treadmill and supine bicycle exercise

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Postinfarction exercise testing is frequently used to evaluate risk for inducible ischemia, and may involve either treadmill or bicycle exercise. Exercise in this clinical situation is frequently submaximal,[1] with either a target heart rate or workload used as a clinical parameter for termination. In the following study we compared the physiologic

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Table I. Treadmill supine bicycle exercise at 70% PMHR and 85% PMHR

	Treadmill	*Bicycle*	*p Value*
70% PMHR			
Oxygen consumption (ml/kg/min)	24.8 ± 2.2	22.7 ± 1.7	NS
Systolic blood pressure (mm Hg)	152 ± 4.9	188 ± 6.8	<0.001
Rate pressure product	20,100 ± 700	25,400 ± 1,000	<0.001
85% PMHR			
Oxygen consumption (ml/kg/min)	36 ± 2.7	33 ± 2.1	<0.05
Systolic blood pressure (mm Hg)	172 ± 4.7	208 ± 6.2	<0.001
Rate pressure product	27,300 ± 800	33,600 ± 900	<0.001
Lactate 5 min after exercise (mmol/L)	2.9 ± .4	5.3 ± .6	<0.001

NS, Not significant.

stress associated with bicycle and treadmill exercise during the degrees of submaximal exercise often used to evaluate postmyocardial infarction patients.

Methods. The study group consisted of 10 healthy male volunteers with a mean age of 30.3 years. No subject was taking medication. Each subject performed treadmill and supine bicycle exercise to 85% predicted maximal heart rate (PMHR; 220 minus age). The order of the two tests was alternated, and tests were performed on separate days within a 5-day period. Supine bicycle exercise was performed on a Collins cycle ergometer. The bicycle exercise protocol involved initial exercise at 25 W/min, with a 20 W increment each minute. The treadmill protocol involved 1 minute stages at the following settings: (1) grade 0,2 mph; (2) grade 0,3 mph; (3) grade 0,4 mph; (4) with all remaining stages at 4 mph and an increase of grade by 2% per stage. The electrocardiogram of each volunteer was monitored continuously during exercise and recovery. Blood pressure was assessed by auscultation every 2 minutes, and at 70% PMHR and at 85% PMHR. Ventilation and gas exchange was measured continuously during exercise with a SensorMedics metabolic measurement cart, and venous lactate was measured after 5 minutes of recovery. Values were compared by using paired *t* tests. Group values are reported as mean ± SEM.

Results. The mean heart rate at 70% PMHR was 133 ± 1 beats/min; the mean heart rate at 85% PMHR was 161 ± 1 beats/min. The mean systemic oxygen consumption during treadmill exercise at 85% PMHR was significantly higher than during bicycle exercise, and venous lactate after treadmill exercise was significantly lower than after bicycle exercise (Table I). Bicycle exercise resulted in significantly higher systolic blood pressure and rate pressure product at 70% PMHR and 85% PMHR than treadmill exercise.

Comments. The data show that at comparable submaximal heart rates, treadmill exercise in normal subjects results in significantly lower cardiovascular stress than supine bicycle exercise. No prior studies compared supine bicycle exercise to treadmill exercise at similar submaximal heart rates although, similar to our results, the results of Nieberber et al.[2] showed that at equivalent submaximal exercise levels patients with coronary disease had higher degrees of cardiovascular stress during upright bicycle exercise than during treadmill exercise. That different exercise modalities result in significantly different degrees of cardiovascular stress at submaximal workloads may be an important consideration when risk is assessed for inducible ischemia after myocardial infarction. Several randomized trials have shown that there is no advantage to aggressive therapy (i.e., routine angiography followed by prophylactic angioplasty after thrombolysis) compared to a conservative strategy (i.e., angiography restricted to patients with either spontaneous or inducible ischemia after thrombolytic therapy),[3-5] but the methods used to assess inducible ischemia in these trials vary; the TIMI (Thrombolysis in Myocardial Infarction) 2 trial, ischemia was assessed by performing supine bicycle exercise to a maximal heart rate of 120 beats/min or a workload of 67 W,[3] and in the Treatment of Post-Thrombolytic Stenosis (TOPS) trial a variety of modalities were used.[4] In clinical practice, risk stratification is most often accomplished by using treadmill exercise.[1] Generalizing the results of a trial such as TIMI 2 to patients undergoing treadmill exercise depends on both modalities creating similar degrees of myocardial stress. However, our study shows that exercise modality plays in important role in the degree of myocardial stress elicited at a particular submaximal workload.

The clinical implications of this study with regard to assessing risk for ischemia after myocardial infarction are limited by the study's use of normal subjects. However, a recent study of patients with coronary artery disease compared hemodynamic and electrocardiographic response to treadmill and supine bicycle exercise also showed that submaximal supine bicycle exercise resulted in significantly higher systolic blood pressure and double product than treadmill exercise.[6] In addition, supine bicycle exercise was determined to be significantly more sensitive for detecting coronary disease than treadmill exercise. Further investigation is needed to determine how submaximal exercise on different modalities affects the assessment of inducible ischemia after myocardial infarction. In conclusion, this report demonstrates that supine bicycle exercise results in significantly higher rate pressure products than treadmill exercise at submaximal heart rates commonly used in postmyocardial infarction risk stratification. The

results have implications for generalizing studies that use bicycle exercise to evaluate inducible ischemia to clinical practice, where such testing is usually performed on a treadmill.

REFERENCES

1. Hamm LF, Crow RS, Stiull GA, Hanan P. Safety and characteristics of exercise testing early after acute myocardial infarction. Am J Cardiol 1989;63:1193-7.
2. Niederberger M, Bruce RA, Kusumi F, Whitkanack S. Disparities in ventilatory and circulatory responses to bicycle and treadmill exercise. Br Heart J 1974;36:377-82.
3. TIMI Study Group. Comparison of invasive and conservative strategies after treatment with intravenous tissue plasminogen activator in acute myocardial infarction. N Engl J Med 1989;320:618-27.
4. Ellis SG, Mooney MR, George BS. Randomized trial of late elective angioplasty versus conservative management for patients with residual stenoses after thrombolytic treatment of myocardial infarction. Circulation 1992;86:1400-6.
5. SWIFT Trial Study Group. SWIFT trial of delayed elective intervention v conservative treatment after thrombolysis with anistreplase in acute myocardial infarction. BMJ 1991;302: 555-60.
6. Wetherbee JN, Bamrah VS, Ptacin MJ, Kalbfleisch JH. Comparison of ST segment depression in upright treadmill and supine bicycle exercise testing. J Am Coll Cardiol 1988;11: 330-7.