Appendix on

Certificate Course in Coronary Artery Disease and Cardiac Rehabilitation 96

Department of Medicine, University of Hong Kong
(Queen Mary Hospital, Grantham Hospital, Tung Wah Hospital)
Princess Margaret Hospital
Hospital Authority
The Hong Kong Society for Rehabilitation -
Community Rehabilitation Network

Content

10. Putting the Risk of Exercise into Perspective **Dr. Barry A. Franklin**
(Franklin B.A., Putting the " Risk of Exercise " in Perspective. Certified News, 4:1-4)

11. Reverting Heart Disease with Diet, Drugs and Exercise **Dr. Barry A. Franklin**

12. Physical Activity Workshop **Dr. Barry A. Franklin**
(Franklin B.A., Innovative Activities for Adult Fitness and Cardiac Exercise Programs: A " Hands-on " Session

Further References

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Risk Stratification:Scores to Predict Severity and Prognosis in Coronary Artery Disease

Vic Froelicher, MD
Symposium 1:
Current Trends
4:30 - 5:30 pm

Stable CAD

Who to Cath ?

- Quality of Life
  - Limitations to activities
  - Medication side effects
- Quantity of Life
  - Clinical Scores to Estimate CV annual mortality and probability of severe CAD
  - Angiographic Subsets

Prognosis

- Congestive Heart Failure
  - 10 to 25% Annual Cardiac Mortality
- Myocardial Infarction
  - Complicated: Shock, CHF, Ischemia...
  - 10% prior/10% in-hospital/10%1st year
- Angina Pectoris
  - Stable: 2% Annual Cardiac Mortality
  - Unstable: 4%

Meta Analysis of Prognosis Post MI

Exercise Test and Follow-Up (N=28)

CHF (Excluded from Test)	**2x**
Resting ST Depression	**3X**
Poor Exercise Capacity	**14/18***
Exercise ST Depression	**15/24**
(Non-Qwave only Studies)	**2/2***
Exercise SBP	**13/18***
Angina	**12/20**
PVCs (frequent)	**14/23**

Prognosis

Meta Analysis of Prognosis in Stable CAD

Exercise Test and Cath (N=9)

Poor Exercise Capacity	6/9
CHF	3/9
ST Depression	
Resting	2/9
Exercise	3/9
Exercise SBP	3/9

Work-up Bias

- In All Studies that Require the Patients to have a Cardiac Catheterization, the Patients are already selected by their physicians for the Cardiac Catheterization Using Clinical and Test Variables
- For the Selection of Variables from a Study to be Applicable to the Patient Presenting for a Work-Up, the Study must be Performed on an Un-selected Population
- Work-Up Bias can Only be Avoided by having the Patient Consent to Catheterization no Matter what the Test Results Are

Clinical Scores

- Survival Analysis
  - Based on Follow-up and Censoring
  - Cox Hazard Function
  - Weighted Coefficents used to construct Equations for Scores and Nomogram
- Probability of Severe Disease
  - Based on Angiography
  - Multiple Logistic Regression
  - Coded Variables x Coefficents added then solved in Natural Log Equation to fit a Sigmoid Curve

Prognosis

Prognostic Scores

- DUKE SCORE
  METs - 5x[mm E-I ST Depression] - 4x[TM Angina Index]
  ***see nomogram
- VA SCORE
  5x[CHF/Dig] + [mm E-I ST Depression] + change in SBP score - METs

E-I = Exercise Induced

Multiple Logistic Regression Equations to Predict Severe CAD

Christian, et al, Ann Intern Med 121:825-832, 1994: age, gender, symptoms, diabetes, peak double product and amount of ST depression.

Detrano, et al, Comp & Biomed Res 25:468-485, 1992: age, gender, symptoms, history of MI or Q waves, METs, peak HR, exercise induced angina, exercise induced hypotension, ST slope and amount of ST segement depression.

Morise, et al, JACC 20:1187-96, 1992: age, gender, symptoms, diabetes, hypertension, cholesterol, obesity, current cigarette use, estrogen, change in systolic blood pressure, ST slope, amount of ST segment depression and negative ST (< 1.5mm upsloping or < 1mm horizontal or downsloping).

Froelicher/Do (1995): age, cholesterol, LVH with strain, change in systolic blood pressure, peak heart rate, ST slope and amount of ST segment depression.

Problems with Prediction Equations

- Misclassification
- Follow-up Confounded by Interventions
- Work-Up Bias
- Skepticism that Simple Variables Can Be Better Than Imaging Technologies
- Differences between Studies as to Variables and Their Coding
- Requires Nomograms or Computers to Calculate

CONCLUSIONS

*ST segments exhibited abnormal depression during exercise and abnormal depression in recovery. (Abnormal ST response)
*The systolic blood pressure response is normal (27 mmHg increase).
*The patient achieved 164% of normal exercise capacity for age, and 110% of normal maximal heart rate for age.
*The patient has a moderate probability of having any clinically significant coronary artery disease and low probability of having severe coronary artery disease.
*Estimated prognosis from treadmill scores is as expected for age, gender and race.

PROGNOSTIC ADDENDUM

*The age expected annual mortality from any cause is 6.5% (National Center for Health Statistics, 1990).
*The Framingham score (Age, Cholesterol, Diabetes, Smoking, LVH) estimates a five year incidence of cardiovascular events (Angina, MI or Death) of 14%.
*The Froelicher score (METs, CHF, SBP rise, and ST depression) estimates an annual cardiovascular mortality of 1.3% (not greater than two times the age expected mortality).
*The Duke Score (METs, ST depression, and treadmill angina) estimates an annual cardiovascular mortality of 1.2% (not greater than two times the age expected mortality).
*The estimated operative mortalities for bypass surgery are 16% (Parsonnet, 1989), 3% (NY State Dept. of Health, 1992) and 1% (VA, 1993).
*The posttest probability for any clinically significant coronary artery disease are 73% (Detrano, 1992), 85% (Morise, 1994) and 32% (Do/Froelicher, 1995).
*The probabilities of having severe coronary artery disease are 18% (Detrano, 1992), 51% (Morise, 1992), 11% (Do/Froelicher, 1995) and 6% (Christian, 1994).

Disclaimer: This Report was computer generated and the results are dependent on rules and correct data entry It must be overread by a physician.

CONCLUSIONS

Simple Clinical and Exercise Scores can be used to Decide which Patients Need Interventions in order to improve their Prognosis.

These Scores could frequently obviate the need for Cardiac Catheterization and Decide which Tests are Appropriate.

The Consensus Approach Appears to Make the Scores Portable and Robust as Well as Provide Excellent Test Characteristics

Risk Stratification of Stable CAD using Clinical and Exercise Test Variables

V. Froelicher, MD
Palo Alto VAMC
Stanford University

Prognosis

Basic Pathophysiologic Features of CAD that Determine Prognosis

- Amount of remaining Myocardium (scar, hypertrophy)
- Perfused viable Myocardium (functioning, collaterals, area in jeopardy)
- Arrhythmic risk

Meta Analysis of Prognosis Post MI

Exercise Test and Follow-Up (N=28)

CHF (Excluded from Test)	2x
Resting ST Depression	3X
Poor Exercise Capacity	14/18*
Exercise ST Depression	15/24
(Non-Qwave only Studies)	2/2*
Exercise SBP	13/18*
Angina	12/20
PVCs (frequent)	14/23

Risk Stratification of Stable CAD

- **Considering:**
  - Clinical Features
  - Exercise Test
  - Cardiac Catheterization

(if the first two accurate enough, then Cath not necessary)

Prognosis

Meta Analysis of Prognosis in Stable CAD

Exercise Test and Cath (N=9)

Poor Exercise Capacity	6/9
CHF	3/9
ST Depression	
Resting	2/9
Exercise	3/9
Exercise SBP	3/9

Survival Analysis

- Time to End Point
- Reliability of Machines
- Censoring - Key Difference

"...associated with time till CV Death"

Censoring (Random)

- Lost to follow-up
- Drop Out
- Termination of Study
- Intervention

Survival Analysis

- Multivariate Analysis
  - Variables with Univariate Risk (Kaplan-Meier) can be associated with CV Death through others (e.g., Digoxin, ST elevation)
  - So Multivariate Analysis preferred (e.g., Cox Hazard function)

End Points

- **Hard**
  - Death (Easiest)
  - CV Death (Best)
  - Type CV Death
  - MI (difficult to Predict)
- **Soft**
  - Interventions (Worst)
  - Angina, Unstable
  - Abnormal Tests

CV Death Spectrum

- Ischemic Death with good Heart Muscle (late)
- CHF Death with Damaged Myocardium (early)
  - Final event often Arrythmia but secondary

Ischemic Death Markers

- **Angina** (Pre-Test Symptoms and Impact during Test)
- **ST Depression** - Rest (?) and Exercise-Induced

CHF Death Markers

- CHF signs/symptoms
- Q waves, Bundle Branch Block
- History of MI
- LVH with strain
- ST elevation over Q waves

Ischemic and/or CHF Markers

- Maximal Exercise Systolic Blood Pressure and Heart Rate
- Exercise Capacity
  - Since these are interrelated, and associated with both ends of the CV death spectrum, they are consistent and powerful.

Prognosis

- Ischemic Markers are associated with a later Time for and lesser Risk of Death
- LV Dysfunction Markers with a sooner Time for and greater Risk of Death

Why Don't the Studies Agree?

- **Misclassification**
  - Variables (e.g., CHF, MI)
  - Endpoints (CV death)
- **Censoring**
  - Interventions not random
- **Work-up Bias**
  - Patients selected for Cardiac catheterization

Work-up Bias

- In All Studies that Require the Patients to have a Cardiac Catheterization, the Patients are already selected by their physicians for the Cardiac Catheterization Using Clinical and Test Variables
- For the Selection of Variables from a Study to be Applicable to the Patient Presenting for a Work-Up, the Study must be Performed on an Un-selected Population
- Work-Up Bias can Only be Avoided by having the Patient Consent to Catheterization no Matter what the Test Results Are

Effect Of Work-Up Bias on Survival Analysis: the Long Beach Study

	Selected For CATH	Unselected For CATH
Number	588	3134
Avg Annual CV Mortality	2.6%	1.5%
CABS in FU	20%	2%
Variables Chosen	CHF, Exertional Hypotension, Resting STd	CHF/Dig, ExSBP, METs, and EI-STd

Are Natural History Studies Valid?

- Intervention Rates about 25% over 5 years if Cath Population; 2% if Non-selected Population
- Censoring removes Patients with Interventions from the Numerator and the Denominator
- Less than 50% of CABS and 10% of Cath Population Patients have 3 VD with EF<50%
- Low to Moderate Risk Patients are High Percentage of those selected for Cardiac Cath or Interventions
  - **For Cath, 50% low risk by Score, 90% low or moderate risk by Cath Anatomy**
  - **For Interventions, 65% by Score and 66% by Cath Anatomy**

Intervention Rates in FU Studies

Study	Years FU	% CABS
CASS	5	36
Italian	5.5	15
Belgian	5	29
Belgian(noMI)	5	28
Duke	5	24
VA CABS Trial	4	24
LongBeachVA	5	20/2

Prognostic Scores

- DUKE SCORE
  METs - 5*[mm E-I ST Depression] - 4*[TM Angina Index]
- VA SCORE
  5*[CHF/Dig] + [mm E-I ST Depression] + change in SBP score - METs
  E-I = Exercise Induced

Simple Clinical and Exercise Scores can be used to Decide which Patients Need Interventions in order to improve their Prognosis.

These Scores could frequently obviate the need for Cardiac Catheterization.

Conclusions (#1) for Prognosis in Stable CAD

Patients can be given relative estimates of their annual cardiovascular Mortality with Medical versus Surgical therapy.

This can be done using Prognostic Scores and Coronary Artery Bypass Surgery Case Mix Data (Parsonnet, 1989)

Conclusions (#2) for Prognosis for Stable CAD

Quality of life issues cannot be resolved with Scores.

These issues require an understanding Physician and an informed Patient.

Conclusions (#3) for Prognosis in Stable CAD

In general, Physicians over-estimate the Danger of Ischemia.
Perhaps if given accurate Mortality Estimates, they would practice less Invasively.
Scores will Lead to Appropriate Care rather than Limit Access to Care

Conclusions (#4) for Prognosis in Stable CAD

CHANGING PARADIGMS AND PERCEPTIONS IN HEART DISEASE AND REHABILITATION

Certificate Course in
Coronary Artery Disease
and Cardiac Rehabilitation 96
February 3-7, 1996
Barry A. Franklin, Ph.D.

Outline

I. Introduction
   A. What Do You See?

II. Bed Rest/Deconditioning
   A. Deleterious Effects
      1. Significance of orthostatic stress

III. Contemporary Treatment of the Cardiac Patient
   A. Thrombolytic Therapy
   B. Shortened Hospital Stay
   C. Stress Testing
   D. Risk Stratification
      1. Influence on return-to-work
   E. Patient Eligibility for Cardiac Rehabilitation
      1. Contraindications to exercise therapy(?)
         a. Myocardial ischemia
         b. Large anterior wall myocardial infarction
   F. Risk of Exercise Therapy

IV. Risk Factor Modification
   A. Cholesterol
   B. Smoking Cessation
   C. Hypertension
   D. Physical Activity
      1. Myths and misconceptions
      2. Risk

V. Psychosocial Considerations
   A. Type "A" Personality
      1. Decreasing significance in secondary prevention
   B. Prognostic Value of "Social Isolation"
      1. Roseto Pennsylvania study
   C. Anger/Hostility

VI. Secondary Prevention: Morbidity/Mortality
   A. Meta-Analyses
      1. Oldridge Study
      2. O'Connor Study
   B. Possible Mechanisms of Benefit

VII. How Much Exercise Is Enough?
   A. Blair Data
   B. Blumenthal Study
      1. Significance of mild-to-moderate training intensities

VIII. Future Directions
   A. Ornish Philosophy
      1. Patient responsibility

IX. Conclusions

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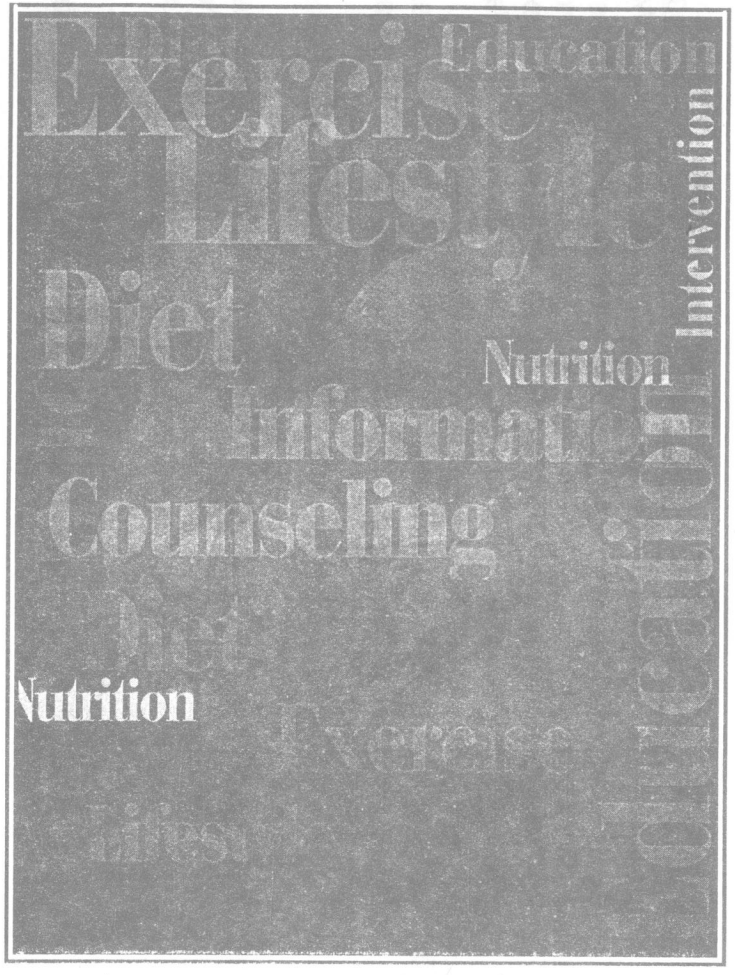
Quick Reference Guide for Clinicians

Number 17

Cardiac Rehabilitation as Secondary Prevention



U.S. Department of Health and Human Services
Public Health Service

Attention Clinicians:

The *Clinical Practice Guideline* on which this *Quick Reference Guide for Clinicians* is based was developed by a multidisciplinary, private-sector panel comprising health care professionals and consumer representatives sponsored by the Agency for Health Care Policy and Research (AHCPR) and the National Heart, Lung, and Blood Institute (NHLBI). Panel members were:

Nanette Kass Wenger, MD (*Co-Chair*)
Erika Sivarajan Froelicher, RN, PhD (*Co-Chair*)
L. Kent Smith, MD, MPH (*Project Director*)
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An explicit, science-based methodology was employed together with expert clinical judgment to develop specific statements on comprehensive, long-term cardiac rehabilitation involving medical evaluation; prescribed exercise; cardiac risk factor modification; and education, counseling, and behavioral interventions. Extensive literature searches were conducted, and critical reviews and syntheses were used to evaluate empirical evidence and significant outcomes. Peer review was undertaken to evaluate the validity, reliability, and utility of the guideline in clinical practice.

This *Quick Reference Guide for Clinicians* presents summary points from the *Clinical Practice Guideline*. The latter provides a description of the guideline development process, thorough analysis and discussion of the available research, critical evaluation of the assumptions and knowledge of the field, more complete information for health care decisionmaking, consideration for patients with special needs, and references. Decisions to adopt particular recommendations from either publication must be made by practitioners based on available resources and circumstances presented by the individual patient.

AHCPR invites comments and suggestions from users for consideration in development and updating of future guidelines. Please send written comments to:

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Quick Reference Guide for Clinicians

Number 17

Cardiac Rehabilitation as Secondary Prevention

U.S. Department of Health and Human Services
Public Health Service
Agency for Health Care Policy and Research
National Heart, Lung, and Blood Institute

AHCPR Publication No. 96-0673
October 1995

Abstract

This *Quick Reference Guide for Clinicians* highlights the conclusions and recommendations from *Cardiac Rehabilitation, Clinical Practice Guideline No. 17*, which was formulated by a panel representing the major health care disciplines involved in cardiac rehabilitation. The conclusions and recommendations were derived from an extensive and critical review of the scientific literature pertaining to cardiac rehabilitation, as well as from the expert opinion of the panel. This guide addresses the role of cardiac rehabilitation and the potential benefits to be derived in the comprehensive care of the 13.5 million patients with coronary heart disease in the United States, as well as the 4.7 million patients with heart failure and the several thousand patients undergoing heart transplantation. This *Quick Reference Guide for Clinicians* highlights the major effects of multifactorial cardiac rehabilitation services: medical evaluation; prescribed exercise; cardiac risk factor modification; and education, counseling, and behavioral interventions. The outcomes of and recommendations for cardiac rehabilitation services are categorized as to their effects on exercise tolerance, strength training, exercise habits, symptoms, smoking, lipids, body weight, blood pressure, psychological well-being, social adjustment and functioning, return to work, morbidity and safety issues, mortality and safety issues, and pathophysiologic measures. Patients with heart failure and after cardiac transplantation, as well as elderly patients, are specifically addressed. Alternate approaches to the delivery of cardiac rehabilitation services are presented.

Suggested Citation

Wenger NK, Froelicher ES, Smith LK, et al. *Cardiac Rehabilitation as Secondary Prevention.* Clinical Practice Guideline. Quick Reference Guide for Clinicians, No. 17. Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research and National Heart, Lung, and Blood Institute. AHCPR Pub. No. 96-0673. October 1995.

Cardiac Rehabilitation as Secondary Prevention

Purpose and Scope

Cardiovascular disease is the leading cause of morbidity and mortality in the United States, accounting for almost 50 percent of all deaths. Coronary heart disease (CHD) with its clinical manifestations of stable angina pectoris, unstable angina, acute myocardial infarction, and sudden death affects 13.5 million Americans. Nearly 1.5 million Americans sustain myocardial infarction each year, of which almost 500,000 episodes are fatal. Myocardial infarction can occur at young age: 5 percent occur in people younger than age 40, and 45 percent occur in people under age 65.

The almost 1 million survivors of myocardial infarction each year and the more than 7 million patients with stable angina pectoris are candidates for cardiac rehabilitation, as are patients following revascularization with coronary artery bypass graft surgery (CABG) (309,000 patients in 1993, 45 percent under age 65) or percutaneous transluminal coronary angioplasty (PTCA) and other transcatheter interventional procedures (362,000 in 1993, 54 percent under age 65). Although several million patients with CHD are candidates for cardiac rehabilitation services, only 11–20 percent have participated in cardiac rehabilitation programs. More recently, among patients with acute myocardial infarction enrolled in the Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries (GUSTO) Trial, 38 percent of U.S. patients and 32 percent of Canadian patients were subsequent participants in cardiac rehabilitation programs.

Heart failure is the most common discharge diagnosis for hospitalized Medicare patients and the fourth most common discharge diagnosis for all hospitalized patients in the United States. Application of cardiac rehabilitation services to patients with heart failure and after cardiac transplantation has gained increasing recognition and acceptance as its benefits and safety are documented. An estimated 4.7 million patients with heart failure may be candidates for cardiac rehabilitation.

Cardiac rehabilitation is characterized by comprehensive long-term services involving medical evaluation; prescribed exercise; cardiac risk factor modification; and education, counseling, and behavioral interventions. This multifactorial process is designed to limit the adverse physiologic and psychological effects of cardiac illness, reduce the risk of sudden death or reinfarction, control cardiac symptoms, stabilize or reverse the atherosclerotic process, and enhance the patient's psychosocial and vocational status. Provision of these services is physician-directed and implemented by a variety of health care professionals.

This guide is designed for use by health care professionals who provide care to patients with cardiovascular disease. These clinicians include physicians (primary care, cardiologists, and cardiovascular surgeons), nurses, exercise physiologists, dietitians, behavioral medicine specialists, psychologists, and physical and occupational therapists. The information can guide clinical decisionmaking regarding referral and followup of patients for cardiac rehabilitation services, as well as administrative decisions regarding the availability of and access to cardiac rehabilitation.

Figure 1 presents the decision tree for cardiac rehabilitation services. This figure describes patient categories addressed by the guide as well as the patient assessment and treatment strategies involved in the delivery of cardiac rehabilitation services. Tables 1 and 2 summarize the scientific evidence on which *Cardiac Rehabilitation, Clinical Practice Guideline No. 17*, is based. The evidence summaries (Tables 1 and 2) display the outcomes pertaining to the two major components of cardiac rehabilitation services: (1) exercise training and (2) education, counseling, and behavioral interventions. *Cardiac Rehabilitation, Clinical Practice Guideline No. 17*, the highlights of which are provided here, offers a more comprehensive presentation of the scientific basis for cardiac rehabilitation services and their outcomes.

The components of cardiac rehabilitation services include exercise training; education, counseling, and behavioral interventions; and organizational issues, including consideration of alternate approaches to the delivery of cardiac rehabilitative care. The physiologic parameters targeted included improvement in exercise tolerance and exercise habits; optimization of risk factor status including improvement in blood lipid and lipoprotein profiles, body weight, blood glucose and blood pressure levels, and cessation of smoking. Emotional responses to living with heart disease must be addressed, including reduction of stress and anxiety and lessening of depression. Functional independence of patients, particularly at elderly age, is an essential goal. Return to appropriate and satisfactory occupation could benefit both patients and society. Throughout, the panel highlighted the added effectiveness of multifactorial cardiac rehabilitation services, integrated in a comprehensive approach.

More than 400 scientific reports were critically reviewed, with 334 included as references in the *Clinical Practice Guideline*. The review process focused on the components of cardiac rehabilitation as specific interventions, with benefits and harms rigorously examined, and attention devoted to the generalizability of published results. When appropriate and necessary, expert opinion was formally derived from the panel to supplement conclusions derived from the comprehensive review of the scientific literature.

The results of cardiac rehabilitation services, based on reports in the scientific literature, are summarized in this *Quick Reference Guide for Clinicians*. The most substantial benefits include:

- Improvement in exercise tolerance.
- Improvement in symptoms.
- Improvement in blood lipid levels.
- Reduction in cigarette smoking.

- Improvement in psychosocial well-being and reduction of stress.
- Reduction in mortality.

The outcomes of application of cardiac rehabilitation services are addressed on the following pages.

Exercise Tolerance

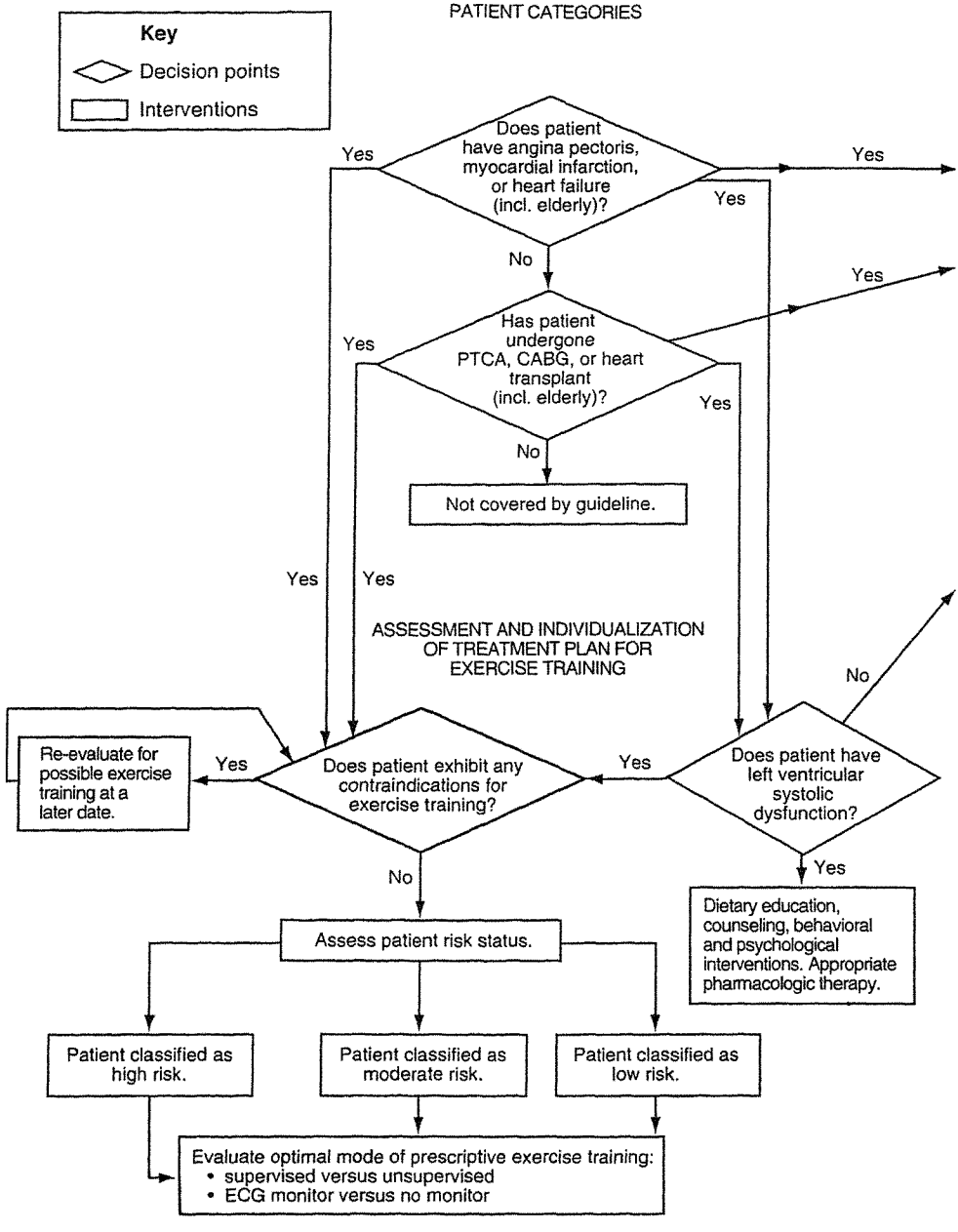
Cardiac rehabilitation exercise training consistently improves objective measures of exercise tolerance, without significant cardiovascular complications or other adverse outcomes. Appropriately prescribed and conducted exercise training is recommended as an integral component of cardiac rehabilitation services, particularly for patients with decreased exercise tolerance. Continued exercise training is required to sustain improved exercise tolerance.

The beneficial effect of cardiac rehabilitation exercise training on exercise tolerance is one of the most clearly established favorable outcomes for coronary patients with angina pectoris, myocardial infarction, CABG, and PTCA and for patients with compensated heart failure or a decreased ventricular ejection fraction or following cardiac transplantation. This approach is particularly beneficial for patients with decreased functional capacity. The large number of studies that reported a favorable outcome allowed the panel to ascertain the characteristics of exercise training that resulted in improved exercise tolerance. The most consistent benefit appeared to occur with exercise training at least three times weekly for 12 or more weeks' duration. The duration of aerobic exercise training sessions varied from 20 to 40 minutes, at an intensity approximating 70–85 percent of the baseline exercise test heart rate.

No increase in cardiovascular complications or other serious adverse outcomes were reported in any randomized controlled trial that evaluated exercise training in patients with CHD. These trials involved patients with various manifestations of CHD including 3,932 patients following myocardial infarction, 745 patients with catheterization-documented CHD, 215 patients following CABG, and 139 patients following PTCA. No deterioration in measures of exercise tolerance was reported in any patient undergoing exercise training, nor did any controlled study document significantly greater improvement in exercise tolerance in control patient groups compared with exercise patient groups.

Limited data fail to demonstrate the efficacy of education, counseling, and behavioral interventions as sole interventions, independent of cardiac rehabilitation exercise training, in improving exercise tolerance. Education and behavioral interventions may improve morale, self-esteem, and adherence to exercise.

Figure 1. Decision tree for cardiac rehabilitation services



Adapted from material provided by Health Economics Research, Inc., Waltham, MA.

ASSESSMENT AND INDIVIDUALIZATION OF TREATMENT PLAN FOR RISK FACTOR MODIFICATION, PSYCHOSOCIAL STATUS

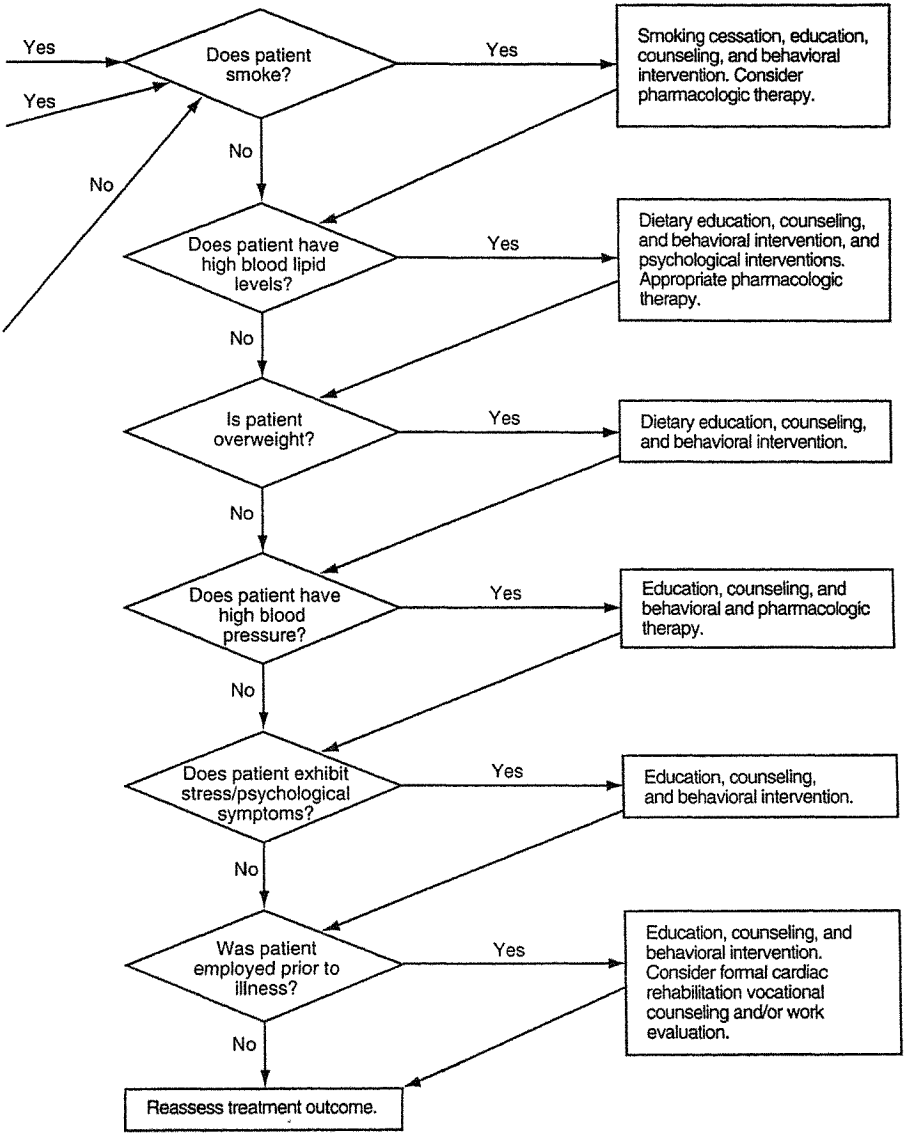


Table 1. Summary of evidence for cardiac rehabilitation outcomes: Effects of exercise training

Outcome	Evidence base[a]				Strength of evidence[b]
	Total number of studies	Randomized studies	Nonrandomized studies	Observational studies	
Exercise tolerance	114	46	25	43	A
Exercise tolerance (strength training)	7	4	3	0	B
Exercise habits	15	10	2	3	B
Symptoms	26	12	7	7	B
Smoking	24	12	8	4	B
Lipids	37	18	6	13	B
Body weight	34	11	7	16	C
Blood pressure	18	9	6	3	B
Psychological well-being	20	9	8	3	B
Social adjustment and functioning	6	2	2	2	B
Return to work	28	10	9	9	A
Morbidity	42 (+2 survey reports)	15	14	13	A
Mortality	31 (+2 survey reports)	17	8	6	B
Pathophysiologic measures:					
Changes in atherosclerosis	9	5	1	3	A/B
Changes in hemodynamic measurements	5	0	0	5	B
Changes in myocardial perfusion/ myocardial ischemia	11	6	2	3	B

Table 1. Summary of evidence for cardiac rehabilitation outcomes: Effects of exercise training (*continued*)

Outcome	Evidence base[a]				Strength of evidence[b]
	Total number of studies	Randomized studies	Nonrandomized studies	Observational studies	
Changes in myocardial contractility, ventricular wall motion abnormalities, and/or ventricular ejection fraction	22	9	5	8	B
Changes in cardiac arrhythmias	5	4	0	1	B
Heart failure patients	12	5	3	4	A
Cardiac transplantation patients	5	0	1	4	B
Elderly patients	7	0	1	6	B

[a]Number of studies from scientific literature by type of study design.

[b]Rating for strength of evidence:

A Scientific evidence from well-designed and well-conducted controlled trials (randomized and nonrandomized) provides statistically significant results that consistently support the guideline statement.

B Scientific evidence is provided by observational studies or by controlled trials with less consistent results.

C Guideline statement supported by expert opinion; the available scientific evidence did not present consistent results or controlled trials were lacking.

Strength Training

Strength training improves skeletal muscle strength and endurance in clinically stable coronary patients. Training measures designed to increase skeletal muscle strength can safely be included in the exercise-based rehabilitation of clinically stable coronary patients, when appropriate instruction and surveillance are provided.

Scientific data demonstrate the effectiveness of resistance exercise training in selected patients with CHD. The absence of signs or symptoms of myocardial ischemia, abnormal hemodynamic changes, and cardiovascular complications in these studies suggests that resistance exercise training is safe for selected coronary patients who have previous-

Table 2. Summary of evidence for cardiac rehabilitation outcomes: Effects of education, counseling, and behavioral interventions

Outcome	Evidence base[a]				Strength of evidence[b]
	Total number of studies	Randomized studies	Nonrandomized studies	Observational studies	
Smoking	7	5	1	1	B
Lipids	18	12	3	3	B
Weight	5	3	1	1	B
Blood pressure	2	0	2	0	B
Exercise tolerance	3	1	1	1	C
Symptoms	4	2	1	1	B
Return to work	3	2	0	1	C
Stress/ psycho-logical well-being	14	7	5	2	A
Morbidity	3	3	0	0	B
Mortality	8	8	0	0	B

[a]Number of studies from scientific literature by type of study design.

[b]Rating for strength of evidence:

A Scientific evidence from well-designed and well-conducted controlled trials (randomized and non-randomized) provides statistically significant results that consistently support the guideline statement.

B Scientific evidence is provided by observational studies or by controlled trials with less consistent results.

C Guideline statement supported by expert opinion; the available scientific evidence did not present consistent results or controlled trials were lacking.

ly participated in rehabilitative aerobic exercise training. Improvement in muscle strength can benefit patients' performance of activities of daily living. The absence of cardiovascular and orthopedic complications in the 3-year followup of strength training was largely attributed to strict preliminary screening and careful supervision. Most studies involved small numbers of low-risk male patients, 70 years or younger, with minimal functional aerobic impairment and with normal or near-normal left ventricular function. The extent to which the safety and effectiveness demonstrated by these studies can be extrapolated to other populations of coronary or cardiac patients (e.g., women, older patients of both genders with low aerobic fitness, patients at moderate-to-high cardiovascular risk) requires study.

Exercise Habits

Cardiac rehabilitation exercise training promotes increased participation in exercise by patients after myocardial infarction and CABG. This effect does not persist long-term after completion of exercise rehabilitation. Long-term cardiac rehabilitation exercise training is recommended to provide the benefit of enhanced exercise tolerance and exercise habits.

There is suggestive evidence that exercise training enhances subsequent exercise habits. A limitation of the scientific data relating to continued exercise habits as a result of rehabilitative exercise training is the self-report nature of the information, which was typically based on questionnaire or physical activity diary data. Despite limited information in the cardiac rehabilitation literature, extensive studies and position statements in populations without apparent heart disease document that regular exercise, including a wide scope of physical activities with a broad range of intensity and duration, has beneficial effects on overall health, morbidity, and mortality. Patients should be encouraged to undertake exercise activities following cardiac exercise rehabilitation that are personally enjoyable and that can be sustained long-term. The panel highlighted the need to encourage women, particularly older women, to participate in cardiac rehabilitation designed to enhance exercise capacity and physical activity.

The panel endorses the position statement of the American Heart Association regarding physical activity, that "regular aerobic physical activity increases exercise capacity and plays a role in both primary and secondary prevention of cardiovascular disease."

Symptoms

Exercise rehabilitation decreases angina pectoris in patients with CHD and decreases symptoms of heart failure in patients with left ventricular systolic dysfunction. Exercise training is recommended as an integral component of the symptomatic management of these patients. Symptoms of angina pectoris are also reduced by cardiac rehabilitation education, counseling, and behavioral interventions alone or as a component of multifactorial cardiac rehabilitation.

Improvement in cardiovascular symptomatic status, both angina pectoris and heart failure symptoms, occurs as a result of cardiac rehabilitation exercise training. Symptomatic outcomes in the scientific studies were confounded by inadequate information regarding changes in medication status, by differing levels of exercise or physical activity, as well as by nonrehabilitation exercise activities of control patients. Change in symptomatic status of cardiac patients often results in changes in medication regimens.

Education and behavioral interventions, either alone or as components of multifactorial cardiac rehabilitation, are associated with a reduction in angina pectoris. Behavioral interventions are generally effective in reducing anginal pain.

Smoking

A combined approach of cardiac rehabilitation education, counseling, and behavioral interventions results in smoking cessation and relapse prevention. Smoking cessation and relapse prevention programs should be offered to patients who are smokers to reduce their risk of subsequent coronary events. Smoking cessation is achieved by specific smoking cessation strategies.

Well-designed education, counseling, and behavioral interventions (relapse prevention) reduce cigarette smoking. Between 17 and 26 percent of patients can be expected to stop smoking, in addition to the spontaneously high smoking cessation rates in most populations soon after myocardial infarction. One effective model includes nurse-managed smoking cessation behavioral intervention with biochemical verification of smoking status. Whether biochemical verification should be recommended for clinical practice is unclear. Scientific evidence, consensus papers, and other scientific reviews in nonrehabilitation settings, including the Surgeon General's messages since 1965, lend strong support that education, counseling, and behavioral interventions are beneficial for smoking cessation. Given the documented benefit of smoking cessation in decreasing coronary risk, specific techniques of proven value in effecting smoking cessation should be incorporated in multifactorial cardiac rehabilitation.

There is little or no evidence of beneficial outcome in smoking cessation resulting from exercise training as a sole intervention.

Lipids

Intensive nutrition education, counseling, and behavioral interventions improve dietary fat and cholesterol intake. Education, counseling, and behavioral interventions about nutrition, with and without pharmacologic lipid-lowering therapy, result in significant improvement in blood lipid levels and are recommended as a component of cardiac rehabilitation. Optimal lipid management requires specifically directed dietary and, as medically indicated, pharmacologic management, in addition to cardiac rehabilitation exercise training.

Efficacy is documented in non-cardiac-rehabilitation settings of intensive nutrition education, counseling, and behavioral interventions on dietary fat intake and blood lipid levels. Results from a meta-analysis of 70 studies indicate that weight reduction

through dietary modification can help normalize plasma lipid and lipoprotein levels in overweight individuals. *The Second Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II)* (NCEP II) recommended a low-density lipoprotein (LDL) cholesterol goal of less than 100 mg/dL for coronary patients. This requires high-intensity intervention that includes education, counseling, behavioral intervention, and adherence and motivational strategies as well as pharmacologic therapy for appropriate patients. Thus, independent effects of education and counseling may be impossible to ascertain.

Improvement in lipid profiles resulting from multifactorial cardiac rehabilitation is well established by review of the scientific literature. Most randomized controlled trials reported beneficial effects on total cholesterol, LDL cholesterol, high-density lipoprotein (HDL) cholesterol, and triglyceride levels in rehabilitation compared with control patients. Well-designed nonrandomized controlled trials reported similar beneficial outcomes. The rehabilitation studies that reported the most favorable impact on lipid levels were multifactorial, that is, providing exercise training, dietary education and counseling, and, in some studies, pharmacologic treatment, psychological support, and behavioral training. These favorable effects on lipid profiles involved patients who were both younger and older than 65 years of age. Cardiac rehabilitation exercise training as a sole intervention has inconsistent effects on lipid and lipoprotein levels.

The panel concurs with the recommendation of the NCEP II regarding the role of physical activity for lipid control, namely "the appropriate use of physical activity is considered an essential element in the nonpharmacologic therapy of elevated serum cholesterol." The panel also noted the results of a major randomized placebo-controlled trial of cholesterol lowering in coronary patients, most with prior myocardial infarction. Patients treated with a cholesterol-altering medication and a Step I diet showed a significant reduction in total mortality, coronary death, and major coronary events compared with diet-plus placebo-treated patients. Favorable results occurred in both men and women patients younger and older than 60 years of age. The panel agreed with the trial conclusions that patients with CHD and diet-resistant cholesterol levels above 210 mg/dL should be considered for treatment with lipid-altering medication.

Body Weight

Multifactorial cardiac rehabilitation that combines dietary education, counseling, and behavioral interventions designed to reduce body weight can help patients lose weight. Education as a sole intervention is unlikely to achieve and maintain weight loss. Cardiac rehabilitation exercise training as a sole intervention also has an inconsistent effect on controlling overweight and is not recommended as an isolated approach for weight loss. The optimal management recommended for overweight patients to promote maintenance of

weight loss requires multifactorial rehabilitation including nutrition education, counseling, and behavioral modification, in addition to exercise training.

Education is a necessary component of a successful weight-reduction intervention but is not sufficient as a sole intervention to effect sustained weight loss. Nutrition education combined with behavioral interventions and prescribed exercise training can achieve modest and sustained weight loss. Results of meta-analysis of 70 studies indicate that weight reduction through dieting can also help normalize plasma lipid and lipoprotein levels in overweight individuals. The panel noted a review of the behavioral therapy literature involving obese patients; state-of-the-art weight loss programs that have been shown to be successful in nonrehabilitation settings are also likely to be successful in a cardiac rehabilitation setting. Expert opinion agrees that multifactorial interventions, with intensive education, counseling, and behavioral intervention, are effective to reduce weight.

Rehabilitative exercise training, as a component of multifactorial intervention, appears beneficial in improving body weight, excess body mass, or percentage of body fat. Exercise training as a sole intervention has no consistent effect, but no exercise-training study specifically targeted overweight coronary patients, and the definition of "overweight" varied among studies.

Blood Pressure

Expert opinion supports a multifactorial education, counseling, behavioral, and pharmacologic approach as the recommended strategy for the management of hypertension. This approach is documented to be effective in nonrehabilitation populations. Neither education, counseling, and behavioral interventions nor rehabilitative exercise training as sole interventions have been shown to control elevated blood pressure levels.

Scientific evidence suggests that cardiac rehabilitation education alone fails to significantly decrease blood pressure. One of the most serious flaws in study designs includes the mixed sample of normotensive patients and a small proportion of hypertensive patients. The panel recommends the application of *The Fifth Report of the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure* (JNC V) educational and behavioral recommendations as an important component of a multifactorial approach to reduce hypertension for the cardiac rehabilitation population. The JNC V recommendations, which were based on the opinion of recognized experts on hypertension, stated that "lifestyle modifications such as weight reduction, physical activity and moderation of dietary sodium are recommended as definitive or adjunctive therapy for hypertension." The JNC V also concluded that the scientific literature does not support use of stress management as a sole intervention for hypertension control and reiterated that relaxation and biofeedback techniques have little effect on hypertension control.

Review of the scientific evidence suggests that exercise-based cardiac rehabilitation has only modest effects in reducing blood pressure levels, but confounding variables include the effects of antihypertensive medications and medication changes. No study was specifically designed to address hypertension control in patients with elevated blood pressures participating in exercise-based cardiac rehabilitation. It is unlikely that hypertensive patients with CHD would be provided solely exercise training without other appropriate therapies such as weight reduction, sodium restriction, moderation or abstinence from alcohol, or pharmacologic therapy, although these components may have been directed by the patient's treating physician.

Comprehensive educational programs should include information about weight management, exercise, and nutrition; they should also provide information about the purpose of medications, their potential side effects, and strategies to improve medication adherence.

Psychological Well-Being

Education, counseling, and/or psychosocial interventions, either alone or as a component of multifactorial cardiac rehabilitation, result in improved psychological well-being and are recommended to complement the psychosocial benefits of exercise training.

Cardiac rehabilitation exercise training, with and without other cardiac rehabilitation services, results in improvement in measures of psychological status and functioning and is recommended to enhance psychological functioning, particularly as a component of multifactorial cardiac rehabilitation. Exercise training as a sole intervention does not consistently improve measures of anxiety and depression.

The scientific literature provides evidence of psychological improvement following education, counseling, and/or psychosocial interventions. Training in behavioral modification, stress management, and relaxation techniques is effective in lowering levels of self-reported emotional stress and in modifying Type-A behavior.

Cardiac rehabilitation exercise training, either alone or as a component of multifactorial rehabilitation, often results in improvement in various measures of psychological status and functioning. This evidence from the scientific literature is consistent with the widespread belief among cardiac rehabilitation professionals that cardiac rehabilitation exercise training improves the sense of well-being among participants, particularly among individuals with high levels of distress at entry into the study. Patients tend to perceive themselves as improving in a number of psychosocial domains, although these perceptions may not be objectively documented. More sensitive tests may have to be developed to better ascertain changes in cardiac patients without specific psychiatric illness.

Studies of exercise rehabilitation as a sole intervention are confounded by the consequences of group interaction, formation of social support networks, peer and professional support, counseling, and guidance, all of which may affect depression, anxiety, and self-confidence.

Social Adjustment and Functioning

Cardiac rehabilitation exercise training improves social adjustment and functioning and is recommended to improve social outcomes.

The scientific literature addressed various measures of social adjustment and functioning in patients following cardiac rehabilitation exercise training including the Sickness Impact Profile scores, leisure and social questionnaire scores, social activity scores, and scores of satisfaction with work and social satisfaction. Randomized controlled trials established that social benefits result from participation in exercise and in multifactorial cardiac rehabilitation. Only two reports involved patients over age 65; social outcomes for this age group may differ from those in the majority of patients studied, younger than 60–65 years.

Return to Work

Cardiac rehabilitation exercise training exerts less of an influence on the rates of return to work than many nonexercise variables including employer attitudes, prior employment status, economic incentives, and the like. Exercise training as a sole intervention is not recommended to facilitate return to work, nor have education, counseling, and behavioral interventions resulted in improvement in rates of return to work. Many patients return to work without formal interventions. However, in selected patients, formal cardiac rehabilitation vocational counseling may improve rates of return to work.

Assessment of return to work as a result of exercise training must be considered within the context of social and political variables that are typically not addressed in the studies of cardiac rehabilitation; these include the political system and social policies of the country in which cardiac rehabilitation occurs. Additional factors include employment statistics for the years of the study, economic incentives or disincentives for patients to return to work, non-patient-related factors such as employer attitudes, and the pre-illness employment status of the patient, among others. Return to work as a measure of outcome of exercise-based cardiac rehabilitation may not be appropriate unless formal vocational rehabilitation services are provided to patients as part of the rehabilitative process.

Although multifactorial cardiac rehabilitation has not been shown to alter the rates of return to work, education and counseling may improve a patient's potential for return to work.

Better understanding (via education) of capabilities and limitations regarding work may influence a patient's self-efficacy for returning to previous employment or for seeking job retraining. A randomized controlled trial in a nonrehabilitation setting of the effects of occupational work evaluation on return to work, involving patients after myocardial infarction, documented a marked reduction in the duration of convalescence.

Morbidity and Safety Issues

The safety of exercise rehabilitation is well established; the rates of myocardial infarction and cardiovascular complications during exercise training are very low.

Cardiac rehabilitation exercise training does not change the rates of nonfatal reinfarction.

Education, counseling, and behavioral interventions as components of multifactorial cardiac rehabilitation may decrease progression of coronary atherosclerosis and lower recurrent coronary event rates.

Appropriately designed and conducted exercise-based cardiac rehabilitation can safely be undertaken in appropriately selected patients undergoing individualized initial assessment and surveillance.

The randomized controlled trials reported in the scientific literature show no evidence for reduction in cardiac morbidity, specifically nonfatal reinfarction, as a result of exercise rehabilitation. No study documented an increase in morbidity comparing rehabilitation patients with control patients among 4,578 patients in the controlled trials reviewed.

A large survey of adverse experiences during rehabilitative exercise training in 142 U.S. cardiac rehabilitation programs (1980–84) reported a very low rate of nonfatal reinfarction of 1 per 294,000 patient-hours. These 1980–84 survey data may not be applicable to the contemporary treatment of coronary patients, including the widespread use of risk stratification procedures following myocardial infarction, the more aggressive management techniques including thrombolytic therapy and myocardial revascularization, as well as current pharmacologic therapies for postinfarction patients (e.g., beta blockers, angiotensin-converting enzyme [ACE] inhibitors) that may further reduce reinfarction and morbidity in coronary patients. The current low nonfatal reinfarction rates may not be amenable to further reduction by exercise training as a sole intervention.

Based on the scientific literature, education, counseling, and behavioral interventions alone may have limited beneficial effect on cardiovascular event rates. Education, counseling, and behavioral interventions designed to encourage patients to adhere to therapies, as a component of multifactorial cardiac rehabilitation, have been associated with reduction in recurrent cardiovascular event rates, as well as with regression of atherosclerosis.

Mortality and Safety Issues

Based on meta-analyses, total and cardiovascular mortality are reduced in patients following myocardial infarction who participate in cardiac rehabilitation exercise training, especially as a component of multifactorial rehabilitation. Education, counseling, and behavioral interventions reduce cardiac and overall mortality rates and are recommended in the multifactorial rehabilitation of patients with CHD.

A survival benefit among patients participating in exercise training as a component of multifactorial cardiac rehabilitation is suggested from review of the scientific data, but this benefit cannot be attributed solely to exercise training because many studies involved multifactorial rehabilitation. Because of the small number of patients in most randomized controlled trials, the panel used results of meta-analyses to gain additional information about mortality outcomes. Two meta-analyses of 21 randomized controlled trials of cardiac rehabilitation that included more than 4,000 patients with CHD established significant mortality reduction, approximating 25 percent at 3 years, in rehabilitation patients compared with control patients. This mortality reduction is similar to that with other interventions for patients with CHD (e.g., trials of beta-blocker drug therapy following myocardial infarction; ACE inhibitor therapy for left ventricular systolic dysfunction and heart failure). The beneficial mortality outcome was greater in the 15 trials that used multifactorial cardiac rehabilitation compared with the 7 trials that used exercise training as the sole intervention.

The randomized controlled trials in the panel's database that reported mortality rates included a total of 7,063 patients. In no trial was the rate of fatal events greater in the intervention group than in the control group.

Most studies involved principally male patients younger than age 65 years following myocardial infarction and excluded high-risk complex patients, limiting the generalizability of the data. The percentage of females, when enrolled, was 20 percent or less. Furthermore, subsequent to the research studies cited as scientific evidence, mortality has been further reduced by nonrehabilitation interventions such as myocardial revascularization procedures and newer pharmacologic agents that have far more powerful effects on survival.

Information obtained from two large surveys of cardiac rehabilitation program responses to questionnaires provided retrospective safety data regarding exercise training. Few fatal cardiac events occurred during or immediately following exercise training: 1 per 116,400 patient-hours of participation in supervised exercise training in the 1978 report and 1 per 784,000 patient-hours in the 1986 report. The data from both survey reports antedate the use of contemporary risk stratification procedures and contemporary medical and surgical therapies for CHD and heart failure. No mortality data were reported by gender or patient age, nor was defini-

tive information available regarding the effect of levels of supervision and electrocardiographic (ECG) monitoring of exercise training.

A variety of education, counseling, and behavioral interventions are associated with reductions in total and cardiac mortality rates. The panel noted the consistency with which decreased mortality rates were reported in the randomized controlled trials of multifactorial cardiac rehabilitation involving education, counseling, and behavioral interventions. The panel recognizes the potential for reducing mortality rates by education, counseling, and behavioral interventions that are designed to reduce cardiac risk, as components of multifactorial cardiac rehabilitation.

Pathophysiologic Measures

Coronary Atherosclerosis

Cardiac rehabilitation exercise training as a sole intervention does not result in regression or limitation of progression of angiographically documented coronary atherosclerosis. Exercise training, combined with intensive dietary intervention, with and without lipid-lowering drugs, results in regression or limitation of progression of angiographically documented coronary atherosclerosis and is recommended.

Hemodynamic Measurements

Cardiac rehabilitation exercise training has no apparent effect on development of a coronary collateral circulation and produces no consistent changes in cardiac hemodynamic measurements at cardiac catheterization. Exercise training in patients with heart failure and a decreased ventricular ejection fraction produces favorable hemodynamic changes in the skeletal musculature and is recommended to improve skeletal muscle functioning.

Myocardial Perfusion and/or Evidence of Myocardial Ischemia

Cardiac rehabilitation exercise training decreases myocardial ischemia as measured by exercise ECG, ambulatory ECG recording, and radionuclide perfusion imaging and is recommended to improve these measures of myocardial ischemia.

Myocardial Contractility, Ventricular Wall Motion Abnormalities, and/or Ventricular Ejection Fraction

Cardiac rehabilitation exercise training has little effect on ventricular ejection fraction and regional wall motion abnormalities and is not recommended to improve measures of ventricular systolic function. The effect of exercise training on left ventricular function in patients after anterior Q-wave myocardial infarction with left ventricular dysfunction is variable.

Occurrence of Cardiac Arrhythmias

Cardiac rehabilitation exercise training has inconsistent effects on ventricular arrhythmias.

A number of scientific reports described the pathophysiologic outcomes of exercise training listed here. These studies explored and at times interrelated pathophysiologic mechanisms whereby exercise training may engender benefits or harms. All reports involved predominantly or exclusively male patients, typically of middle age, with few or no elderly patients studied; these demographic constraints limit the generalizability of the outcome data.

Multifactorial cardiac rehabilitation, including exercise training and dietary intervention, with and without the use of lipid-altering drugs, effected regression or limited progression of angiographically documented coronary atherosclerosis. The effect of exercise training as a sole intervention is not impressive. However, subsequent coronary events may be related to factors other than change in arterial luminal diameter, that is, factors promoting plaque stability versus rupture, which may be related to circulating lipid levels, among others.

Development of an angiographically documented coronary collateral circulation has not been demonstrated with exercise training; it occurred only with progression of the underlying coronary atherosclerosis. No prominent or consistent changes in cardiac hemodynamic measurements at cardiac catheterization occurred as a result of exercise training. In patients with heart failure and a decreased ventricular ejection fraction, improvement occurred in leg hemodynamic parameters with exercise, supporting the favorable effect of exercise training on the skeletal musculature.

The beneficial effects of exercise training on myocardial perfusion and/or measures of myocardial ischemia included less ischemic ECG abnormalities at exercise testing and during ambulatory ECG recording. Resolution of reversible thallium perfusion defects in the randomized controlled trials was also greater among exercising than nonexercising patients.

Most of the studies that examined the effect of usual rehabilitative exercise training on measures of myocardial function showed no significant difference in ejection fraction or regional wall motion abnormalities between exercising and control groups. Apparently spontaneous improvement in resting ejection fraction after myocardial infarction occurred in both exercise and control populations in several randomized clinical trials, rendering suspect described improvements in ejection fraction in observational studies. A nonrandomized controlled study of patients following anterior Q-wave myocardial infarction and decreased ejection fraction showed worsening of ejection fraction and wall motion asynergy in exercising compared with nonexercising patients. Two subsequent randomized controlled trials in patients following anterior Q-wave myocardial infarction with baseline decreased ejection fraction documented comparable spontaneous deterioration in global and regional left ventricular function in exercising and control patients.

Studies that described changes in ventricular arrhythmias related to exercise rehabilitation provide inconsistent outcomes. No randomized controlled trial reported a significant arrhythmia-related adverse clinical outcome.

Rehabilitative exercise training in patients with heart failure and moderate-to-severe left ventricular systolic dysfunction improves functional capacity and symptoms, without changes in left ventricular function. Cardiac rehabilitation exercise training is recommended to attain functional and symptomatic improvement.

Rehabilitative exercise training in patients following cardiac transplantation improves measures of exercise tolerance and is recommended for this purpose.

In the early years of exercise rehabilitation, cardiac enlargement, decreased left ventricular ejection fraction, and overt cardiac failure were considered relative or absolute contraindications to exercise training. Only in recent years has exercise training been undertaken in these patients; even these recent trials reflect only limited concomitant use of contemporary vasodilator drug therapies, particularly ACE inhibitors, which are now considered the standard of care for heart failure. The panel concurs with the recommendation of the AHCPR publication *Heart Failure: Evaluation and Care of Patients With Left-Ventricular Systolic Dysfunction, Clinical Practice Guideline No. 11*, that "patients with heart failure due to left-ventricular systolic dysfunction should be given a trial of ACE inhibitors unless specific contra-indications exist."

Most studies of exercise training of patients with heart failure and moderate-to-severe left ventricular dysfunction do not demonstrate deterioration in left ventricular function. Peripheral (skeletal muscle) adaptations appear to mediate the improvement in exercise tolerance. Exercise training augments the symptomatic and functional benefits of ACE inhibitor therapy. Low- to moderate-intensity exercise and home exercise regimens provide benefit, but adverse events may occur in this high-risk patient group.

In summary, although the studies of exercise training have been limited by small numbers and young populations consisting predominantly of men, and had CHD as the major etiology of heart failure, exercise training in patients with heart failure and decreased ventricular systolic function resulted in documented improvement in functional capacity. Data reinforce that the favorable training effects in these patients are due predominantly to adaptations in the peripheral circulation and skeletal musculature rather than adaptations in the cardiac musculature.

Cardiac transplantation, too, is a relatively recent surgical intervention; even more recent for cardiac transplantation patients is the frequent application of exercise training. The few studies reported demonstrate improvement in exercise capacity in these medically complex patients, who are often markedly deconditioned prior to cardiac transplantation. Pretransplantation rehabilitative strength training may enhance preoperative status and operative recovery; effects of strength training after cardiac transplantation require study.

Elderly Patients

Elderly coronary patients have exercise trainability comparable to younger patients participating in similar exercise rehabilitation. Elderly female and male patients show comparable improvement. Referral to and participation in exercise rehabilitation is less frequent at elderly age, especially for elderly females. No complications or adverse outcomes of exercise training at elderly age were described in any study. Elderly patients of both genders should be strongly encouraged to participate in exercise-based cardiac rehabilitation.

Elderly patients constitute a high percentage of those with myocardial infarction, CABG, and PTCA. Elderly patients are also at high risk of disability following a coronary event.

Although few studies and no randomized controlled trials addressed the efficacy and safety of exercise training and multifactorial rehabilitation at elderly age, the available studies provide important new information of beneficial functional improvement from exercise training for current clinical practice.

Special effort is recommended to overcome the obstacles to entry and participation in cardiac rehabilitation services for elderly patients.

Alternate Approaches to the Delivery of Cardiac Rehabilitation Services

Alternate approaches to the delivery of cardiac rehabilitation services, other than traditional supervised group interventions, can be implemented effectively and safely for carefully selected clinically stable patients. Transtelephonic and other means of monitoring and surveillance of patients can extend cardiac rehabilitation services beyond the setting of supervised, structured, group-based rehabilitation. These alternate approaches have the potential to provide cardiac rehabilitation services to low- and moderate-risk patients, who comprise the majority of patients with stable CHD, most of whom do not currently participate in structured supervised rehabilitation.

Recent studies have explored new approaches to deliver cardiac rehabilitation services, with the goals of increasing availability and decreasing costs, while preserving efficacy and safety. Case management approaches to exercise training, smoking cessation, diet–drug management of hyperlipidemia, and providing emotional support and guidance to patients as needed that rely on telephone contact can be provided to appropriately selected patients with CHD.

The generalizability of these case management systems to other treatment settings including university centers, public and community hospitals, and clinics will depend largely on formulas for reimbursement for services and the extent of physician support for this approach, as well as

State regulations regarding medical and health care practices. Within each of these settings, managed care programs seeking optimal methods for coronary risk factor reduction and exercise rehabilitation may favor case management systems that provide convenient, individualized health care at low cost.

The feasibility, safety, efficacy, and economic impact of these alternate approaches have to be assessed in more diverse populations of patients with stable CHD, particularly elderly patients, those with ventricular dysfunction, and other patients of higher risk status.

Adherence

Adherence to cardiac rehabilitation services may improve patient outcomes. Adherence to cardiac rehabilitation services may be enhanced by clear communication; emotional support; understanding the patient's (and family's) values, viewpoints, and preferences; and integration of the intervention into the patient's lifestyle.

Cost

Limited data suggest that multifactorial cardiac rehabilitation is a cost-effective use of medical care resources.

A limited number of economic evaluations of cardiac rehabilitation in patients after coronary events demonstrated favorable economic outcomes. Although none of these studies provided comprehensive economic analyses, the costs of cardiac rehabilitation have to be considered in the perspective of benefits of such rehabilitation. At relatively low cost, clinical benefits are attained, as are favorable economic outcomes. Nonetheless, application of longer term multifactorial cardiac rehabilitation services may entail increased costs.

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Amount of Exercise Necessary for the Patient with Coronary Artery Disease

Barry A. Franklin, PhD, Seymour Gordon, MD, and Gerald C. Timmis, MD

Exercise training can both protect against and provoke sudden coronary death.[1] Accordingly, we are obliged to provide "safe" exercise recommendations, reducing the risk of cardiovascular complications to a minimum, while prescribing sufficient exercise to promote desired training effects. The intensity of exercise needed to attain health-related benefits may differ from what is generally prescribed for cardiorespiratory conditioning. Lower levels of physical activity than previously recommended have been shown to reduce the risk of certain chronic degenerative diseases and yet be insufficient to improve the aerobic capacity or maximal oxygen uptake ($\dot{V}O_2$max).[2] Low to moderate intensity exercise training ($\leq$60% $\dot{V}O_2$max) can elicit beneficial physiologic and psychosocial changes and possibly reduce cardiovascular-related mortality. Moreover, it appears that low- and high-intensity exercise training regimens produce comparable improvements in functional capacity and high-density lipoprotein (HDL) cholesterol, at least over the initial 3 months of conditioning.[3] This review clarifies the amount of exercise required to promote favorable adaptation and improvement in functional capacity, cardiac function, coronary risk factors, psychosocial well-being and morbidity/mortality (Figure 1) in patients with coronary artery disease (CAD).

Functional capacity: Recent studies suggest that low to moderate intensity exercise training will maintain or improve, or both, functional capacity after an acute coronary event. Simple exposure to orthostatic or gravitational stress during the bed rest stage of hospital convalescence may obviate much of the deterioration in exercise performance that follows myocardial infarction.[4] Moreover, a "spontaneous" increase in $\dot{V}O_2$max occurs in many deconditioned patients with CAD soon after hospital discharge,[5] presumably because the aerobic requirements of many daily activities exceed the "threshold" intensity for training.[6]

EFFECTS OF BED REST ON AEROBIC CAPACITY AND BENEFIT OF REGULAR CARDIOVASCULAR ORTHOSTATIC STRESS: Prolonged bed rest is seldom used any more in the care of patients with CAD because of the shortened hospital stay. Nevertheless, it has been shown to result in physiologic deconditioning and a significant decrease in the $\dot{V}O_2$max.[7] Although a traditional explanation for this phenomenon is the absence of daily physical activity, it appears that the deterioration in performance may simply reflect the lack of exposure to chronic orthostatic stress.

Convertino et al[8] studied changes in $\dot{V}O_2$max before and after 14 days of bed rest using daily 210-minute treatments using a reverse gradient garment that simulated the effects of standing. The $\dot{V}O_2$max decreased only 6% in subjects who received venous pooling treatment, compared with a 15% decrease in nontreated subjects (Table I). Moreover, a significantly greater decrease in $\dot{V}O_2$max has been demonstrated during upright than during supine exercise after bed rest, i.e., 17 versus 7%.[9] These data have important implications for inpatient cardiac rehabilitation. The loss of exposure to gravitational stress alone during the bed rest phase of hospital convalescence contributes to the reduction in $\dot{V}O_2$max after myocardial infarction, independent of myocardial damage. Thus, it appears that the deterioration in exercise performance with bed rest may be lessened simply by regular exposure to orthostatic stress, such as intermittent sitting or standing during the hospital confinement period.[4] Structured, formalized in-hospital exercise programs after acute myocardial infarction appear to offer little additional physiologic or behavioral (self-efficacy) benefits over routine medical care.[10,11]

SPONTANEOUS IMPROVEMENT IN AEROBIC CAPACITY: A significant increase in aerobic capacity, corresponding to 2 to 3 METs (1 MET = 3.5 ml O_2/kg/min), generally occurs between 3 and 11 weeks after clinically uncomplicated myocardial infarction, even in patients who undergo no formal exercise training (Figure 2).[12] Similar improvements have been reported for patients who have undergone percutaneous transluminal coronary angioplasty[13] or coronary revascularization surgery.[14] This spontaneous improvement in aerobic capacity has, from a hemodynamic perspective, been attributed to increases in peak heart rate and oxygen pulse, the latter reflecting increases in stroke volume, arteriovenous oxygen difference, or both.[12] Research indicates that self-care and other out-of-hospital activities performed by cardiac patients soon after hospital discharge frequently lead to sustained increases in heart rate and oxygen uptake that exceed frequently recommended levels for home activities.[6] These transient fluxes in cardiorespiratory activity may promote a training effect and account, at least in part, for the spontaneous improvement in aerobic capacity during the early weeks after myocardial infarction.

DeBusk et al[5] studied 70 patients with CAD soon after hospital discharge to assess whether the spontaneous increase in $\dot{V}O_2$max could be further augmented

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with exercise training. After symptom-limited treadmill exercise testing 3 weeks after infarction, 40 men were assigned to undergo 8 weeks of home (n = 12) or medically supervised gymnasium (n = 28) exercise training, whereas the remaining 30 served as control subjects, receiving no formal exercise instructions. During this 8-week interval, peak oxygen uptake increased significantly in all 3 groups: gymnasium training, from 6.6 to 11.0 METs (66%); home training, from 7.3 to 10.3 METs,

TABLE 1 Mean Changes in Maximal Oxygen Uptake ($\dot{V}O_2$max) Before and After Bed Rest

Remedial Treatment Mode	Bed Rest (days)	$\dot{V}O_2$max (liters/min)		
		Before	After	% Δ
None	14	3.9	3.3	−15
Venous pooling	14	3.3	3.1	−6

Adapted from *J Appl Physiol.*[8]
% Δ = percent change.

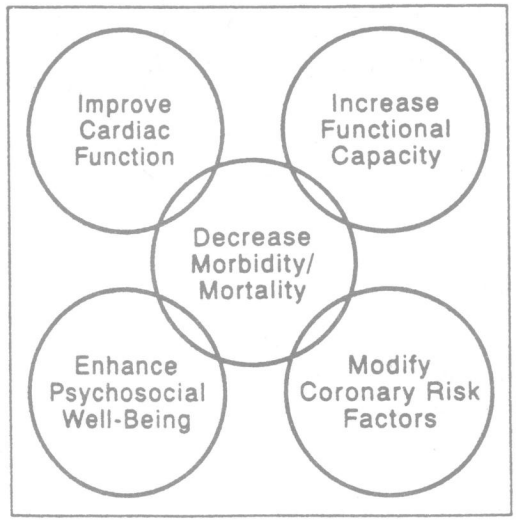

FIGURE 1. Common rationale for exercise training of patients with coronary artery disease.

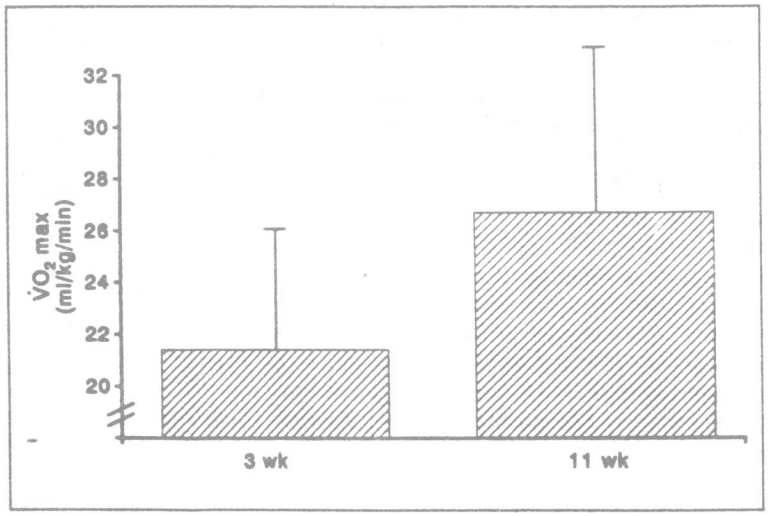

FIGURE 2. Improvement in aerobic capacity (maximal oxygen uptake [$\dot{V}O_2$ max]), expressed as ml/kg/min, after clinically uncomplicated myocardial infarction in patients who underwent no formal exercise training. Values represent mean ± SD ($p < 0.001$). (Adapted from Savin et al.[12])

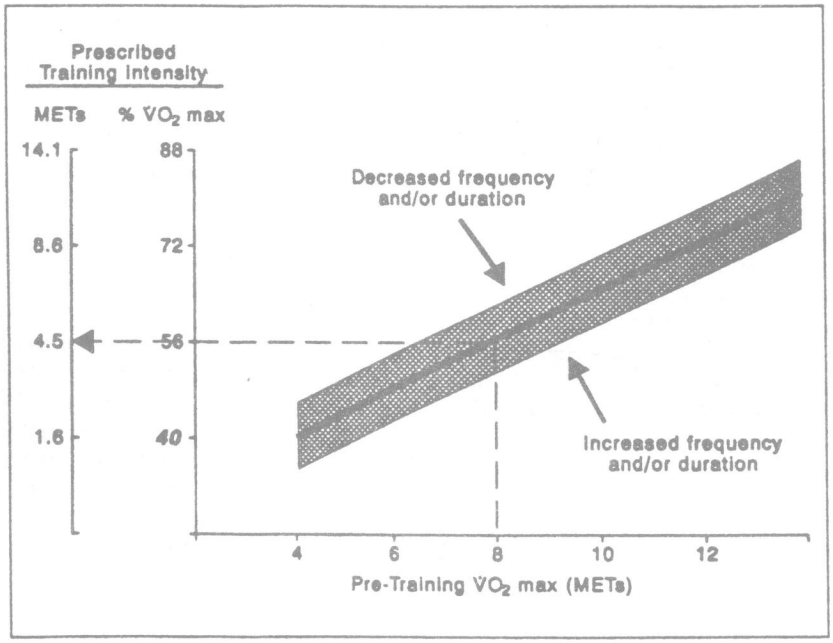

FIGURE 3. Theoretical relation between aerobic capacity (METs) and the minimal intensity for exercise training, expressed as a percentage of the maximal oxygen uptake ($\dot{V}O_2$ max). The threshold intensity for training increases in direct proportion to $\dot{V}O_2$ max before training; however, it can be modulated by altering the exercise duration or frequency, or both. For example, a patient with a peak capacity of 8 METs would exercise at approximately 56% of his $\dot{V}O_2$ max, or 4.5 ± 0.5 METs, to further increase his functional capacity.

(41%); and no training, from 7.0 to 9.4 METs (34%). The investigators concluded that formal exercise training soon after clinically uncomplicated myocardial infarction may not be required to restore aerobic capacity "to values approximating those of sedentary men of similar age without heart disease."

THRESHOLDS FOR TRAINING: For most deconditioned adults and patients with CAD, the threshold intensity for exercise training probably lies between 40 and 60% of the $\dot{V}O_2max$.[15] Improvement in aerobic capacity with low to moderate training intensities suggests that the interrelation among the training intensity, frequency and duration may permit a decrease in the intensity to be partially or totally compensated for by increases in the exercise duration or frequency, or both (Figure 3). Moreover, recent studies have shown similar training effects in subjects who completed three 10-minute bouts of moderate intensity exercise per day versus those who performed one "long" exercise bout of 30 minutes, 5 days per week for 8 weeks.[16] Thus, relative increases in functional capacity appear to depend more on the subject's initial fitness and the total amount of exercise accomplished or calories expended, than on the specific exercise frequency, intensity or duration.

TRAINING ACTIVITIES: VALUE OF WALKING: The most effective training activities employ large muscle groups, are maintained continuously, and are rhythmic and aerobic in nature; examples include walking and jogging, swimming, and mobile or stationary cycle ergometry. Walking is the most facile and easily regulated exercise for cardiorespiratory conditioning. Extremely slow walking (<2 miles per hour) approximates 2 METs and may impose metabolic loads sufficient for exercise training in deconditioned subjects.[17] Moreover, fast walking provides an intense enough activity to increase aerobic capacity and decrease body weight and fat stores in sedentary, middle-aged men.[18] In 1 study of 343 healthy adults (165 men, 178 women), 67% of men and 91% of women achieved a training heart rate (defined as ≥70% of maximal heart rate) when asked to "walk" a mile briskly.[19] In men >50 years of age, 83% achieved a training heart rate. These findings suggest that many patients with CAD can improve cardiorespiratory fitness by walking alone.

Cardiac function: The effects of chronic exercise training on the autonomic nervous system act to reduce myocardial demands at rest and during exercise. Vagal tone appears to be increased at rest, whereas sympathoadrenergic drive (circulating catecholamines, particularly norepinephrine) is decreased during exercise.[20] The result is a reduction in the heart rate-blood pressure product at any given oxygen uptake or submaximal work load, even when low to moderate exercise training intensities are used.[21]

The effects of physical conditioning on myocardial perfusion and ejection fraction are less clear. It has been suggested that a higher heart rate-blood pressure product at the onset of angina or ischemic ST-segment depression, or both, may reflect an improved myocardial oxygen supply. Some investigators have used thallium-201 exercise testing and multiple-gated image acquisition scans on subjects before and after training programs to assess changes in cardiac function. Although the findings have been contradictory, improvements have been reported both with and without vigorous exercise training regimens. In contrast, angiographic studies in group exercise trials have, without exception, yielded disappointing results.[22]

Ehsani et al[23] showed that prolonged and intense medically supervised endurance exercise training can, in selected cardiac patients, result in a higher heart rate-blood pressure product at the onset of ischemic ST-segment depression (≥0.1 mV), implying an increase in the delivery of oxygen to the myocardium. This regimen (80 to 90% of $\dot{V}O_2max$ for 1 hour, 4 to 5 sessions per week for 1 year) also resulted in improvements in stroke volume and ejection fraction, suggesting a direct cardiac benefit in some, but not all patients.[24] More recently, Schuler et al[25] studied the effects of a low-fat/low-cholesterol diet and regular, high-intensity exercise in patients with CAD. After 1 year, patients demonstrated less exercise-induced myocardial ischemia by scintigraphic criteria, despite an increased peak heart rate-blood pressure product. Similar benefits, however, have

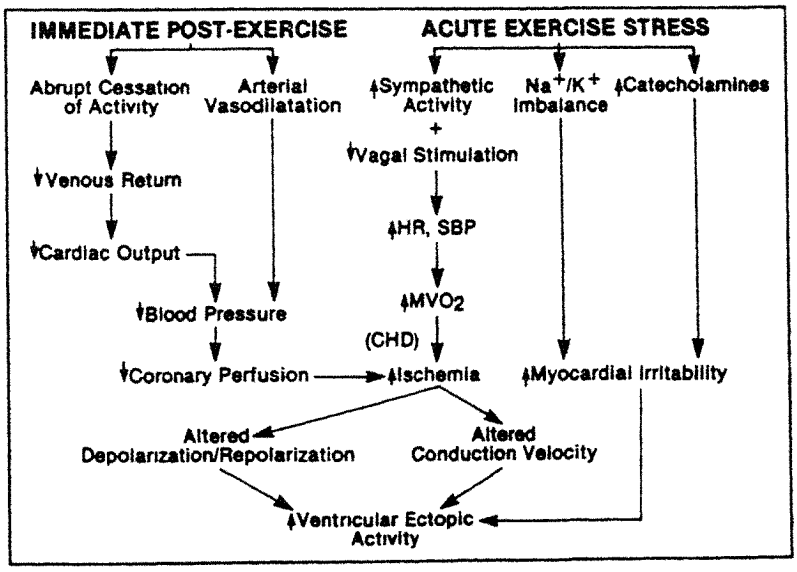

FIGURE 4. Physiologic alterations accompanying acute exercise and recovery, and their possible sequelae. CHD = coronary heart disease; HR = heart rate; MVO_2 = myocardial oxygen uptake; NA^+/K^+ = sodium/potassium ion; SBP = systolic blood pressure. (Reprinted with permission from *J Cardiac Rehabil.*[29])

been demonstrated with more moderate exercise training regimens. For example, Laslett et al[26] reported an increase in the heart rate-blood pressure product at the onset of ischemic ST-segment depression in exercise-trained patients with CAD. Froelicher et al[27] used thallium-201 exercise testing and multiple-gated image acquisition analysis scans on subjects before and after a combined arm-leg training program that lasted 3 to 12 months. Six of 16 patients showed improvement in left ventricular ejection fraction or exercise thallium images, or both, after training. Of the remaining 10 patients, 2 had images that worsened and 8 showed no change.

SAFETY OF HIGH-INTENSITY TRAINING REGIMENS: The incidence of cardiovascular complications during exercise is considerably greater among cardiac patients than among presumably healthy adults. Cobb and Weaver[28] emphasized that cardiac arrest associated with exercise testing and training in patients with CAD is typically a primary arrhythmic event not due to acute myocardial infarction. The additional risk of cardiac arrest during exercise, compared with that at other times, may be >100-fold during or soon after vigorous physical exertion.

By increasing myocardial oxygen demands and simultaneously shortening diastole, and thus coronary perfusion time, exercise may evoke a transient oxygen deficit in the subendocardial tissue, which can be exacerbated by a decreased venous return secondary to abrupt cessation of activity. Ischemia can alter repolarization, depolarization and conduction velocity, triggering serious ventricular arrhythmias which, in extreme cases, may be the harbingers of ventricular tachycardia or fibrillation, or both. Furthermore, intracellular sodium-potassium imbalance, catecholamine excess and increased sympathetic outflow may be arrhythmogenic (Figure 4).[29]

The safety of high-intensity exercise training regimens has been challenged in retrospective reports of patients with CAD who developed cardiac arrest or ventricular fibrillation during or shortly after medically supervised rehabilitation exercise.[30,31] Hossack and Hartwig[31] identified patients at increased risk of untoward events as those having a markedly ischemic exercise electrocardiogram, an above-average functional capacity, or a record of poor compliance to the prescribed training heart rate range (i.e., exercise intensity violators). These and other recent data[32] suggest that unconventionally vigorous exercise is associated with an increased risk of cardiovascular complications in patients with CAD.

Coronary risk factors: Aerobic exercise training programs can result in moderate losses in body weight, moderate to large losses in body fat, and small to moderate increases in lean body weight. Endurance exercise can also promote decreases in blood pressure (particularly among hypertensives),[33] total blood cholesterol, serum triglycerides, and low-density lipoprotein (LDL) cholesterol, and increases in the "antiatherogenic" HDL cholesterol subfraction.[34]

OBESITY: Exercise programs to reduce body weight and fat stores should be performed at least 3 days/week and include sustained activity of an endurance nature to promote a caloric expenditure of 300 to 500 kcal per session.[15] For many patients this may be best accomplished by regular walking over an extended duration. Exercise rated between "fairly light" and "somewhat hard" is generally appropriate for weight reduction programs, corresponding to 60 to 70% of the maximal heart rate, which is equivalent to 40 to 60% $\dot{V}O_2$max. During this relative intensity, free fatty acids are used preferentially as a fuel source, while blood lactate levels generally remain low, allowing the individual to exercise for sustained periods.

TABLE II Effects of Exercise on Total and Low-Density Lipoprotein Cholesterol with Body Weight Changes

Body Weight	Total Cholesterol (mg/dl)	LDL Cholesterol (mg/dl)
→	−7*	−3*
↓	−13*	−11*
↑	+3	+3

*$p < 0.05$
Adapted from *JAMA* [38]
LDL = low-density lipoprotein, → = no change, ↓ = decrease ↑ = increase.

HYPERTENSION: A meta-analysis of 25 relevant studies over the last 20 years appears to strengthen the case for moderate-intensity exercise training in the treatment of mild hypertension.[35] The studies involved men between 15 and 70 years of age and the training programs varied from 1 to 12 months in duration. Approximately two-thirds of the experimental groups in these studies demonstrated statistically significant decreases in blood pressure, averaging about 10 mm Hg for both systolic and diastolic blood pressure. Moderate-intensity training, corresponding to 40 to 60% $\dot{V}O_2$max, seemed to be just as effective as high-intensity training, and possibly even more so. Recent studies suggest that moderate aerobic exercise should not be considered a replacement for pharmacologic therapy in patients with mild hypertension.[36]

BLOOD LIPIDS AND LIPOPROTEINS: The results of 66 exercise studies, statistically aggregated using meta-analysis, showed that physical training produced modest changes in blood lipids and lipoproteins.[37] In the average exercising subject, total cholesterol decreased 10 mg/dl ($p < 0.01$), total triglycerides decreased by 16 mg/dl ($p < 0.01$), HDL cholesterol increased by 1 mg/dl (p = not significant), LDL cholesterol decreased by 5 mg/dl ($p < 0.05$), and the total/HDL cholesterol ratio decreased by 0.5 ($p < 0.01$). None of the changes for the control groups were significant. Higher initial levels of total cholesterol, total triglyceride, and total/HDL cholesterol ratio, and lower initial levels of HDL cholesterol, resulted in greater postconditioning decreases and increases, respectively. Although total cholesterol decreased as the exercise duration (hours) increased, lower training intensities were associated with more beneficial changes in lipids and lipoproteins.

As a follow-up to this analyses, data from 95 exercise studies were partitioned into those in which subjects gained, maintained, or lost body weight.[38] When body weight decreased or remained unchanged, total cholesterol and LDL cholesterol levels decreased significantly

TABLE III Cardiac Rehabilitation Trial Results

Trials	No. of Pts Randomized		Entry after MI (mos)	Length of Follow-Up (mos)	Deaths (no.)		Mortality (%)		Effectiveness (%)
	Control	Intervention			Control	Intervention	Control	Intervention	
Sanne[47]	157	158	3	48	35	28	23	18	22
Hakkila[48]	81	77	On hospital discharge	24	10	8	12	10	15
Kentala[49]	146	152	2	12	32	26	22	17	22
Palatsi[50]	200	180	2	29	28	18	14	10	29
Kallio[51]	187	188	1	36	56	41	30	22	27
Shaw[52]	328	323	2–12	30–54	24	15	7	5	37
Shephard[53]	354	379	12	48	26	36	7	10	−30
Roman[54]	100	93	2	108	27	16	6	4*	38

*Mortality expressed in %/year of patients followed up
MI = myocardial infarction

(Table II). However, when body weight increased, cholesterol and LDL cholesterol levels increased. These results suggest that exercise-mediated reductions in cholesterol and LDL cholesterol are greatest when concomitant body weight losses occur.

Although cross-sectional and longitudinal comparisons have shown that vigorous physical activity is associated with an increased HDL cholesterol, recent studies indicate that chronic low-intensity, long duration physical activity (measured in miles walked per day) may play an important role in increasing both the HDL cholesterol and HDL_2 cholesterol.[39] It has been suggested that approximately 1,000 kcal per week of additional energy expenditure, equivalent to walking or running 8 to 10 miles, may represent the threshold required to increase HDL cholesterol.[40] However, prospective studies in cardiac patients have shown that increases in HDL cholesterol may be achieved at an even lower exercise dosage.[34,41]

Psychosocial well-being: Another rationale for exercise training of patients with CAD is that it improves psychosocial functioning. Anecdotal reports and uncontrolled studies frequently describe improvements in psychological well-being in relation to exercise-based cardiac rehabilitation programs. Subjective comments of improvement after exercise training often include an increase in self-confidence, particularly in relation to exertional tasks, an improved sense of well-being, decreased depression and hypochondriasis levels, reduced tension and fatigue, better sleep, and improved sexual relations. However, it remains unclear to what extent these changes reflect the exercise program, per se, or "spontaneous psychological recovery" from the acute cardiac event. In the few randomized, controlled studies reported,[42–44] exercise training had little or no effect on psychosocial functioning.

Stern and Cleary[42,45] reported results from the National Exercise and Heart Disease Project. Six hundred fifty-one men, aged 30 to 64 years, who had at least 1 myocardial infarction 8 weeks to 36 months earlier, completed a 6-week low-level exercise program in which intensity was limited to a maximum of 72% of age-predicted heart rate. Although this initial program was designed, in part, to help identify potential dropouts, it resulted in positive psychosocial, sexual and vocational changes.[45] After the prerandomization phase, compliers were randomly assigned to a moderate to high intensity supervised exercise program 3 days/week or to a nonexercising control group. No differences were noted between the groups at the 6-month, 1- and 2-year follow-up examinations.[42]

Blumenthal et al[46] compared the effects of 12 weeks of moderate to high intensity (65 to 75% $\dot{V}O_2max$) versus low-intensity (<45% $\dot{V}O_2max$) exercise on psychosocial functioning in 70 patients with a recent (<1 year) myocardial infarction. No differences were found between groups in any measure of psychosocial functioning, including anxiety, depression, stress, anger and type A behavior. The investigators suggested that group therapy or stress management may be more effective than exercise in reducing the psychological dysfunction complicating acute myocardial infarction.

Morbidity and mortality: Eight of the major randomized trials of rehabilitation with exercise after myocardial infarction[47–54] (Table III) involved 3,103 patients who were followed for 1 to 9 years. Effectiveness of these interventions, as expressed by the formula: [(control mortality-intervention mortality/control mortality) × 100] showed a beneficial trend toward increased survival (7 of 8 trials), but only one study[51] attained statistical significance. Because these studies were limited by sample size, recent attempts have been made to pool data from similar randomized clinical trials. Two meta-analyses have now shown that cardiac rehabilitation provides a 20 to 25% reduction in total and cardiovascular-related mortality; however, neither report found a significant decrease in the rate of nonfatal recurrent myocardial infarction.[55,56] The exercise dosage administered in these studies was only 50 to 75% $\dot{V}O_2max$ for 20 to 60 minutes 2 to 4 times per week, except for 1 of the studies[50] in which daily home exercises were scheduled. Although concomitant life-style changes, enhanced medical surveillance, or both, may have contributed to the survival advantage, the importance of these data is substantial. The reduction in fatal cardiac events after exercise-based cardiac rehabilitation is similar to that demonstrated with other, more expensive therapies including coronary artery bypass surgery and other medical regimens (β blockers, lipid-lowering agents and antihypertensive treatments). Current thrombolytic and revascularization procedures, which markedly decrease early postinfarction mortality,

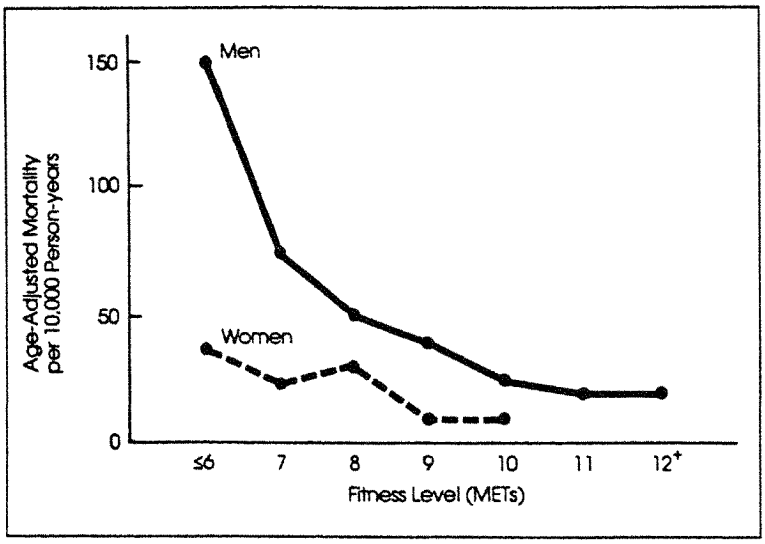

FIGURE 5. Age-adjusted, all-cause mortality rates per 10,000 person-years of follow-up by physical fitness (METs) achieved during maximal treadmill exercise testing. (Adapted from Blair et al.[58])

would likely diminish the impact of adjunctive cardiac rehabilitation programs on survival.[57]

RELATION BETWEEN FITNESS AND ALL-CAUSE MORTALITY: Blair et al[58] identified a low level of aerobic fitness as an independent risk factor for all-cause mortality. The investigators prospectively studied 10,224 men and 3,120 women who were given a preventive medical examination and a maximal treadmill exercise test to assess their $\dot{V}O_2max$. Over an average follow-up of slightly >8 years, 240 men and 43 women died. In general, the higher the initial level of fitness, the lower the subsequent mortality rate from cancer and heart disease (Figure 5), even after statistical adjustments were made for age, coronary risk factors, family history of heart disease, and follow-up interval. However, there appeared to be no additional benefit (i.e., lower mortality) associated with fitness levels higher than 9 METs in women and 10 METs in men. Moreover, the greatest reduction in risk for both men and women occurred with progression from the lowest fitness category (≤6 METs) to the next lowest level (7 METs), suggesting that even a modest improvement in fitness among the most unfit confers a substantial protective benefit. These findings and other recent analyses[2,59] suggest that the optimal amount of energy expenditure for reducing heart disease mortality can be accomplished by a brisk walk of 30 to 70 minutes each day. This is encouraging from a preventive medicine and public health standpoint, since most people should be able to schedule this amount of leisure-time physical activity into their daily routine.

Conclusion: Low to moderate intensity exercise training can produce beneficial changes in functional capacity, cardiac function, coronary risk factors, psychosocial well-being and possibly improve survival in patients with CAD. These findings may be especially relevant for the inactive patient in whom the subjective discomfort of vigorous physical training may serve as a deterrent to long-term compliance with exercise therapy. Unless future studies can show that the added benefits of high-intensity exercise outweigh the potential risks, it appears prudent to recommend more moderate exercise regimens for patients with CAD. We must conclude, as Hippocrates[60] summarized it, that:

> ". . . all parts of the body which have a function, if used in *moderation* and exercised in labours in which each is accustomed, become thereby healthy, well-developed and age more slowly, but if unused and left idle they become liable to disease, defective in growth, and age quickly."

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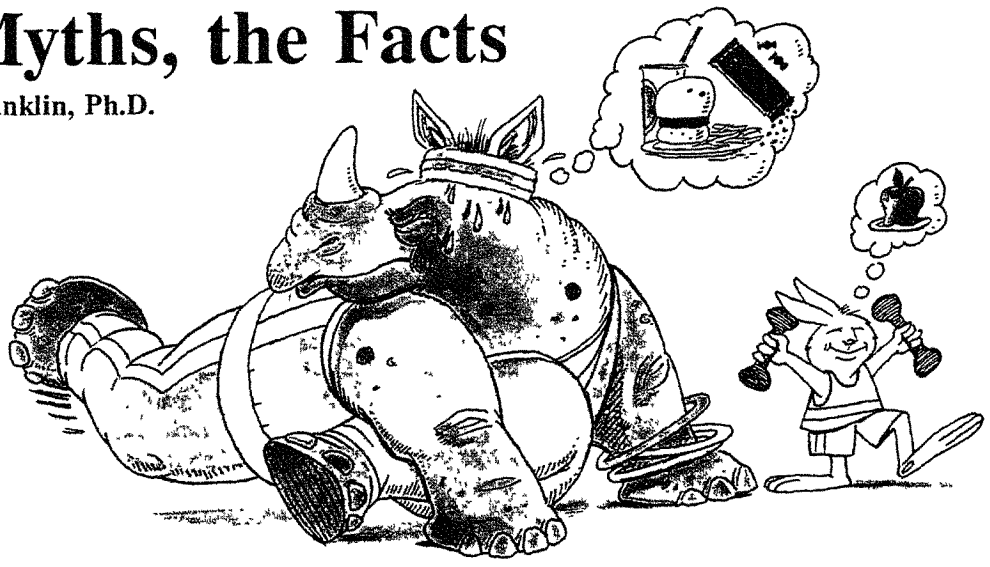
Exercise and Weight Loss: The Myths, the Facts

by Barry A. Franklin, Ph.D.



Approximately 25% of . . . men and 40% of women . . . reported that they were currently trying to lose weight. . . . The average man wanted to lose 30 pounds and to weigh 178 pounds; the average woman wanted to lose 31 pounds and to weigh 133 pounds.

—*American Journal of Public Health,* September 1992

I can't exercise because I'm too fat. . . . Exercising makes me ravenous and then I eat too much. . . . I'd look ridiculous in a jogging suit. . . . I'm too tired to exercise.

—arguments against exercise

Obesity is a serious medical concern for a large percentage of the population. Being overweight has been directly or indirectly linked to several chronic health problems, including coronary heart disease, diabetes, certain types of cancer, osteoporosis, and gallbladder disease. Consequently, weight reduction is often prescribed in the prevention and medical treatment of many cardiorespiratory, metabolic, and musculoskeletal conditions.

The simplistic views that obesity is due solely to overeating or that it can be effectively treated through caloric restriction are not valid. While it is true that some obese persons eat prodigious amounts of food, many eat less than their leaner counterparts. What often distinguishes those who are significantly overweight from their leaner counterparts is that they are far less physically active.

The role of physical activity in weight-reduction programs has generally been minimized, particularly when compared with dietary intervention. However, exercise, with or without caloric restriction, offers several important advantages over caloric restriction alone. Regular physical activity improves musculoskeletal and cardiorespiratory fitness. Moreover, many forms of exercise offer enjoyable leisure-time activities, whereas dieting for most people is difficult and unpleasant. Finally, weight lost through exercise consists primarily of fat loss, whereas dieting (especially crash or fad dieting) is more likely to lead to the loss of water and vital lean tissue.

Unfortunately, overweight people are often confronted with myths and misconceptions regarding the role of exercise in reducing body weight and fat stores. They may also be easily deceived by an overwhelming number of gimmicks, gadgets, and fads that are promoted as "miracle" methods of reducing—few of which are legitimate and many of which may be dangerous.

Caloric cost of exercise: many theories

The effectiveness of exercise in the control of body weight has been discounted by many who claim the amount of activity required for caloric output to be significantly affected is prohibitive. To support this, they often point to energy equivalent tables that present the rates of caloric expenditure for various physical activities (usually in kilocalories burned per minute per kilogram of body weight). On the basis of such tables, it would seem that to work off one pound (0.454 kg) of fat, one would have to walk for 14 hours or split wood for 7 hours or play volleyball for 11 hours.

Exercise proponents, however, may argue that the calorie-expending effects of physical activity do not depend on a single bout of exercise but are cumulative. If one chops wood for one 20-minute period a day or walks for 40 minutes a day, one could lose a pound of fat in 21 days and reduce body weight by about 7.7 kg (17 lb) in a year. Still others would

counter that the true caloric effect of exercise must consider the "net energy cost" of each exercise bout (calculated as the total energy cost of the exercise minus the energy cost of normal activity during the same time period). For most individuals such a net cost is about two kilocalories per minute less than the gross cost. Thus, if one walks 60 minutes and expends 300 kcal, the net cost of the exercise is 180 kcal, not 300 kcal. Because a pound of fat equals 3,500 kcal and losing weight requires a negative caloric balance, it is apparent that if net energy expenditure alone is considered, a substantial amount of exercise is required for facilitating a significant weight loss.

Fortunately, the caloric expenditure associated with exercise is *not* limited to the time of the activity alone. It is well documented that in the postexercise recovery period more oxygen is consumed than is normally required for sustaining resting metabolism. Because each liter of oxygen consumed is equivalent to approximately five kilocalories, this results in an additional energy expenditure subsequent to the cessation of physical activity. The delayed postexercise return of oxygen consumption to resting levels has been attributed to numerous factors, including the biochemical replenishment of adenosine triphosphate, which serves as the "fuel" for all the energy-requiring processes within the body's cells; augmented cardiopulmonary metabolism; increased body core temperature; and elevation of certain hormones, especially epinephrine and norepinephrine.

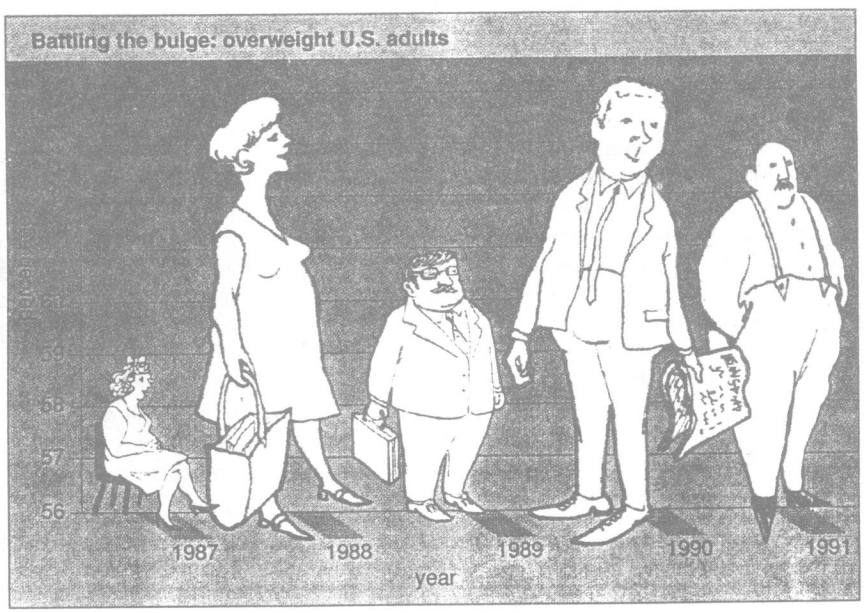
Physiologists have quite clearly demonstrated that exercise *is* beneficial in the control of body weight. This is so not only for the energy losses incurred during the exercise but because a considerable additional caloric expenditure occurs following exercise.

Walking versus running versus bicycle riding

It is worthwhile to consider the comparative energy expended by three common forms of exercise. The caloric cost of walking as opposed to running has been the subject of considerable controversy. Some claim that the longer exercise duration involved in walking a given distance results in approximately the same caloric expenditure as running that same distance over a shorter period of time. The laws of physics would appear to support this hypothesis in that a given weight moves a given distance by both methods.

On the other hand, numerous studies have demonstrated that running a given distance expends more calories than does walking the same distance. The gross caloric costs of walking and running are approximately 1.15 kcal per kilogram of body weight per mile (1 mi = 1.61 km) and 1.7 kcal/kg/mi, respectively. Unless an individual walks at a very slow pace or runs at an extremely fast pace, the caloric cost for a given distance is relatively independent of speed. Although many obese persons may be unable to run, a substantial energy expenditure can result if they walk a longer distance.

Outdoor bicycling is an energy-efficient method of covering distance. Although the energy cost of bicycle riding varies with the type of bicycle, the mechanical efficiency of the rider, and his or her body weight, the gross caloric cost approximates 0.6 kcal/kg/mi. But again, as with walking and running, the caloric cost for a given distance is relatively independent of speed. The cyclist thus expends approximately one-half the kilocalories of the brisk walker and one-third those of the runner covering the same distance. Expressed another way, the energy cost of bicycling three miles



is the approximate equivalent of walking one and a half miles or running one mile. The table provides a comparison of the gross caloric expenditure per mile at various body weights for walking, running, and outdoor cycling.

Calories burned: running, walking, cycling

body weight		kcal/mi*		
(lb)	(kg)	running	walking	bicycling (outdoors)
110	50	85	58	30
132	60	102	69	36
154	70	119	81	42
176	80	136	92	48
198	90	153	104	54
220	100	170	115	60
242	110	187	127	66
264	120	204	138	72

*one mile = 1.61 km

Exercise: effects on appetite

A common excuse for not exercising is that physical activity always increases appetite and caloric intake, negating the energy expenditure of the exercise itself. Although it is true that an increase in food intake generally parallels an increase in physical activity, this relationship appears to hold only within a certain activity zone.

In a classic experiment conducted several decades ago, researchers demonstrated that at extremely high or low levels of daily energy expenditure, appetite no longer worked as a guide to balancing food intake and the amount of energy used. Rats that were exercised daily for up to one hour decreased their food intake and body weight in comparison with sedentary control animals. When the exercise duration was increased beyond one hour, food intake increased but only to the extent that body weight was maintained. At exhaustive levels of exercise (*i.e.,* six hours a day), both food intake and body weight decreased.

More recently, investigators measured caloric intake and physical activity in industrial workers. Five different occupational activity categories were determined: sedentary, light work, medium work, heavy work, and very heavy work. The researchers found that there was a linear relationship between activity level and caloric intake in those in the middle three categories (the light-to-heavy activity range), but those in the sedentary range tended to weigh more and consumed a greater number of calories, while those engaged in very heavy work consumed more but weighed less. The results thus substantiated the earlier findings in animals.

In humans most research indicates either no change in food intake with moderate exercise of extended duration or slight decreases with vigorous exercise of shorter duration. This latter effect (appetite suppression) is attributed, at least in part, to the increased level of catecholamines evoked by strenuous exercise. Catecholamines include adrenal hormones (*e.g.,* epinephrine and norepinephrine) that may have a role in appetite-control mechanisms.

Shrink those hips: the myth of spot reduction

In the U.S., where the health and aesthetic disadvantages of excess adiposity are well recognized, "spot reducing" is a multimillion-dollar industry, with health spas and personal trainers promising that people can "take inches off the waist, thighs, or buttocks—without dieting and in just minutes a day." The concept is based on the belief that it is possible to selectively "burn off" fat from a particular part of the body by exercising that body area. However, considerable research casts doubt on the validity of spot reduction.

In the early 1970s researchers first tested the proposition that one can selectively reduce fat in one part of the body. They compared the circumferences and amounts of subcutaneous fat in both arms of accomplished tennis players. It was hypothesized that if exercise of a particular body part selectively reduced fat tissue in that area, then the racquet (playing) arm should have less fat than the inactive (nonplaying) arm. Although circumference measures in the playing arm were greater than in the nonplaying arm (owing to muscular hypertrophy), measures of skin-fold thickness revealed no difference between the two arms in subcutaneous fat deposits.

Perhaps the most convincing evidence against spot reduction comes from more recent studies of the effects of localized abdominal exercise training on regional fat, or adipose cell size. Fat biopsies were taken from the abdomen, buttocks, and upper back in 13 male subjects before and after a rigorous 27-day abdominal exercise (sit-ups) training program. Fat-cell diameter decreased significantly at all three sites, with no significant differences in the rate of change between sites. Although conventional sit-ups are commonly promoted as a way to reduce abdominal fat, this experiment demonstrated that such exercise does not preferentially reduce the amount of subcutaneous fat in the abdominal region alone.

The physiological explanation behind spot reduction is that exercising a muscle causes fatty acids (a primary energy source in muscular exercise, particularly exercise of mild to moderate intensity) to diffuse into the muscle in the "target" area from its overlying fat pad. Instead, it appears that fatty acids from adipose tissue stores *throughout the body* are mobilized during exercise to supply the needed energy fuels. Perhaps one reason why spot-reducing exer-

cise sometimes appears to work is that if the exercise intensity is sufficiently strenuous and the exercise duration long enough, fat from the entire body, including the target area, will be reduced.

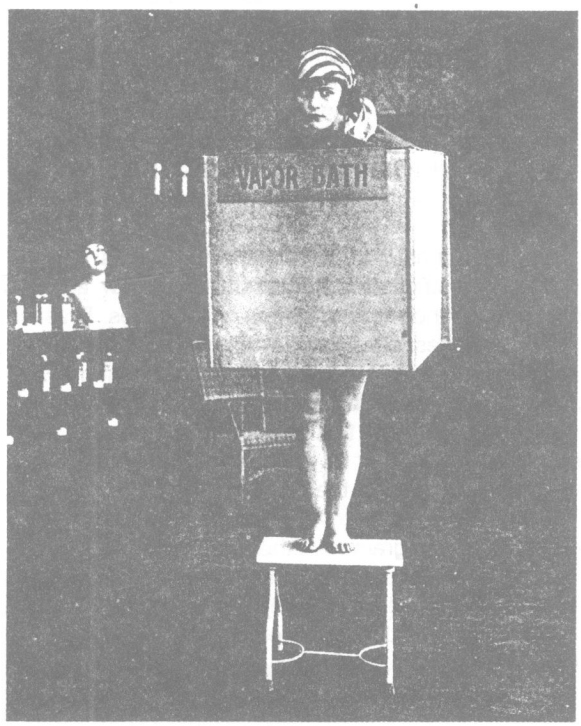
Effortless exercise

Passive exercise that "does the work for you" has been touted as an effective way to reduce or redistribute body fat. Do these approaches have any merit?

Let a machine do the work. Several years ago researchers investigated the validity of the weight-reducing claims made for the mechanical vibrating machines commonly found in health clubs and gymnasiums. It was hypothesized that if the devices oxidized or "massaged away" body fat, as their promoters say they do, then the individual's oxygen uptake and/or blood fat levels should be increased during the "workout."

Thirteen men, some considerably overweight, were subjected to a 15-minute period of abdominal vibration. During and after the exercise bout, oxygen consumption was measured (as a reflection of caloric cost). In addition, venous blood samples were drawn before and shortly after the exercise and again several hours later. Results showed that blood fats remained essentially unchanged as a result of the vibration. Average caloric cost of the 15-minute exercise session, including the postvibration recovery period, was only 11.4 kcal more than each man would have expended had he remained just sitting for an equivalent period of time. This "net cost" represents approximately 1/19 of an ounce of fat. Thus, losing a pound of fat (3,500 kcal) would require 307 such 15-minute periods of abdominal vibration, or roughly six exercise sessions per week for a year! The investigators concluded that "the vibrator is not to be taken seriously as a device to assist in fat reduction or shifting of fat deposits within the body." Such devices are, at best, capable of moving fatty tissue, not *re*moving it.

The desire to melt away fat effortlessly is not new; this "vapor bath" contraption from earlier times differs little from the rubberized "sweat" suits and heated belts on the market today. The temporary dehydration that occurs only creates the illusion *of fat loss—and can be dangerous.*



Culver Pictures

Sweat it off. Special weight-reducing exercise garments, including heated belts and rubberized suits, are semipermeable or impermeable to moisture, and their promoters claim that by wearing them one can lose weight more rapidly—through increased sweating but without increasing exercise intensity or activity level. In other words, the clothing does all the work.

Most such garments promote excessive dehydration (loss of body water) by heat, localized pressure, or tissue compression. Although circumference measures of various body areas (*e.g.,* waist, thighs, upper arms) as well as scale weight may in fact decrease until rehydration occurs, these losses are unrelated to any actual or lasting reductions in body weight or fat stores. Nevertheless, manufacturers capitalize on the short-lived, or apparent, losses to highlight the supposed benefits of their products.

In fact, impermeable or semipermeable exercise clothing acts to increase water losses and restrict evaporative cooling. The loss of water causes a reduction in effective circulating blood volume and a subsequent drop in blood pressure. As a result, the heart rate increases disproportionately to keep up with increasing metabolic demands during exercise. Rubberized sweat suits worn during exercise trap sweat between the suit and the skin. Trapped sweat cannot evaporate to cool the body, and convective heat loss is also inhibited, thus depriving the body of its normal mechanisms for cooling. The increased metabolic rate from exercising, coupled with the added burden imposed on the body's temperature-regulating mechanism, may result in a severe rise in body core temperature and potentially serious heat-related disorders.

The ideal garment for reduction of body fat would be clothing that promotes energy expenditure, caloric restriction, or both. Unfortunately, no material has yet been found that effects these necessary lifestyle changes. It is not surprising that some manufacturers have modified their advertising to include the recommendation (in small print, of course) that a diet or exercise program accompany wearing of their garment to maximize its effectiveness.

Reduce horizontally. The late Robert M. Hutchins, former chancellor of the University of Chicago and

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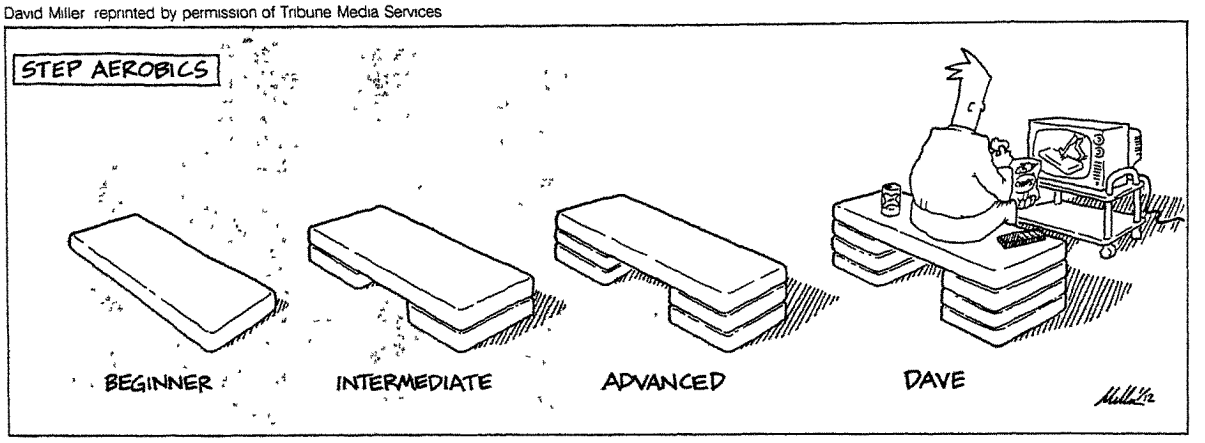

chairman of the Board of Editors of *Encyclopædia Britannica,* once said: "Whenever I feel like exercise, I lie down until the feeling goes away." Promoters of some electrical muscle stimulators (EMS) would have people believe that such behavior can actually result in a workout. Advertisements in supermarket tabloids tout these devices for everything from weight loss to body shaping and toning to spot reducing to the removal of cellulite (dimpled fat that often appears on the hips, thighs, and buttocks). For example, ads claim that "microelectroimpulses" provide the same figure-toning results as "3,000 sit-ups without moving an inch" or "10 miles of jogging while lying flat on your back."

Unlike many gimmicks, EMS units have a legitimate basis. They are often used by physical therapists in the treatment of certain medical conditions—*e.g.,* decreasing pain and swelling, enhancing strength in atrophied muscles, promoting healing after certain injuries or some kinds of surgery, and relaxing muscle spasms. These purposes are a far cry from the sensational claims for EMS products for effortless weight loss or body shaping. The only reduction emanating from advertised EMS devices occurs in the consumer's billfold.

The Food and Drug Administration, the federal agency that regulates the marketing of "medical devices" and evaluates their safety and effectiveness, considers muscle stimulators that are promoted or used for "body shaping and contouring" to be misbranded and fraudulent. Accordingly, the agency has recently banned the sale and distribution of many devices whose manufacturers have failed to substantiate their advertisements.

In addition to the deceptive claims, EMS devices can be dangerous if improperly used. There have been numerous reports of electric shocks and burns resulting from the products, and they can be especially hazardous to pregnant women, patients with cardiac pacemakers, and people who have epilepsy. Like most other effortless-exercise products, EMS units, at the very least, may be worthless; on the other hand, they can be quite dangerous.

To exercise or not to exercise?

While it is doubtful that long-term compliance with any sort of exercise program will be easy for the dedicated "couch potato," exercise (alone or in combination with caloric restriction) is an important, and probably the best, way to achieve lasting and meaningful weight loss. The balance between intensity and duration of exercise should be regulated to yield a high total caloric expenditure, approximating 300 to 500 kcal per exercise session, amounting to 1,000 to 2,000 or more kcal per week. Exercise should be engaged in at least three days per week and should include sustained large muscle activities that are maintained continuously and are rhythmic and aerobic in nature. The exerciser needs to choose an appropriate form of exercise that is enjoyable and suitable for his or her lifestyle—an activity he or she will stick with. Reductions in body weight and fat stores can even result from brisk walking programs, provided that the walking duration is sufficient (exceeds 30 minutes several times a week).

Long-term treatment of obesity that relies chiefly on caloric restriction is successful in as few as 5% and probably no more than 20% of all cases. Drastic dietary changes often lead to recidivism and concomitant fluctuations in body weight—a phenomenon known as the "rhythm method of girth control." Recent studies suggest that long-term compliance with exercise therapy may be no better. Clearly, misinformation is a big part of the problem.

Sensible caloric restriction coupled with an enjoyable exercise program nonetheless offers the greatest potential for success. Until these simple tenets are realized, many will continue to embrace the opinion voiced so eloquently by Shakespeare's Hamlet, Prince of Denmark: "There's a divinity that shapes our ends" (Act V, scene 2).

Comparison of Stress Testing Modalities

Vic Froelicher, MD
Symposium 4:
Exercise and Heart Disease
5:05- 5:25 pm

Probability of Coronary Disease for Middle-Aged Males or Post Menopausal Females Pre/Post Any Non-Invasive Test

Chest Pain Character	Pre-Test	Post Abnormal Test	Post Normal Test
Typical Angina	90%	98%	75%
Atypical Angina	50%	90%	25%
Non-Angina	10%	45%	4%
None	2%	6%	<1%

Stress Tests

	▪ **Exercise**	▪ **Non-Exercise**
ECG	FP inferior leads, V5 most info	Inferior leads and elevation/reciprocal
WHO?	METs/SBP for prognosis in lower risk patients	Patients who cannot exercise (PVD, CVA, Para)
IF Exercise Test Normal?	More sensitive add-on or ...	Patients who give inadequate effort

Mechanisms:
Add-ons/Non-Exercise Stressors

- **Persantine/Adenosine-Thallium/isonitriles**
  - perfusion, coronary steal
- **Dobutamine/Arbutamine-Echocardiography**
  - wall motion abnormality, increased myocardial oxygen demand
- **Exercise Echo/CKG/Apex**
  - wall motion abnormalities in recovery
- **Supine Bike-Echo/RN Ventriculography**
  - increased myocardial oxygen demand during exercise
  - wall motion and other abnormalities

Comparison of Diagnostic Tests

	Test (MI excld, WU bias)	(N)	Sens	Spec
Exercise Stress	ECG	147	68%	77%
	Thallium (planar/spect)	59	79%	87/74
	RNV	10	92%	65%
	ECHO	10	86%	86%
Non-Exercise Stress	Dobutamine Echo	5	88%	84%
	Persantine Thallium	11	85%	91%

Guyatt Rules for Believing an Evaluation of a Diagnostic Test

1. **Identification of Comparison Groups with One Group Free of Disease**
2. **Consecutive or Randomly Selected Patients for Whom the Diagnosis is in Doubt**
3. **Separate Analysis of Patients Likely to Have the Disease (ie, Post-MI patients)**
4. **Blind Comparison of the Test with a Reliable Standard**

..... If Believability Criteria met then the Test can be put into Practice

Work-up Bias

- In All Studies that Require the Patients to have a Cardiac Catheterization, the Patients are already selected by their physicians for the Cardiac Catheterization Using Clinical and Test Variables
- For the Selection of Variables from a Study to be Applicable to the Patient Presenting for a Work-Up, the Study must be Performed on an Un-selected Population
- Work-Up Bias can Only be Avoided by having the Patient Consent to Catheterization no Matter what the Test Results Are

Consensus Approach

- Standard Exercise ECG Test
- Published Multi-variate Equations
- Consensus Agreement with Thresholds for Low, Intermediate or High Risk for Any or Severe Angiographic CAD
- 90%/90% Sensitivity/Specificity
- See Abstracts

Trainability of Arms Versus Legs in Men with Previous Myocardial Infarction*

Barry A. Franklin, Ph.D.;† Lauren Vander, M.S.;
David Wrisley, M.D.;‡ and Melvyn Rubenfire, M.D., F.C.C.P.

(Chest 1994; 105: 262-64)

RPP = rate-pressure product

Cardiorespiratory and metabolic adaptations to exercise training appear to be largely specific to the muscle groups that have been trained. Limb-specific adaptations have been reported for submaximal exercise heart rate, blood lactate, and pulmonary ventilation.[1-3] These findings suggest that a substantial portion of the conditioning response derives from peripheral rather than central changes, including cellular and enzymatic adaptations that increase the oxidative capacity of chronically exercised skeletal muscle.[4]

The lack of interchangeability of training benefits from one set of limbs to another appears to discredit the general practice of limiting exercise training to the legs alone. Many recreational and occupational activities require arm work to a greater extent than leg work.[5] Although patients with coronary artery disease (CAD) are often advised to train their arms as well as their legs,[6-7] few data are available regarding the relative trainability of the upper extremities. Accordingly, we studied whether the upper extremities respond to aerobic exercise conditioning in the same qualitative and quantitative manner as do the lower extremities, when training intensity, frequency, and duration are equated.

Methods

Subjects

Our study population consisted of 13 men who had sustained a previous myocardial infarction. Four patients demonstrated electrocardiographic (ECG) changes consistent with anteroseptal infarction, one of combined anteroseptal and inferior infarction, and three each of either inferior or posterior infarction. Two patients had nondiagnostic ECGs. Eight of the 13 (62 percent) were receiving ß-blockers. Their mean (±SD) age was 57 ± 8 years; height, 176 ± 5 cm; and weight, 81 ± 6 kg. All subjects were participants in the Sinai Hospital of Detroit outpatient cardiac rehabilitation program (phase 2).

Arm and Leg Exercise Testing

Subjects reported to the laboratory for arm or leg exercise testing on two separate occasions. Leg tests were performed on a cycle ergometer (Schwinn model EX 2), and arm work was done on a modified version of the device, as previously described.[8] This ergometer is designed to be rate independent within a specified range (*ie*, power output and oxygen uptake are independent of the pedaling or cranking rate).

Protocol for the arm and leg tests was identical, consisting of progressive continuous exercise to volitional fatigue. The initial work load was 150 kpm/min, with increments of 150 kpm/min at each 3-min exercise stage. Maximum effort was determined to be the work load at which the established pedal speed (50 to 60 revolutions per minute) could no longer be maintained. Respiratory variables, heart rate, blood pressure (auscultatory method), and rate-pressure product (RPP) were determined at submaximal and maximal exercise. The ECG was monitored by oscilloscope, with three-channel (V_1, V_5, and a VF) recordings obtained throughout the exercise test and 12-lead ECGs recorded at the end of each exercise stage and during maximal exercise. The RPP was calculated as follows: ([heart rate × systolic blood pressure]/100).

Cardiopulmonary data were obtained by a specific system (Erich Jaeger Ergo-Pneumotest/Dataspir EDV 8 system). The gas analysis system includes a two-way (inspired-expired) low dead space breathing valve; oxygen and carbon dioxide analyzers using the fuel cell and infrared absorption principles, respectively; pneumotachographic volume measurement; and a computer assembly for on-line 30-s calculations, corrected to body temperature, atmospheric pressure saturated, or standard temperature and pressure, dry, of oxygen uptake (V_{CO_2}), expressed in liters per minute (liters·min^{-1}) or METs (1 MET = 3.5 ml·kg^{-1}·min^{-1}), minute ventilation, carbon dioxide production (V_{CO_2}), and respiratory exchange ratio (V_{CO_2}/V_{O_2}). Before each test, the pneumotachometer was referenced with a 1-L syringe, and the gas analyzers were calibrated with room air and a certified O_2/CO_2 concentration (Ohio Medical Products, Inc).

Electrocardiographic and heart rate determinations were made with a specific system (Marquette Computer-Assisted System) for exercise. The ECG was calibrated to 1 mV/10 mm deflection before each test.

All subjects repeated the arm and leg exercise tests after a physical training program.

Physical Training Program

The aerobic circuit training program included three exercise sessions per week for 6 weeks. Each session included a warm-up period (5 to 10 min), an endurance phase (30 min), and a cool-down. Training involved alternating upper and lower extremity exercise devices, 5-min at each of six stations, including treadmill walking,

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rowing, bench stepping, arm, leg, and arm-plus-leg ergometry. The same exercise training duration was used for the arms and legs, 15 min each.

Each patient's work rate (intensity) during the endurance phase was directed at achieving a minimum of 70 percent to a maximum of 85 percent of the peak heart rate achieved during his initial arm or leg exercise test. Actual intensity was verified by continuous ECG-telemetry monitoring. Participants were instructed to adjust their work rate to meet their prescribed intensity range.

Data Analyses

Statistical analyses included calculations of means, standard deviations (SD), and standard errors. Student's *t* test for paired observations was used to calculate differences between before and after physical conditioning. To assess relative trainability, the noncorrelated *t* test was used to compare the mean percentage change in selected cardiorespiratory variables during arm and leg exercise. Statistical methods were those outlined by Steel and Torrie.[9]

Results

All 13 subjects completed the preconditioning and postconditioning arm and leg tests without demonstrating significant ST-segment depression (≥ 1.0 mm horizontal or downsloping), serious ventricular arrhythmias, or abnormal blood pressure responses or symptoms. Thus, all exercise tests were terminated due to volitional fatigue. No serious cardiac or orthopedic complications occurred during the physical training program. The mean attendance for all participants was 92 percent (16.5/18 exercise sessions).

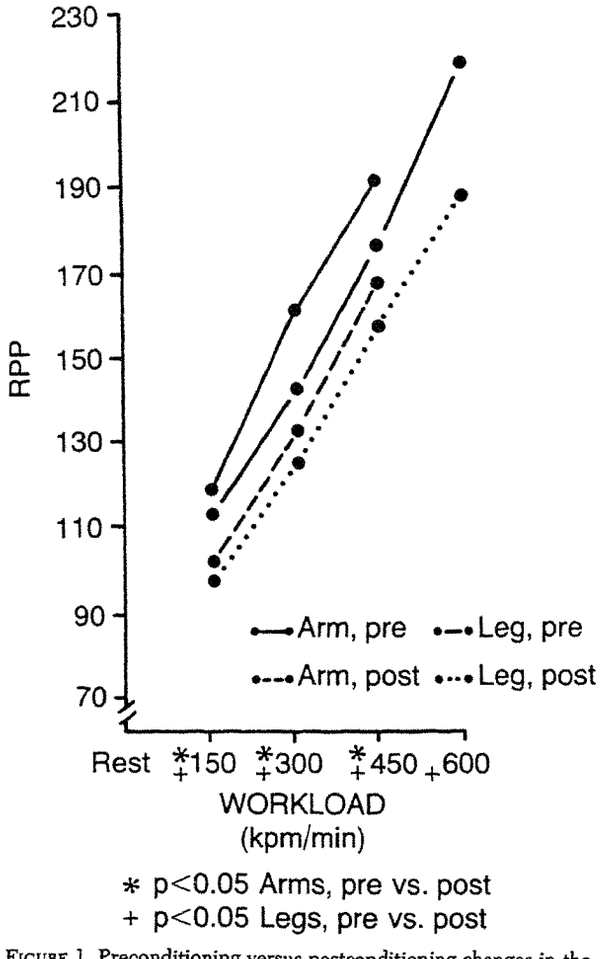

FIGURE 1. Preconditioning versus postconditioning changes in the mean rate-pressure product (RPP) during submaximal arm and leg exercise in men with previous myocardial infarction.

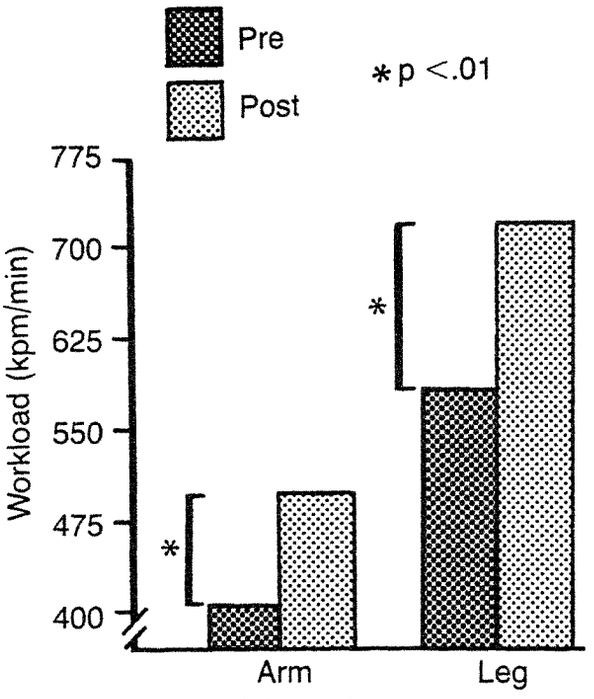

FIGURE 2. Average work loads (kpm/min) during maximum arm and leg exercise testing before and after training in men with previous myocardial infarction.

At a given submaximal work load, heart rate, systolic blood pressure, and RPP were slightly higher during arm work than during leg work. The postconditioning RPP was significantly decreased ($p<0.05$) during arm and leg exercise at work loads ≤600 kpm/min (Fig 1). Relative decreases in RPP at 150, 300, and 450 kpm/min were similar for arm and leg exercise: 14 versus 13 percent, 18 versus 13 percent, and 12 versus 11 percent, respectively.

Mean (± SD) peak work load during arm exercise testing increased from 404 (± 142) kpm/min to 500 (± 166) kpm/min. Average work load during maximum leg exercise testing increased from 583 (± 221) kpm/min to 715 (± 139) kpm/min (Fig 2). Thus, peak power output during arm and leg ergometry increased 24 and 23 percent, respectively. Maximal oxygen consumption (Vo_2 max) increased 13 percent during arm ergometry, from 4.6 to 5.2 METs, and 11 percent during leg ergometry, from 5.4 to 6.0 METs (Fig 3).

Discussion

This study evaluated the effectiveness of a combined arm-leg aerobic circuit training program in patients with CAD. However, in contrast to previous studies,[10-12] cardiorespiratory and hemodynamic responses to upper and lower extremity exercise testing were obtained before and after the conditioning regimen. Moreover, we used an identical intensity, frequency,

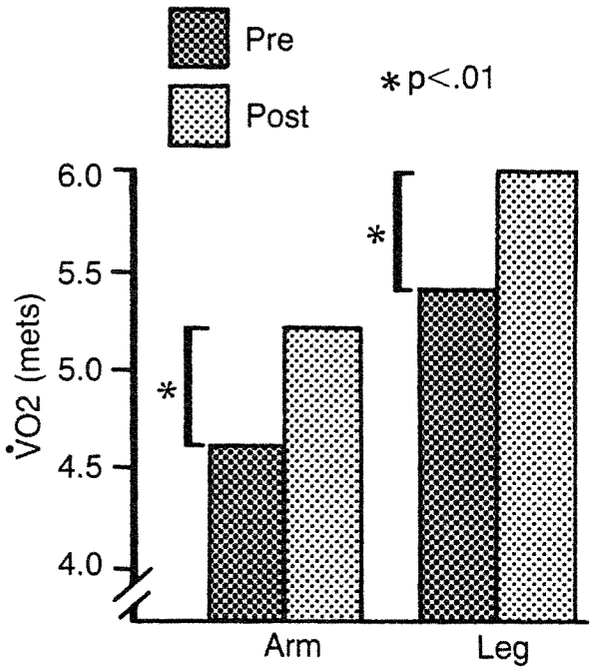

FIGURE 3. Mean Vo_2 max values, expressed as METs, during arm and leg exercise testing before and after training in men with previous myocardial infarction.

and duration for upper and lower extremity conditioning to assess whether the arms respond to exercise training in a similar manner as the legs.

The potential for differences in relative trainability between the upper and lower extremities was first suggested by Fardy et al[13] who reported a greater arm Vo_2 max than leg Vo_2 max when aerobic capacity was expressed per milliliter of limb volume. Because the arms were approximately one third the volume of the legs and the Vo_2 max for the arms was two thirds that of the legs, Vo_2 max for arm exercise was twice that of the legs. One possible explanation for this is that the arm muscles contain a greater proportion of high oxygen extraction (oxidative) muscle fibers than the legs. The investigators cautioned, however, that the apparent twofold oxygen use per milliliter of arm volume may be spurious, because arm ergometry also uses muscle groups of the back, shoulders, and chest.

As reported by others,[14-16] submaximal arm exercise was performed at a greater physiologic cost than was leg exercise. Postconditioning RPPs during submaximal arm and leg ergometry were similarly decreased, while arm and leg Vo_2 max increased 13 and 11 percent, respectively. Peak power output increased 24 percent during arm ergometry and 23 percent during leg ergometry.

The increase in Vo_2 max in patients with CAD ranges from 11 percent to 56 percent after 3 months of leg training and averages about 20 percent in most studies.[17] Similar increases in upper body fitness have been reported after arm training programs of comparable duration.[18] Slightly lower improvements in Vo_2 max in the present study may be attributed to the relatively brief physical conditioning program (6 weeks), the apportioning of arm and leg training time per exercise session (15 min each), or both.

In summary, our findings suggest that the upper extremities respond to aerobic exercise conditioning in the same qualitative and quantitative manner as the lower extremities, showing comparable relative decreases in submaximal RPP and increases in peak power output and Vo_2 max for both sets of limbs when the same exercise training intensity, frequency, and duration are used for the arms and legs.

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Aerobic exercise training programs for the upper body

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ABSTRACT

FRANKLIN, B. A. Aerobic exercise training programs for the upper body. *Med. Sci. Sports Exerc.*, Vol. 21, No. 5 (Supplement), pp. S141–S148, 1989. Sufficient data are available to support the inclusion of upper body or combined arm-leg training in a comprehensive physical conditioning program. There is now evidence to suggest that initial fitness, as well as the intensity, frequency, and duration of training, may be important variables in determining the extent of cross-training benefits from the legs to the arms, and vice versa. Nevertheless, the limited degree of transfer of training benefits from one set of limbs to another appears to discount the practice of emphasizing leg training alone. Aerobic exercise programs for the upper body may yield significant central ($\dot{Q}$ and SV) and peripheral ($a\text{-}\bar{v}O_2$ difference) adaptations to support improvements in peak oxygen uptake ($\dot{V}O_{2peak}$) during arm and leg work, especially in subjects who are initially unfit, with the more dominant effects specific to the upper extremities. Finally, an arm exercise prescription that is based on the maximal heart rate derived from leg testing may result in an inappropriately high target heart rate for arm training. Workloads (kg $\cdot$ min^{-1}) considered appropriate for leg training will generally need to be reduced by 50–60% for arm training.

UPPER BODY EXERCISE, ARM TRAINING, ARM $\dot{V}O_{2peak}$, TRAINING SPECIFICITY, ARM EXERCISE PRESCRIPTION

Considerable information is available on exercise training techniques, particularly those involving the lower extremities (37,40). Until recently, however, there was little or no emphasis on dynamic arm exercise training, despite research that strongly supports the inclusion of this type of exercise in adult fitness and cardiac rehabilitation programs (7,27). This review summarizes the physiological basis and rationale for complementary upper body exercise regimens, with specific reference to selected arm training studies and guidelines for arm exercise prescription.

IMPLICATIONS FOR UPPER BODY TRAINING PROGRAMS: ARE TRAINING EFFECTS TRANSFERABLE?

The extent to which training effects are transferable remains a controversial issue (7). Nevertheless, the question is an important one from a practical perspective, as such information has relevance to the mechanisms underlying cardiovascular training effects and implications for exercise prescription. Several investigators have used training programs involving either arm or leg exercise in an attempt to clarify whether or not the physical conditioning effects can be generalized to exercise with untrained limbs (10,11,62). Others have studied the acute physiological responses to one- and two-leg work (13) and the peripheral and central adaptations to one-legged exercise (55).

Evidence "against" the Transfer of Training Effects

Clausen et al. (10) initially demonstrated that leg training caused a substantial decrease in the heart rate response to leg exercise, but not to arm exercise. Conversely, arm training resulted in a relative bradycardia in response to submaximal arm exercise, but not to leg exercise (Fig. 1). Similar "muscle specific" adaptations have been shown for blood lactate (42) and pulmonary ventilation (54), expressed as the ventilatory equivalent for oxygen ($\dot{V}E/\dot{V}O_2$) (Fig. 2).

Stamford et al. (60) studied the effects of high intensity arm or leg training on the peak oxygen uptake ($\dot{V}O_{2peak}$) of the upper and lower extremities in relatively fit subjects. The physical conditioning program included only three 10 to 15 minute sessions per week for 10 weeks. Subjects who participated in the arm training regimen ($N = 8$) demonstrated a 19% increase in peak $\dot{V}O_2$ during arm work; in contrast, leg $\dot{V}O_{2max}$ in these subjects increased only slightly, from 42.7 to 43.1 ml$\cdot$kg$^{-1}\cdot$min^{-1}. Similarly, leg-trained subjects ($N = 9$) demonstrated a 15% increase in leg $\dot{V}O_{2max}$, whereas arm $\dot{V}O_{2peak}$ remained unchanged.

Studies of single leg training have also shown no transfer of training effects to the untrained limb, but a small increase in two-leg maximal exercise performance, as measured by somatic oxygen uptake (13,55).

Additional evidence, compatible with the above research, indicates that arm training *per se* has little

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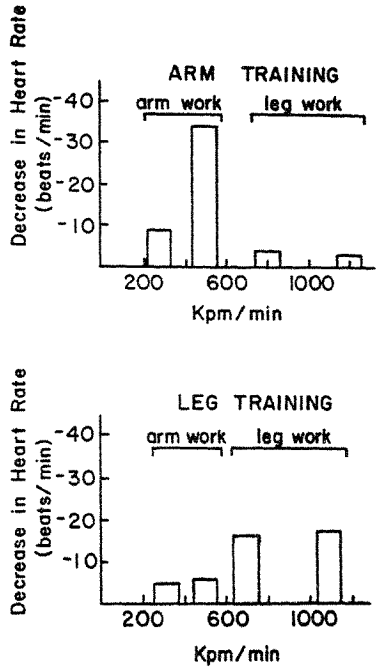

Figure 1—Group mean heart rate and workload response to arm and leg exercise before and after training. *Top*: Arm training markedly reduced the heart rate response to arm exercise; however, the heart rate response to leg exercise decreased only slightly after arm training. *Bottom*: Leg training markedly reduced the heart rate response to leg exercise; however, the heart rate response to arm exercise decreased only slightly after leg training. (Adapted from Clausen et al. (10).)

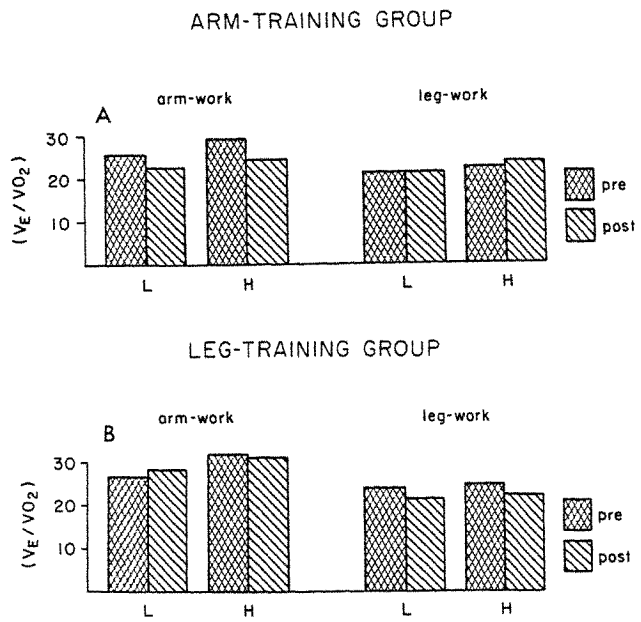

Figure 2—Ventilatory equivalents ($\dot{V}E/\dot{V}O_2$) during light (L) and heavy (H) submaximal arm and leg work before and after (A) arm training and (B) leg training. (Adapted from Rasmussen et al. (54).)

influence on the retention of leg training effects (50). These findings would appear to contradict the widely held notion that reversion to an alternative training modality during recovery from injury will result in a slowing of the detraining process, if not an absolute preservation of the cardiorespiratory fitness level.

Further support for the lack of transfer of training effects comes from the work of Gaffney et al. (31), who attempted to achieve a leg training effect at minimal levels of cardiorespiratory stress. Low-level calisthenic exercises (knee-bend, heel-lift, hip flexion, and hip extension) were performed separately with each leg, requiring less than one-third of the $\dot{V}O_{2max}$ obtained during two-leg cycle ergometer exercise. Heart rates during training were less than 125 beats·min^{-1}. The program significantly decreased the heart rate response to calisthenic exercise, but the cardiorespiratory responses to two-leg cycle ergometry remained unchanged.

Evidence "for" the Transfer of Training Effects

In contrast to the aforementioned reports, several studies (11,43,47,62) have shown some transfer of training effects, i.e., increased $\dot{V}O_{2max}$ or decreased submaximal exercise heart rate with untrained limbs, providing evidence for central circulatory adaptations to endurance training.

In a follow-up to their earlier study, using a larger number of subjects, Clausen et al. (11) confirmed that arm training failed to affect the cardiovascular response to leg exercise; however, leg training caused a significant reduction in submaximal heart rates during both forms of exercise.

Lewis et al. (43) studied the effects of arm or leg ergometer training on the peak $\dot{V}O_2$ during upper and lower extremity work in inactive subjects. The exercise program included four 30-min sessions each wk for 11 wk. Arm trained subjects ($N = 5$) demonstrated significant improvements in peak $\dot{V}O_2$ during arm and leg testing, 35% and 12%, respectively. Leg-trained subjects ($N = 5$) also showed postconditioning increases in peak oxygen uptake during leg (15%) and arm (9%) exercise. However, the findings suggested that additional limb-specific training would be required to maximize the conditioning response, particularly for the upper extremities, since the cross-trained improvement in arm $\dot{V}O_{2peak}$ was considerably below that achieved with arm training alone.

Thompson et al. (62) studied the cardiorespiratory responses of trained and untrained limbs in men with angina pectoris before and after 8 wk of arm ($N = 4$) or leg ($N = 7$) exercise training. Subjects trained for 40 min per session, 3 d·wk^{-1}, at or near the anginal threshold. Time to angina increased 3.6 min during trained limb and 1.6 min during untrained limb exercise. At a constant submaximal workload, the rate-pressure product was reduced by 35 and 18% during trained and untrained limb exercise, respectively. The arm-trained group demonstrated a 19% increase in peak $\dot{V}O_2$ during arm work and a 10% increase in leg $\dot{V}O_{2max}$; the leg-trained group showed a 10 and 8% improvement in peak oxygen uptake during leg and

arm work, respectively. It was concluded that physical training improves the exercise capacity of untrained limbs in patients with angina pectoris by a generalized training effect not dependent on adaptations in trained skeletal muscle.

More recently, Loftin et al. (44) reported that endurance arm training of inactive subjects elicited significant circulorespiratory function adaptations to support improved peak oxygen uptake in both arm and leg exercise, 32% and 7%, respectively.

Although the conditions under which the interchangeability of arm and leg training effects may vary, there is evidence (Table 1) to suggest that the initial fitness of the subjects as well as the intensity, frequency, and duration of training may be important variables in determining the extent of cross-training benefits to untrained limbs (43). For example, Magel et al. (45) reported that the treadmill $\dot{V}O_{2max}$ increased only slightly after arm training in subjects with a relatively high pretraining aerobic capacity, from 56.4 to 57.2 $ml \cdot kg^{-1} \cdot min^{-1}$. In contrast, low initial arm and leg $\dot{V}O_{2peak}$ in unfit normal subjects (43) and men with angina pectoris (62) may have provided the potential for transfer effects to exercise with untrained limbs.

Rationale for Aerobic Exercise Training Programs for the Upper Body

Unfortunately, leg training programs fail to accommodate individuals who cannot perform sustained lower extremity exercise, including paraplegics, amputees, or those with orthopedic problems, neurologic disorders, disabling arthritis, or severe peripheral vascular disease (22,40). In addition, the limited degree of crossover of training benefits from one set of limbs to another appears to discount the practice of emphasizing walk-jog or cycle ergometer training exclusively. Since many recreational and occupational activities require sustained arm work to a greater extent than leg work (25,26,39), it appears reasonable to encourage individuals to train the arms as well as the legs, with the expectation of attenuated cardiorespiratory, hemodynamic and perceived exertion responses to both forms of effort.

Although upper body exercise for cardiac patients has been traditionally proscribed, numerous studies (5, 12,16,62) have now demonstrated the safety and effectiveness of arm exercise training in this population. Moreover, arm exercise in those with heart disease has not been associated with an increased incidence of dysrhythmias, ischemic ST-segment depression, or angina pectoris (23).

Several investigators (10,24) have suggested that therapeutic training programs should include the type of isometric and dynamic arm and leg exercise that most closely corresponds to that required for the person's daily activity. The rationale for this recommendation is that training effects tend to be activity-specific (7). Accordingly, such regimens should serve to maximize

TABLE 1. Age, subject charactenstics, description of exercise program, and changes in trained or untrained limb $\dot{V}O_{2peak}$ after arm, leg, or combined arm-leg training in normal, cardiac, and wheelchair-confined individuals.

Reference	Subjects	Mean Age (yr)	Exercise Program Characteristics					Type Test	$\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)		
			Intensity	Session (min)	Frequency (sessions · wk^{-1})	Type	Duration (wk)		Pre	Post	%Δ
Clausen et al. (11)	3 N	21–30	>170 beats · min^{-1}	35	5	CE	5	CE	46.4	54.3	17
								AE	36.5	40.2	10
Pollock et al. (52)	8 D							AE	20.5	24.4	19
	11 N	38	≈80–85% HR_{max}	30	3	AE	20	AE	23.3	32.5	39
Magel et al. (45)	9 N	College students	≥85% HR_{max}	20	3	AE	10	AE	33.9	39.3	16
								T	56.4	57.2	1
Stamford et al. (60)	8 N	20	≥180 beats · min^{-1}	10	3	AE	10	AE	36.9	43.9	19
								CE	42.7	43.1	1
	9 N	19	≥180 beats · min^{-1}	15	3	CE	10	CE	42.1	48.4	15
								AE	37.0	37.0	0
Lewis et al. (43)	5 N	20	≥75–80% $\dot{V}O_{2max}$	30	4	AE	11	AE	22.8	30.8	35
								CE	37.2	41.7	12
	5 N	22	≥75–80% $\dot{V}O_{2max}$	30	4	CE	11	CE	39.2	45.1	15
								AE	25.0	27.3	9
Thompson et al. (62)	4 AP	60	To angina	40	3	AE	8	AE	12.1*	14.4*	19
								CE	13.5*	14.9*	10
	7 AP	56	To angina	40	3	CE	8	CE	14.3*	15.8*	10
								AE	13.1*	14.1*	8
Mostardi et al. (48)	6 N	31	80–95% HR_{max}	NG	3	AE, CE	6	CE	39.2	44.5	14
	5 N	30	80–95% HR_{max}	NG	3	CE	6	CE	41.3	46.8	13
DiCarlo (18)	1 SCI	24	80% HR_{max}	15–30	3	AE	8	AE	11.0	17.0	55
DiCarlo et al. (20)	4 SCI	24	60–80% HR_{max}	37	3	AE	5	AE	16.0	26.4	65
DiCarlo (19)	8 SCI	24	50–60% HRR	15–30	3	AE	8	AE	12.1	23.5	94
Loftin et al. (44)	38 N	18–35	70–90% HRR	32	4	AE	5	AE	NG	NG	32
								CE	NG	NG	7

Abbreviations: N = normals; D = disabled; SCI = spinal cord injured; AP = angina pectoris; NG = not given; CE = cycle ergometer (legs); T = treadmill; AE = arm ergometer.
* Peak $\dot{V}O_2$ values were calculated from experimental data provided.

the conditioning response through increased crossover of training benefits to real life situations.

CHRONIC ADAPTATIONS TO UPPER BODY TRAINING: RELATIVE ROLES OF CENTRAL VERSUS PERIPHERAL FACTORS

Although the acute cardiorespiratory responses to arm exercise have been well-documented (2,6,58,59, 61), few data are available regarding the effect of upper body training on the determinants of peak $\dot{V}O_2$ during arm or leg exercise, specifically cardiac output ($\dot{Q}$), stroke volume (SV) and arterial-venous oxygen difference (a-$\bar{v}O_2$ difference). With lower extremity training, the improvement in $\dot{V}O_{2max}$ appears to be more dependent on central then peripheral circulatory changes, at least for middle-aged and older men (38). On the other hand, for cardiac patients with impaired left ventricular function, it appears that peripheral adaptations predominate (17).

Magel et al. (45) studied the metabolic and cardiovascular adaptations to aerobic arm training in nine male college students. The subjects participated in 10 wk of interval training for 20 min per session, 3 $d \cdot wk^{-1}$, at a workload that elicited a heart rate of at least 85% of each subject's peak heart rate as determined by arm ergometry. The increase in peak oxygen uptake during arm work, from 33.9 to 39.3 $ml \cdot kg^{-1} \cdot min^{-1}$, was attributed to a widened a-$\bar{v}O_2$ difference, since peak $\dot{Q}$, SV, and heart rate were unchanged. These findings suggest that the conditioning response to arm training derives from extracardiac or peripheral factors, for example, alterations in blood flow and cellular and enzymatic adaptations in the trained limbs alone (13, 41,55).

In contrast, other investigators have reported significant increases in submaximal and peak $\dot{Q}$ following endurance arm training programs (11,44,59). These increases presumably contributed, at least in part, to concomitant improvements in leg $\dot{V}O_{2max}$.

Recently, Loftin et al. (44) reported that an aerobic arm training regimen in women (aged 18 to 35 yr) elicited significant central ($\dot{Q}$ and SV) and peripheral (a-$\bar{v}O_2$ difference) adaptations to support improvements in peak oxygen uptake during arm and leg work, with the more dominant effect specific to the upper extremities (Table 2). The investigators suggested that the subjects' low initial peak $\dot{V}O_2$ values during arm and leg exercise may have provided the potential for improvements in both central and peripheral metabolic and circulatory function.

Together, these studies provide some insight into the mechanisms underlying the physiologic responses to endurance arm training and the degree of adaptation in untrained limbs (i.e., transfer of training). It appears that arm training is not as effective as leg training in eliciting systemic or general effects, since it is carried out at relatively low levels of somatic oxygen uptake (7). Conditioning the upper extremities at 70% of the peak arm $\dot{V}O_2$ usually requires less than 50% of the two-leg $\dot{V}O_{2max}$. However, regular upper body aerobic exercise may yield significant improvements in central circulatory function during arm and leg work, particularly in subjects who are initially unfit (44). Moreover, SV may actually become the primary determinant of the peak $\dot{V}O_2$ during arm exercise (8).

ARM EXERCISE PRESCRIPTION

Guidelines for arm exercise prescription should include recommendations regarding four variables: (a) the "target" or training heart rate; (b) the relationship between the percentage of relative oxygen uptake (% $\dot{V}O_{2max}$) and relative heart rate (% HR_{max}) during arm ergometry; (c) the power output ($kg \cdot m \cdot min^{-1}$) that will elicit the required metabolic load for training; and (d) the proper training equipment or modalities.

Arm Exercise Training Heart Rate

Although the prescribed heart rate for upper body endurance training ideally should be derived from arm ergometer testing, this may not always be practical. Consequently, arm training heart rates are often "extrapolated" from treadmill or cycle ergometer test results. To assess the validity of this practice, we reviewed the peak heart rates (HR_{peak}) during arm and leg ergometry in normal and cardiac men, and in normal women. Table 3 shows that mean peak (or maximal) heart rates obtained during arm ergometry are equivalent to 88–98% of the maximal heart rates obtained during leg ergometry, with a mean value of 93%. In our previous studies of healthy men and women (29,63), individual peak heart rates during arm ergometry were 2–35 $beats \cdot min^{-1}$ lower than for leg ergometry.

TABLE 2. Comparison of the percent changes in central and peripheral determinants of arm and leg $\dot{V}O_{2peak}$ following endurance arm training

	$\dot{V}O_{2peak}$		$\dot{Q}$	SV	HR	a-$\bar{v}O_2$ difference
	($l \cdot min^{-1}$)	($ml \cdot kg^{-1} \cdot min^{-1}$)	($l \cdot min^{-1}$)	($ml \cdot beat^{-1}$)	($beats \cdot min^{-1}$)	($ml \cdot dl^{-1}$ blood)
Arm	33*	32*	14*	11*	2	16*
Leg	7*	7*	6*	10	−3	2

Adapted from Loftin et al. (44).
* $P < 0.05$ (pre vs post).

TABLE 3. Comparison of the peak heart rate (HR_{peak}) in response to arm and leg exercise in men and women.

Reference	HR_{peak} (beats·min^{-1}) Arms	Legs	HR_{peak} Diff. (legs − arms)	HR_{peak} Ratio (%) (arms/legs)
Men (normal)				
Åstrand and Saltin (2)	177	190	13	93
Stenberg et al. (61)	178	188	10	95
Bar-Or and Zwiren (4)	173	195	22	89
Bergh et al. (6)	176	189	13	93
Davis et al. (14)	184	193	9	95
Fardy et al. (22)	174	185	11	94
Magel et al. (45)	174	195	21	89
Bouchard et al. (9)	183	186	3	98
DeBoer et al. (15)	167	190	23	88
Sawka et al. (56)	169	179	10	94
Franklin et al. (29)	172	184	12	93
Pimental et al. (51)	181	188	7	96
Gleim et al. (32)	172	187	15	92
Men (cardiac patients)				
Schwade et al. (57)	122	129	7	95
DeBusk et al. (16)	142	145	3	98
Women (normal)				
Vander et al. (63)	169	177	8	95
Gleim et al. (32)	166	184	18	90
Mean	169	181	12	93

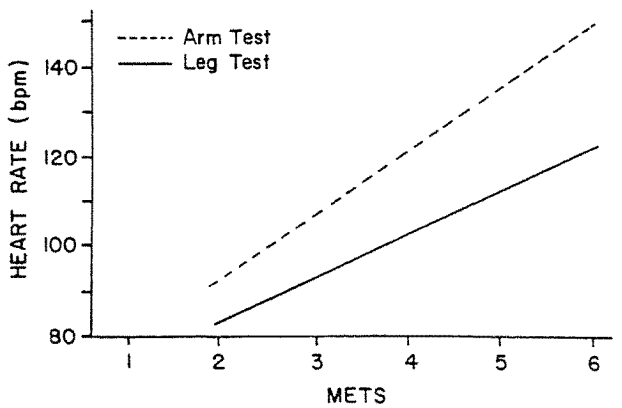

Figure 3—Comparison of the peak oxygen uptake (METs) and heart rates derived from continuous multistage arm ergometer and leg (treadmill) tests in a patient with severe arteriosclerosis in the lower extremities (Adapted from Fardy et al. (22).)

Therefore, an arm exercise prescription that is based on the chronotropic response to treadmill or leg ergometer testing may result in an inappropriately high target heart rate for arm training (29). As a general guideline, we have reduced the prescribed heart rate for leg training by approximately 10 beats·min^{-1} for arm training, using perceived exertion as a complementary method for delineating the appropriate exercise intensity.

Case report. Fardy et al. (22) provided a case report illustrating the importance of an arm ergometer evaluation in establishing the recommended heart rate for arm training. The case involved a 40-yr-old male whose peripheral vascular disease severely limited his ability for sustained walking. Preliminary arm ergometer and treadmill testing revealed a higher peak heart rate during the former, 148 vs 123 beats·min^{-1}, although peak $\dot{V}O_2$ was virtually identical for both exercise modalities (6 METs) (Fig. 3). End-points for arm and leg working capacity were muscle fatigue and ischemic pain, respectively. The patient's prescribed arm training heart rate, calculated at 85% of the peak heart rate, varied considerably depending on whether the arm or leg test results were used, corresponding to arm training heart rates of 126 or 105 beats·min^{-1}, respectively. Although the subject's functional capacity remained unchanged following a prescription that was based on the results of the initial treadmill test, he demonstrated significant improvement when the exercise training heart rate was subsequently increased on the basis of the arm ergometer test.

Relationship between % $\dot{V}O_{2max}$ and % HR_{max}

Research has shown that chronic exercise training at 57–78% $\dot{V}O_{2max}$, equivalent to approximately 70–85% of maximal heart rate (% HR_{max}), elicits favorable physiologic and metabolic adaptations that serve to enhance oxygen transport capacity (40). Since the arm and leg regressions of the % $\dot{V}O_{2max}$ on % HR_{max} are nearly identical (Fig. 4), it appears that a given percentage of peak heart rate during arm exercise (i.e., 70–85%) results in a percentage of arm $\dot{V}O_{2peak}$ that is comparable to that of leg exercise (i.e., 57–78% $\dot{V}O_{2max}$). These findings are important in that the prescribed heart rate for arm training is based on the same heart rate-oxygen uptake regression for leg training. Moreover, recent studies indicate that the heart rate-oxygen uptake relation that is determined during a graded treadmill test can be generalized to combined arm and leg exercise when the intensity is ≅70% $\dot{V}O_{2max}$ (33).

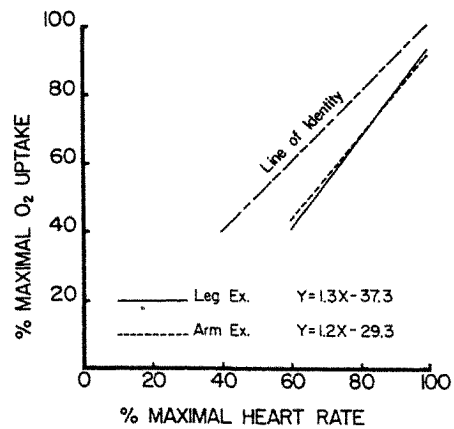

Figure 4—Regression lines during arm and leg exercise show a similar relationship between relative oxygen uptake, expressed as percent $\dot{V}O_{2max}$, and relative heart rate, expressed as percent HR_{max}. In the bivariate linear regressions, y = percent $\dot{V}O_{2max}$ and x = percent HR_{max}. (Adapted from Fardy et al. (22).)

Workloads Appropriate for Arm Training

In establishing the workload that is appropriate for arm training, it is important to emphasize that, at a given submaximal workload, arm exercise is performed at a greater physiologic cost than is leg exercise, but maximal responses are generally lower during arm exercise (2,6,32,61). Therefore, chronotropic and aerobic reserves, relative to incremental loading, are attenuated for arm training as compared with leg training, necessitating reduced workloads for the former.

In our experience, workloads approximating 40–50% of those used for leg training are appropriate for arm training (28). In other words, a subject using 300 kg·m·min^{-1} for leg training would use 120–150 kg·m·min^{-1} for arm training, demonstrating similar heart rates and perceived exertion ratings at these workloads. Others (57) have also noted comparable rate-pressure products at arm workloads approximating half of those used for leg exercise (Fig. 5).

Aerobic requirements of arm ergometry. The relative oxygen cost of arm exercise, expressed as ml·kg^{-1}·min^{-1} or METs (1 MET = 3.5 ml·kg^{-1}·min^{-1}), may be estimated from the cycle ergometer power output (kg·m·min^{-1}), corrected for body weight. Our previous studies (29) showed that the regression of oxygen uptake ($\dot{V}O_2$) on power output during arm ergometry was y = 3.06 × + 191 (y = $\dot{V}O_2$ in ml·min^{-1}; x = power output in kg·m·min^{-1}), where r = 0.91 and Sy·x = 191.6. Since arm $\dot{V}O_2$ (ml·min^{-1}) at a given workload demonstrated the least variability between subjects, Table 4 was constructed to predict arm $\dot{V}O_2$ in ml·kg^{-1}·min^{-1}, based on a constant absolute $\dot{V}O_2$ with a variable subject body weight (50–110 kg). These data complement previous studies (1,21,46,49,53) that facilitate the prediction of "steady-state" oxygen uptake during leg exercise, expressed as ml·kg^{-1}·min^{-1} or METs, from

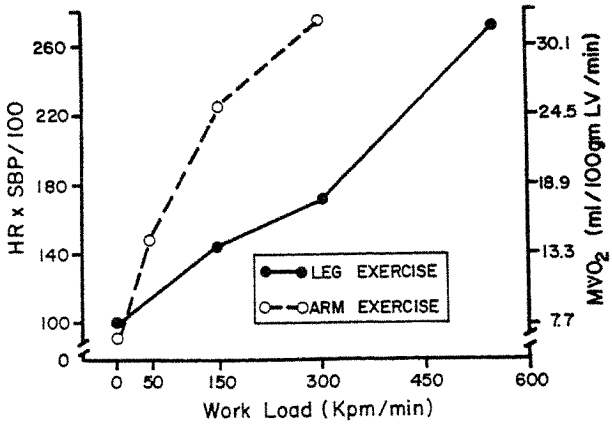

Figure 5—Rate-pressure product (HR × SBP/100) and estimated myocardial oxygen consumption ($M\dot{V}O_2$) during arm and leg exercise in patients with ischemic heart disease. Mean values for the rate-pressure product at 600 kpm·min^{-1} during leg work were not significantly different from mean values at 300 kpm·min^{-1} during arm work. (Adapted from Schwade et al. (57).)

TABLE 4. Aerobic requirements of arm ergometry.

Workload* (kg·m·min^{-1})		150	300	450	600	750
$\dot{V}O_2$ (ml·min^{-1})		648	1104	1562	2079	2431
Body Weight						
(lb)	(kg)	Oxygen consumption (ml·kg^{-1}·min^{-1})				
110	50	13.0	22.1	31.2	41.7	48.7
132	60	10.9	18.6	25.9	34.7	40.6
154	70	9.1	15.8	22.4	29.8	34.7
176	80	8 1	13.7	19.6	25.9	30.5
198	90	7.4	12.3	17.5	23.1	27.0
220	100	6.7	11.2	15.8	20.7	24.2
242	110	6.0	10 2	14.4	18.9	22.1

Adapted from Franklin et al. (29).
* Table discontinued above 750 kg·m·min^{-1} due to small sample size (N = 1).

walking or jogging speed and percent grade, stepping height and frequency, outdoor cycling speed, or the stationary cycle ergometer load corrected for body weight.

Equipment/Training Modalities

Specially designed arm ergometers are particularly good for upper body training. Other equipment suitable for upper extremity training includes rowing machines, weight training apparatus, wall pulleys, light dumbbells, vertical climbing devices, and cross-country skiing simulators. Walking while swinging 0.45–2.27 kg handheld weights or wrist weights can increase the oxygen consumption by 2.1–25.5 ml·kg^{-1}·min^{-1} at any given pace (3,34,64), allowing the conditioning effect to be experienced in the upper and lower extremities. However, careful observation of the blood pressure response to exercise with hand or wrist weights should be conducted before prescribing this form of exercise to hypertensive patients where an increase in cardiac afterload would be contraindicated (35).

Another excellent arm training device, particularly applicable to the gymnasium environment, includes a plastic buoy on two 6-m waxed ropes attached to four plastic handles, as previously described (30). The buoy is moved back and forth by alternately opening and closing a pair of handles.

Our experience with combined arm-leg ergometry indicates that it is more readily tolerated than arm or leg training alone. This observation has been reported by others (61) and is apparently due to the fact that more muscle mass is involved in combined arm-leg ergometry. In addition, it suggests that the perception of effort is related more to the metabolic rate per area of muscle than to the absolute oxygen uptake *per se* (48). Recently, Gutin et al. (36) found that assigning some of the power output to the arms allowed a greater metabolic load to be maintained with no greater cardiovascular or subjective strain. The investigators suggested that combined arm-leg ergometry might be par-

ticularly valuable for aerobic conditioning, cardiorespiratory rehabilitation, and weight control.

CONCLUSION

It is apparent that there is still a lack of basic knowledge regarding the degree of adaptation in untrained limbs (i.e., transfer of training). Discrepancies between studies may be attributed in part to differences in initial subject fitness, the conditioning regimens employed (i.e., intensity, frequency, duration), or both. Nevertheless, sufficient data are available to support the inclusion of arm or combined arm-leg training in a comprehensive physical conditioning program. We must conclude, as Blomqvist (7) so elegantly summarized it in 1985, that:

> ". . . .in a general sense the physiologic data support the concept that therapeutic exercise programs should not be limited to dynamic leg exercise but should include upper body activities. Exercise specifically designed to improve muscle strength may be beneficial, and the exclusion of all activities requiring predominantly static efforts is not warranted."

Like all statements of wisdom, this synopsis appears to be reasonably well-founded and practical, so clearly evident that we feel we should have known it all along.

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SPORTS MEDICINE DIGEST

UNIVERSITY OF CALIFORNIA, SAN DIEGO UCSD

Motivating Patients to Exercise:Strategies to Increase Compliance

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The cardiorespiratory and health benefits of regular physical activity are well documented. Aerobic exercise training decreases the heart rate and blood pressure at rest and at any given level of exertion. Regular physical activity also increases the maximal oxygen consumption or aerobic capacity.

There are many other health benefits associated with regular exercise participation, including increased bone density, enhanced glucose tolerance, an improved coronary risk factor profile, and reduced cardiovascular-related mortality (*N Engl J Med* 1993; 328:333-57).

Figure 1 shows the theoretical relationship between health and fitness benefits expected from increasing doses of exercise. Health benefits can occur at lower levels or intensities of exercise—amounts that may not necessarily improve cardiovascular fitness. Thus, even a modest amount of exercise, if performed regularly, may confer a substantial health benefit.

The compliance problem

While many persons can be encouraged to initiate an exercise program, motivating them to continue can have a favorable impact on public health. Unfortunately, negative variables often outweigh the positive variables contributing to sustained participant interest and enthusiasm. Such imbalance (Figure 2) leads to a decline in adherence while program effectiveness diminishes.

Adult fitness and cardiac rehabilitation programs have reported dropout rates ranging from 9% to 87% (mean 45%). Thus, it appears that exercise is similar to other health-related behaviors, in that typically half or less of those who initiate the behavior will continue.

To understand why people sometimes lack the motivation for regular physical activity, one must first acknowledge a simple yet important fact: exercise is voluntary and time-consuming. Therefore, it may extend the day or compete with other valued interests and responsibilities of daily life.

In one study, patients undergoing gymnasium-based exercise training spent more time in their cars going to and from the programs than patients in a home-training comparison group spend on their cycle ergometers (*Am J Cardiol* 1985;55:251-7).

Motivational strategies

Research and empiric experience suggest that certain program modifications and motivational strategies may enhance participant interest and compliance. These include:

• **Encourage regular exercise participation.** According to recent clinical studies, the single most important factor determining patients' participation in exercise was receiving a strong recommendation from their primary care physician (*Arch Intern Med* 1992;152:1033-5). Therefore, it is important for primary care physicians to discuss these issues with patients.

• **Establish short-term goals.** Participants should be oriented toward intermediate steps that are both realistic and attainable—a principle tenet of sound goal-setting. The key is to draw the patients' focus away from final

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objectives.

• **Minimize injury with a moderate exercise prescription.** Inordinate physical demands, particularly during

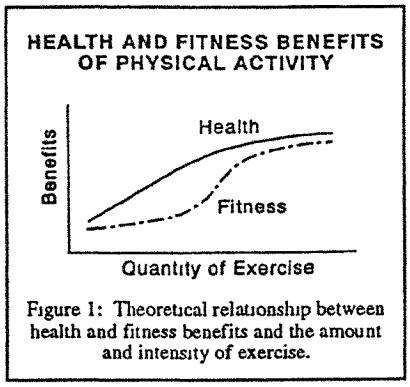

Figure 1: Theoretical relationship between health and fitness benefits and the amount and intensity of exercise.

the initial weeks of an exercise program, often result in muscle soreness, orthopedic injury, and attrition. The fitness leaders should recognize that excessive exercise frequency (>5 days/week), duration (>45 min/session), and/or intensity (>90% aerobic capacity) offer the participant little additional gain in aerobic fitness but disproportionately increase the incidence of injury. Exercise rated as 11 to 15 (Figure 3) is generally sufficient to promote favorable adaptation and improvement. A recommended program for beginners is to walk 20 to 30 minutes every other day.

• **Advocate group participation.** Commitments made as part of a group tend to be stronger than those made independently. The stimulus of the group often provides the incentive to continue during periods of sagging interest. Poorer long-term adherence has been reported in programs where one exercises alone, compared to those that incorporate group dynamics.

• **Emphasize variety and enjoyment.** Calisthenics, when relied on too heavily in an exercise program, readily become monotonous and boring, leading to poor exercise adherence. Programs that are most successful are those that are pleasurable and offer the greatest diversification.

• **Provide positive reinforcement through periodic testing.** Exercise testing, body fatness assessment, and serum lipid profiling should be performed prior to the start of the conditioning program and at regular intervals thereafter, to assess the individual's response to the exercise stimulus. Favorable changes in these evaluations can serve as powerful motivators that produce renewed interest and dedication.

• **Recruit spouse support of the exercise program.** The importance of this influence became evident in one study that showed that the husband's adherence to the exercise program was directly related to the wife's attitude toward it (*Public Health Rep* 1970;85:905-11). Of those men whose spouses had a positive attitude toward the program, 80% demonstrated a good to excellent adherence pattern. However, when the spouses' attitudes were neutral or negative, only 40% showed good to excellent adherence patterns.

• **Include an optional recreational game to the conditioning program format.** The standard warm-up, endurance, and cool-down sequence used in most adult fitness programs offers little in terms of variety or fun. Game modifications which serve to minimize skill and competition and maximize participant success are particularly important. Through such modifications, the exercise leader can emphasize the primary goal of the activity: enjoyment of the game for its own sake.

• **Establish regularity of workouts.** If individuals start their workouts at the same time each day, they will accept them as part of their routine schedule, and exercise will become habitual. Availability of morning and evening sessions should serve to further increase the compatibility of an exercise commitment with the varied schedule of participants.

• **Use progress charts to record exercise achievements.** Research substantiates the importantce of immediate positive feedback on reinforcement of health-related behaviors. A progress chart that permits participants to record daily exercise achievements can facilitate this objective.

• **Recognize individual accomplishments.** Peer recognition is a powerful motivator. To this end, an annual awards ceremony or banquet is recommended. Recognition of participant accomplishments can be made in the form of inexpensive trophies, plaques, ribbons, or certificates.

• **Provide quality, enthusiastic exercise leaders.** Although many variables affect participant exercise compliance, perhaps the most important is the exercise leader. Exercise leaders should be well-trained, highly motivated, innovative, and enthusiastic. The Table lists recommended behavioral strategies of the good exercise leader. Workshop and certification offerings by the American College of Sports Medicine and other professional organizations serve to promote "quality control" and knowledge and proficiency standards for program personnel.

Conclusion

Exercise must be recognized as a lifetime pursuit and not a program of 10-12 weeks' duration with long-lasting residual effects. The individual must develop an attitude toward exercise that reinforces adherence. Primary care physicians, physical thera-

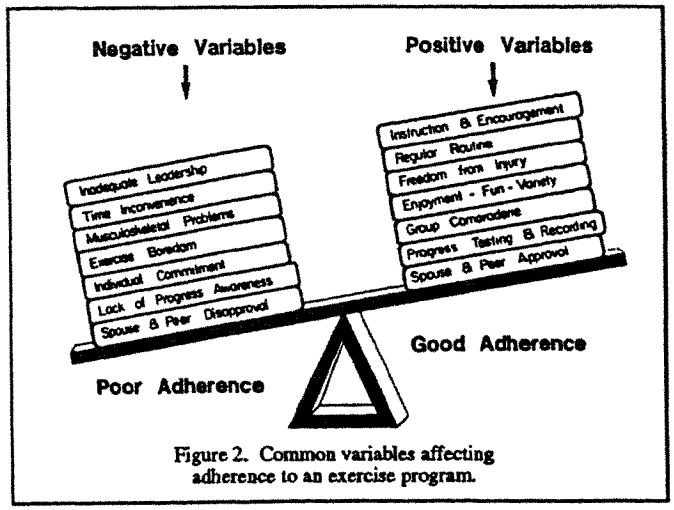

Figure 2. Common variables affecting adherence to an exercise program.

Figure 3. Perceived exertion scale with descriptive "effort ratings."

pists, trainers, exercise physiologists, and other health professionals can play a critical role in favorably modifying the exercise habits of the persons they counsel.

Behavioral strategies of the good exercise leader

1. Show a sincere interest in the participant.
2. Be enthusiastic in your instruction and guidance.
3. Develop a personal association with each participant.
4. Learn participants' names.
5. Consider the reasons why adults exercise (i.e., health, recreation, weight loss, social, personal appearance) and allow for individual differences.
6. Initiate participant follow-up (e.g., written notes or telephone calls) when unexplained absences occur in succession.
7. Participate in the exercise session yourself.
8. Honor special days (e.g., birthdays) or exercise accomplishments with t-shirts, ribbons, or certificates.
9. Attend to orthopedic and musculoskeletal problems.
10. Counsel participants on proper foot apparel and exercise clothing.

Suggested reading

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Editorial Board Comments

[All of these are critical factors to remember. As health care and reimbursement change, a greater responsibility is going to be placed not only on the individual patient but also on his or her family and friends to provide the positive reinforcement and "coaching" that was previously supplied by the health care community.—JEZ] ❑

Benign Exertional Headache

According to the International Headache Society, benign exertional headache (BEH) is any headache precipitated by any form of exercise (especially weightlifting, swimming, and running) or activity (such as sneezing, laughing, or defecating) in the absence of any intracranial disorder (*J Gen Intern Med* 1993;8:333-41).

These headaches are four times more common in men, and twice as frequent in persons over age 40. The headaches have a quick onset, are intense and sharply localized, and usually brief in duration. However, some patients have exertional headaches lasting up to 16 hours associated with nausea and photophobia.

BEH is usually bilateral but may be unilateral in up to 35% of individuals. Pain may be worse in the occipital, frontal, or temporal regions, and is usually not associated with nausea or vomiting. Most patients are pain-free between attacks, but some may have a dull, aching pain lasting for several hours.

Possible mechanisms

The mechanism underlying these headaches is not known, but most experts believe that in some way increased intracranial pressure is involved, putting traction on the pain-sensitive dura.

Although by definition these headaches are benign, several organic conditions—intracranial diseases, craniospinal abnormalities, and certain metabolic and endocrine diseases—can cause exertional headaches and these must be excluded by a thorough work-up.

Up to 25% of patients with BEH have recent histories of respiratory or dental infections. Patients with chronic obstructive pulmonary disease get headache with exertion when there is elevation of PCO_2, and those with acute anemia may get headache due to increased oxygen demand and the concomitant increase in blood flow during exercise.

Treatment

After the diagnosis of BEH is made, treatment depends on the frequency and severity of headaches. If infrequent and always associated with a specific activity, the only treatment necessary may be reassurance about the benign nature of the headaches, simple analgesics taken before anticipated exertion, and/or endurance training.

On the other hand, patients with frequent headaches need prophylactic treatment. In one series, indomethacin (Indocin), in doses ranging from 25 mg to 150 mg daily, was found to be effective for 86% of patients after 1 to 4 weeks of treatment—but the relief was present only during treatment, and indomethacin has severe gastrointestinal side-effects in some patients.

If indomethacin fails, ergonovine, naproxen sodium, and phenelzine—drugs used to treat patients with migraine—may be tried. Propranolol, which is effective in the prophylaxis of migraine, has not been found to be useful in patients with BEH.

Patients who fail to respond to treatment may require a search for

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ACSM CERTIFIED NEWS

A Newsletter for Certified Clinical and Health & Fitness Professionals

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Putting the "Risk of Exercise" in Perspective*

CEC SELF TEST NUMBER 1 *(1.0 CEC)*

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Reports of cardiovascular complications associated with physical exertion have appeared in both the medical literature[1] and the lay press,[2] suggesting that strenuous physical activity may actually precipitate untoward events in some persons. There is also more than anecdotal evidence of an association between heavy physical exertion and sudden cardiac arrhythmic death.

Recently, the commonly held notion that vigorous physical activity can trigger myocardial infarction was strongly substantiated by two consecutive reports in the prestigious *New England Journal of Medicine* — one by Mittleman *et al.*[3] in the U.S., the other by Willich *et al.*[4] in Germany. Both studies concluded that a period of strenuous physical activity is associated with a transient increase in the risk of having an acute myocardial infarction, particularly among persons who are habitually sedentary. These findings have, unfortunately, led to some disconcerting coverage in the media and popular press: "Exercise can kill you; details at eleven."

The purpose of this article is to put in perspective the concerns raised by these studies, so as to enable exercise leaders to more accurately respond to their negative implications and reassure prospective clients of the value of initiating a regular exercise program.

METHODOLOGICAL CONSIDERATIONS

To clarify the role of physical exertion in triggering myocardial infarction, two different research teams working independently in two countries, using similar epidemiologic methods, reported results that were remarkably similar and mutually confirmatory. In each study, the researchers interviewed over 1,000 patients (mostly men) within two weeks of their hospitalization for acute myocardial infarction. Interviews were designed to assess the usual frequency of strenuous physical exertion, as well as the type and intensity of physical activity that preceded the onset of symptoms and myocardial infarction. Both groups of researchers defined "strenuous physical exertion" similarly — an energy expenditure of 6 or more metabolic equivalents (METs), where 1 MET approximates 3.5 ml of oxygen per kilogram of body weight per minute (ml/kg/min). Table 1 illustrates several common occupational and leisure-time activities that approximate or exceed 6 METs.

Although there were subtle differences between the two studies, each addressed three important questions:

- What percent of all heart attacks occur during or soon after strenuous physical exertion?
- Does strenuous physical activity increase the "relative risk" of a heart attack?
- Are persons who are habitually active at a lower risk of an exertion-induced myocardial infarction?

WHAT DID THE RESEARCHERS FIND?

Approximately 4 and 7 percent of the patients in the U.S. and German studies, respectively,[3,4] reported that strenuous physical exertion preceded their heart attack. These percentages are similar to those reported in previous studies relating physical exertion to the frequency of cardiac events.[5,6] Thus, only a relatively small fraction of the infarctions (about 1 in 20) appeared to be triggered by vigorous physical exertion.[7] In approximately 95 percent of all cases, the infarctions came on during lower levels of physical activity or while the patients were at rest.

Overall, the studies showed the "relative risk" of a heart attack during or soon after strenuous physical exertion was two to six

continued next page. . .

times greater than the risk during periods of lighter activity or no exertion, the studies showed. However, the relative risk varied greatly depending on the patient's usual frequency of physical activity. An interesting observation by both studies was the protective effect of regular exercise in decreasing the risk of acute myocardial infarction. In the German study,[4] patients who exercised less than four and four or more times per week had relative risks of 6.9 and 1.3, respectively. The U.S. study[3] revealed that among persons who usually exercised less than one, one to two, three to four, or five or more times per week, the respective relative risks were 107, 19.4, 8.6, and 2.4. In other words, the chance of a sedentary person suffering an exertion-related heart attack was nearly 50 times that encountered by persons who exercise five or more times per week. Exercising just one or two times per week cut the risk by more than 80 percent.

RELATIVE VERSUS ABSOLUTE RISK

Although it appears that vigorous physical exertion increases the incidence of acute myocardial infarction, especially in persons who are habitually sedentary, it is important to clarify the difference between absolute risk and relative risk. Based on data from the Framingham Study, the absolute risk that a 50-year-old nonsmoking, nondiabetic man will have a myocardial infarction during a given one-hour period is approximately one in one million.[8,9] Using the new U.S. study[3] results, if this man was habitually inactive but engaged in vigorous physical activity during that hour, his relative risk would increase 107 times, but his absolute risk during that hour would still be only 1 in 9,346.

MECHANISMS TRIGGERING MYOCARDIAL INFARCTION

Although the reasons that strenuous physical exertion sometimes precipitates myocardial infarction are unknown, it is reasonable to suggest that the increases in heart rate and blood pressure that accompany acute exercise may give rise to hemodynamic stresses that disrupt vulnerable atherosclerotic plaque and lead to thrombosis and occlusion of a coronary vessel.[7] An increase in the blood clotting mechanism has also been reported in sedentary persons who engage in sporadic high intensity exercise.[10]

DOES THE BENEFIT OUTWEIGH THE RISK?

These new reports seem alarming and contradictory to those of us who have been led to believe that regular exercise reduces the risk of heart disease and its consequences. Clearly, the risk of cardiovascular complications is transiently increased during vigorous exercise compared with that at other times.[11-13] This appears to be particularly true among persons with latent or known heart disease who are unaccustomed to exercise. The "critical question," however, is whether the cardiovascular benefits of regular exercise outweigh the risk.

Although these just-published reports[3,4] demonstrated that an isolated, acute bout of vigorous physical exertion can increase the transient risk of myocardial infarction, numerous studies have shown that regular exercise participation decreases the long-term risk of coronary events.[14,15] Endurance exercise training increases physical work capacity and reduces myocardial demands at rest and at any given level of submaximal exertion. Chronic exercise promotes reductions in body weight, fat stores, blood pressure (particularly among individuals with hypertension), serum triglycerides, and low-density lipoprotein (LDL) cholesterol, with increases in the "protective" high-density lipoprotein (HDL) cholesterol fraction.[16] Decreased vulnerability to dysrhythmias[17] and increased resistance to ventricular fibrillation[18] have also been postulated as mechanisms compatible with a specific, training-related protection against sudden coronary

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death. The interesting observation by both Mittleman *et al.*[3] and Willich *et al.*[4] that regular exercise provides protection against the triggering of myocardial infarction by strenuous exertion provides additional evidence for encouraging physical activity.

To answer the question "Is vigorous exercise worth the risk?," Siscovick and associates[19] studied the incidence of sudden death during vigorous physical exertion. The relative risk of cardiac arrest during exercise compared with that at other times was 56 times greater among men with low levels of habitual activity and only 5 times greater among men with high levels. However, the total risk of cardiac arrest among habitually active men was only 40 percent of that for sedentary men. These earlier findings agree with the U.S.[3] and German[4] studies and support the hypothesis that vigorous physical activity both protects against and provokes cardiac events.[20] At this time, considering the data on both sides of this issue, the benefits of exercise outweigh the risks for most people.

IDENTIFYING THE PERSON "AT RISK"

Due to the vagaries of the atherosclerotic process, the accuracy in predicting which persons will have a cardiovascular complication during exercise remains imperfect. Thompson and associates[21] noted that neither superior athletic ability, habitual physical activity, nor the absence of cardiac risk factors guarantees protection against an exercise death. Forewarning symptoms appeared to present the only clue to impending cardiovascular complications. Similarly, Noakes *et al.*[22] reported a high prevalence of these types of forewarning symptoms in distance runners who experienced exercise-related cardiovascular complications: 81 percent developed warning symptoms, yet the majority of these runners continued to train without seeking medical advice.

A relative risk (for acute myocardial infarction) of 107 for some sedentary adults undertaking 6 or more METs of exertion argues for pre-screening and careful initiation of individuals into exercise. Although exercise stress testing is widely recommended to establish the safety of vigorous exercise participation, several studies have reported normal exercise ECG responses in persons who subsequently experienced cardiovascular complications during exercise.[21,23] These findings, coupled with the extremely low rate of cardiovascular complications in asymptomatic persons who exercise, the high costs of mass stress testing, and the uncertainties associated with exercise-induced ST-segment depression in persons with a low pre-test risk of coronary heart disease, suggest that it is impractical to use exercise testing to forestall serious cardiac events in all asymptomatic persons who exercise.[24]

In summary, the need for routine exercise testing for asymptomatic active people remains controversial. Perhaps one alternative to exercise testing as a regular or unvarying screening procedure lies in categorizing patients according to age, coronary risk factors, and the presence or suspicion of disease, and screening them according to the category within which they are placed.

Accordingly, the American College of Sports Medicine[25] recommends maximal exercise stress tests for the following individuals starting an exercise program:

- Apparently healthy men older than 40 and women older than 50 who want to begin a program of vigorous exercise (activity performed above 60 percent of aerobic capacity [$VO_{2\,max}$]);
- Asymptomatic men and women with two or more major coronary risk factors who wish to take up vigorous exercise; and,
- Individuals who have — or have symptoms suggestive of — cardiac, pulmonary, or metabolic disease.

RECOMMENDATIONS

Recommendations to reduce the incidence of cardiovascular complications during exercise are listed below:[12,13]

- Ensure medical clearance and follow-up, including serial exercise testing. These are essential components of exercise therapy for "high risk" adults or patients with cardiovascular disease.
- Establish an emergency plan. The fitness staff should be prepared to handle cardiovascular complications, including cardiopulmonary resuscitation (CPR) and patient stabilization for transport to an emergency center. To this end, emergency drills and CPR practice should be conducted regularly.

Table 1.
Common Activities Classified as "Strenuous Physical Exertion" (≥6 METs)

Self Care or Home	Occupational	Recreational	Exercise Training
Digging in garden	Carpentry*	Tennis (singles)	Walking (≥ 4.5 mph)
Lawn mowing	Shoveling dirt*	Downhill skiing	Swimming (breast stroke)
Climbing stairs	Pneumatic tools*	Basketball	Jogging (≥ 5 mph)
Carrying objects (≥ 30 pounds)	Digging ditches*	Mountain climbing*	Rowing
Sawing wood*	Lumberjack*	Paddleball	Heavy calisthenics
Heavy shoveling*	Heavy laborer*	Handball	Rope jumping
Snow shoveling*			

*May produce excessive myocardial demands because of arm work or isometric exercise.

continued next page.

- Emphasize appropriate warm-up and cool-down procedures. A gradual warm-up (*e.g.*, brisk walking or mild resistance cycling) may decrease the occurrence of abnormal responses that can occur with sudden strenuous exertion.[26] The cool-down enhances venous return, thereby reducing the possibility of post-exercise hypotension and related events. Furthermore, the cool-down combats the potential deleterious effects of the rise in plasma catecholamine levels that follow exercise.[27]
- Encourage a mild-to-moderate intensity exercise prescription. The lower the intensity, the less likely that an exercise-related cardiovascular complication will occur. Moreover, a reduced training intensity may be partially or totally compensated for by more frequent or longer training sessions.[16] Borg's[28] perceived exertion scale is a useful and important adjunct to heart rate as an intensity guide. Ratings greater than 13 to 15, corresponding to "somewhat hard" to "hard," indicate an exercise intensity that is too high, regardless of the heart rate response.
- Promote participant education. It is important that clients know their prescribed heart rate range for training, how to take their pulse accurately, and how to recognize serious warning signs and symptoms. Participants should be counseled to discontinue exercise and seek medical advice if they experience abnormal heart rhythms, chest pain or pressure, or dizziness.[29]

CONCLUSION

The risk of cardiovascular complications appears to increase transiently during strenuous physical exertion compared with the risk at other times. This seems particularly true among persons with latent or documented heart disease who are habitually sedentary. On the other hand, the overall risk of a cardiac event appears to be reduced in persons who are regular exercisers. These considerations should help fitness professionals to put the "risk of exercise" in proper perspective.

About the Author —

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See Self Test Number 1 on page 14.

INNOVATIVE ACTIVITIES FOR ADULT FITNESS AND CARDIAC EXERCISE PROGRAMS: A "HANDS-ON" SESSION

Certificate Course in
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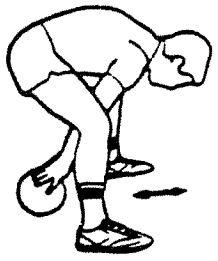

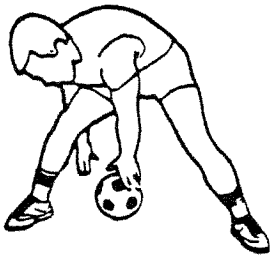

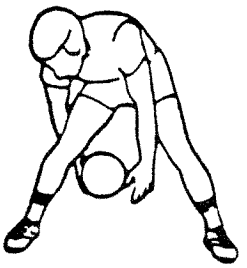

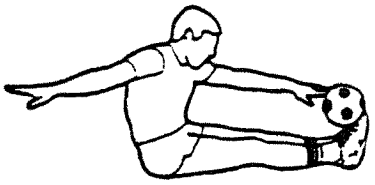

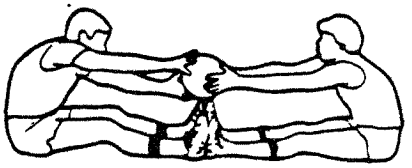

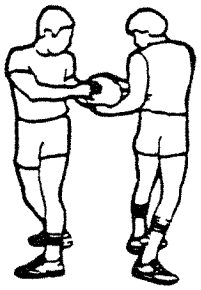

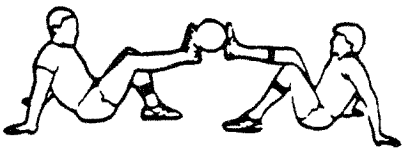

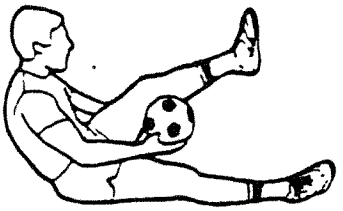

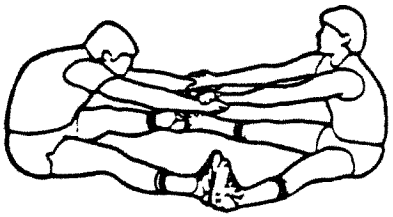

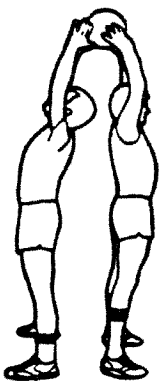

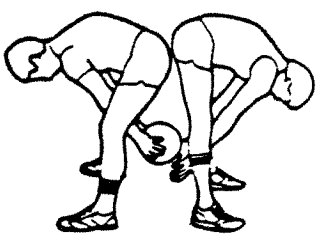

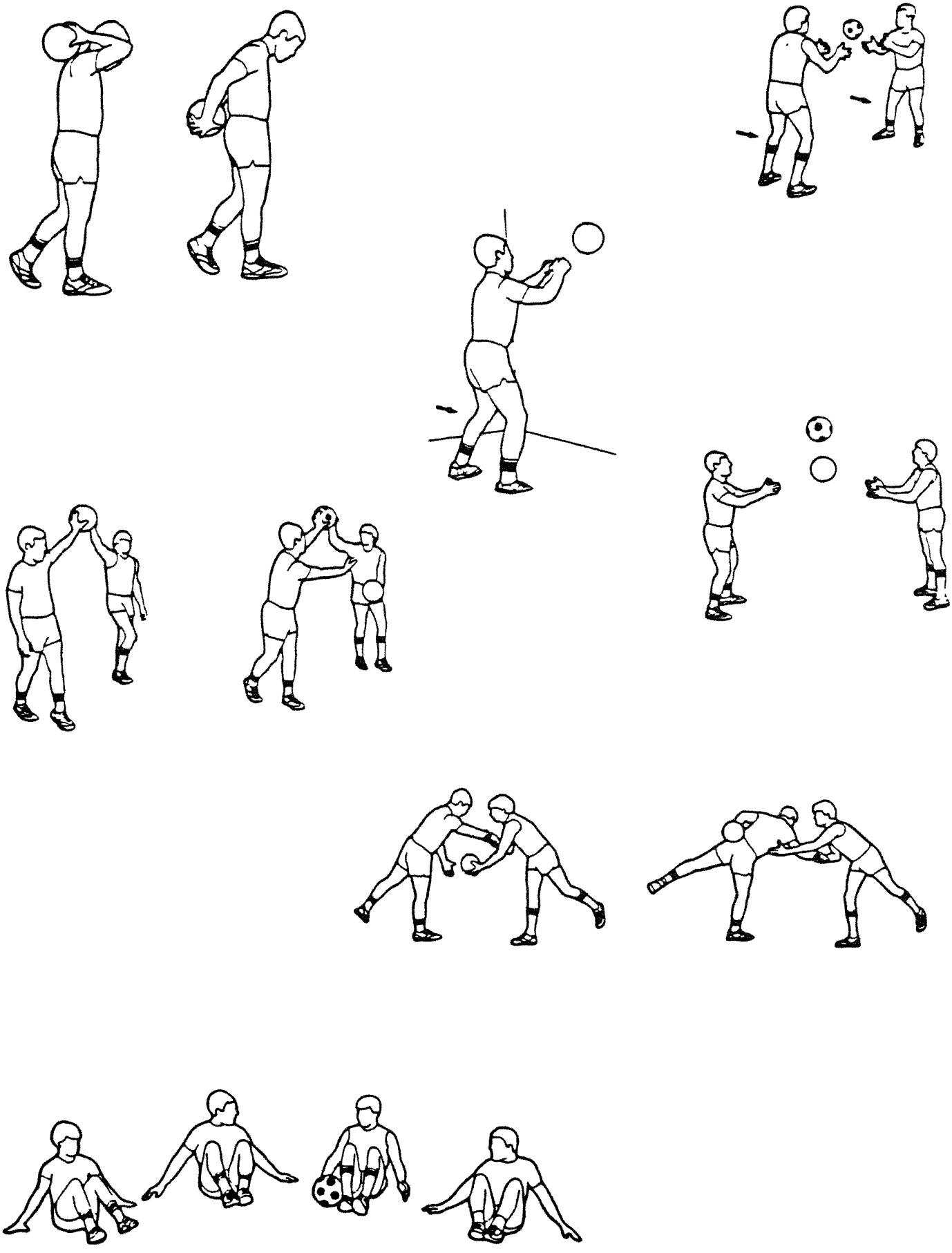

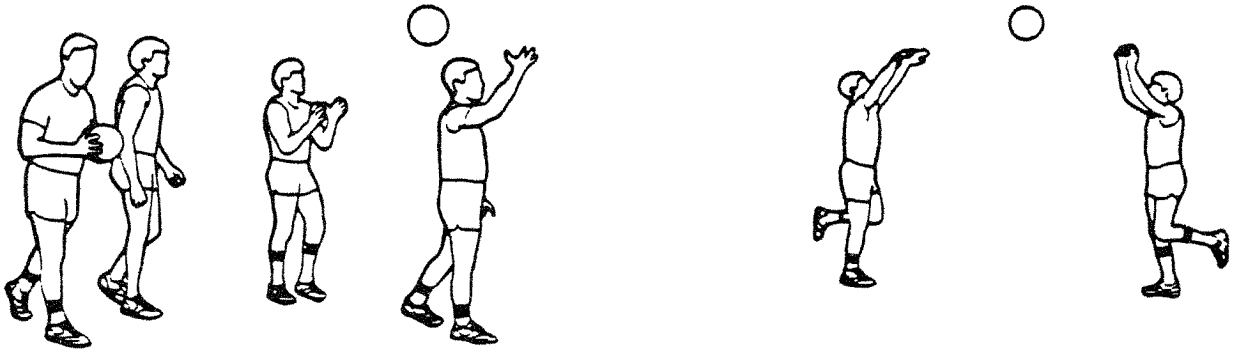

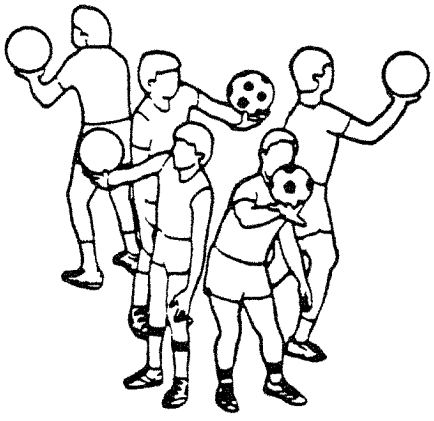

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Resistance Training in Cardiac Rehabilitation

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Cardiac patients require a minimum level of strength for daily living, similar to persons without heart disease. Mild-to-moderate resistance training can provide a safe and effective method for improving strength and cardiovascular endurance, modifying coronary risk factors, and enhancing psychosocial well-being in this population. Nevertheless, proper preliminary screening, appropriate prescriptive guidelines, and careful supervision are important.

Although cardiac exercise programs have traditionally emphasized dynamic lower extremity exercise, increasing research[1] suggests that complementary resistance training, when appropriately prescribed and supervised, has favorable effects on strength,[2–6] cardiovascular endurance,[2,6] hypertension,[7–9] hyperlipidemia,[7,9,10] and psychosocial well-being.[3,11,12] This article reviews the role of resistance training in cardiac rehabilitation, with specific reference to its inherent isometric component, physiologic basis and rationale, safety, and efficacy. Participation criteria and prescriptive guidelines are also provided.

ISOMETRIC EXERCISE

Although isometric or combined isometric and dynamic (isodynamic) exercise has traditionally been discouraged in patients with coronary disease (CAD), numerous studies suggest that these types of exercise may be less hazardous than was once presumed, particularly in patients with minimal functional aerobic impairment and normal left ventricular (LV) function.[13,14] Cardiac patients with good residual LV function generally show appropriate increases in cardiac output during the higher afterload conditions imposed by isometric work and only minor rises in LV end diastolic pressure.[15] Global LV function also remains stable in trained cardiac men during sustained small and large muscle isometric exercise performed at 30% of maximal voluntary contraction; however, such patients may demonstrate the appearance of new wall motion abnormalities.[16]

Several studies have shown that isometric exertion, regardless of the percentage of maximal voluntary contraction (MVC) used, generally fails to elicit angina pectoris, ischemic ST segment displacement, or worrisome ventricular arrhythmias among selected cardiac patients.[17–19] The rate-pressure product, estimated myocardial oxygen consumption, and coronary flow requirements are lower during maximal isometric exercise than during maximal isotonic exercise, primarily due to a lower peak heart rate response (Figure 1).[17–27] Increased subendocardial perfusion, secondary to elevated diastolic blood pressure, and decreased venous return, LV diastolic volume, and wall tension may also contribute to the lower incidence of ischemic responses during isometric or isodynamic effort.[17,18,21,22,28] The myocardial oxygen supply/demand relationship appears to be favorably altered by superimposing static on dynamic effort, so

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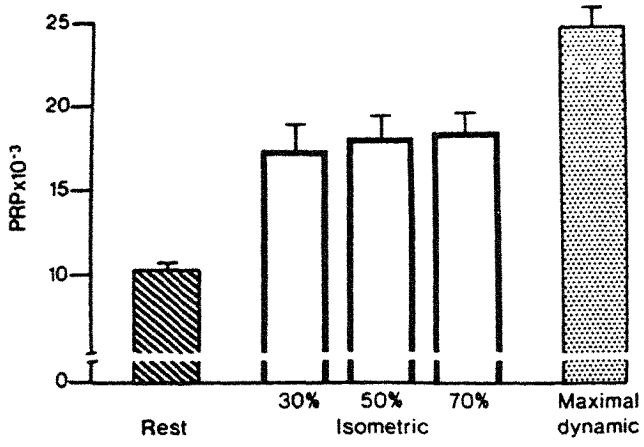

Figure 1. Systolic brachial artery pressure times heart rate (PRP × 10^{-3}) during upright rest, sustained handgrip at 30%, 50%, and 70% of maximal isometric tension, and symptom-limited bicycle ergometer exercise. The rate-pressure product was significantly greater ($P < 0.01$) during dynamic exercise than during 70% of maximal isometric tension. Values represent mean (± SE). (Reproduced from Ferguson, RJ, Côté P, Bourassa MG, *et al., Journal of Cardiac Rehabilitation* 1981;1:121–127, with permission).

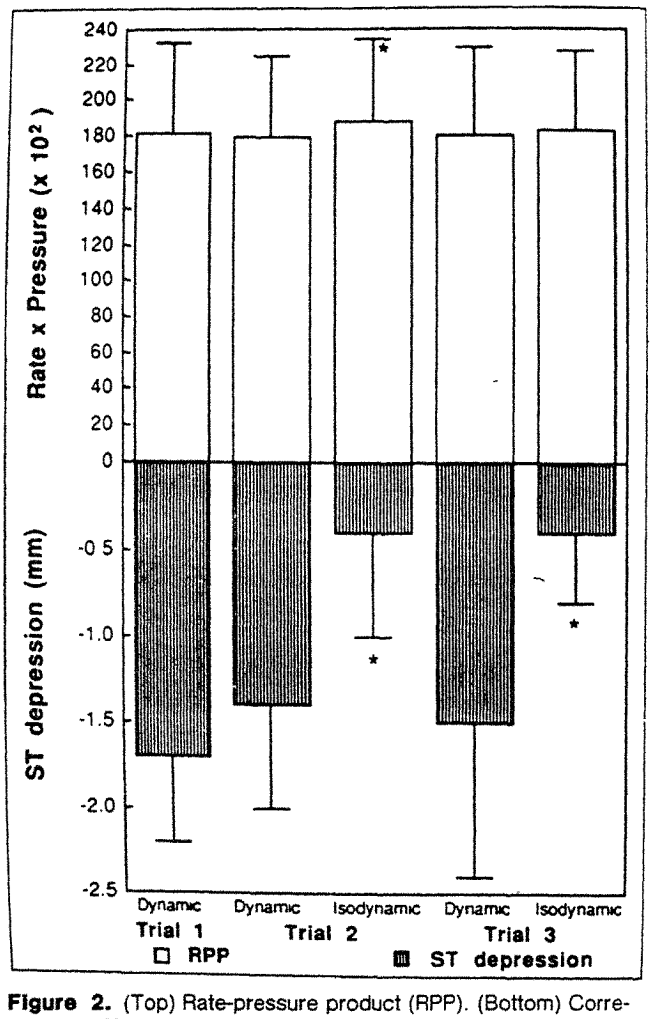

Figure 2. (Top) Rate-pressure product (RPP). (Bottom) Corresponding ST segment depression. Trial 1 is baseline dynamic exercise. Trials 2 and 3 are dynamic and isodynamic exercise. Values are mean (± SD). $^*P < 0.001$ for differences between dynamic and isodynamic exercise trials. (Reproduced from Bertagnoli K, Hanson P, Ward A, *Am J Cardiol* 1990;65:314–317, with permission).

that ischemic electrocardiogram (ECG) responses are attenuated at rate pressure product values that elicit significant ST segment depression during dynamic exercise (Figure 2).[18,28] These findings are changing the cautious attitude toward isometric exertion (and strength training) for coronary patients, particularly in regard to vocational counseling and exercise prescription.[29,30] Some clinicians have suggested that the inclusion of isometric exercise and isodynamic arm exercises in medically supervised exercise programs may improve the ability of many cardiac patients to respond to reasonable occupational and leisure activities.[19]

RATIONALE FOR RESISTANCE TRAINING

Blomqvist[31] summarized the acute and chronic responses to alternate modes of physical training, particularly arm exercise, and concluded that:

> . . . in a general sense the physiologic data support the concept that therapeutic exercise programs should not be limited to dynamic leg exercise but should include upper body activities. Exercise specifically designed to improve muscle strength may be beneficial, and the exclusion of all activities requiring predominantly static effects is not warranted.

Several lines of evidence seem to support resistance exercise as an adjunct to conventional leg training in cardiac patients whose occupational or leisure activities require muscular strength or endurance. Many tasks of daily life require static or isodynamic efforts, often involving the arms rather than the legs.[31] Because the pressor response to static exertion is proportionate to the relative intensity (percent of MVC) (Figure 3),[32] duration, and muscle mass involved,[33–35] increased muscular strength should result in an attenuated blood pressure response to any given load because the load now represents a lower percentage of the MVC.

Although high-resistance, low-repetition weight training has little or no effect on cardiorespiratory fitness, circuit weight training programs can be designed to elicit small-to-moderate increases (~5%) in aerobic capacity.[36] The contention that weight training does little to benefit cardiovascular function

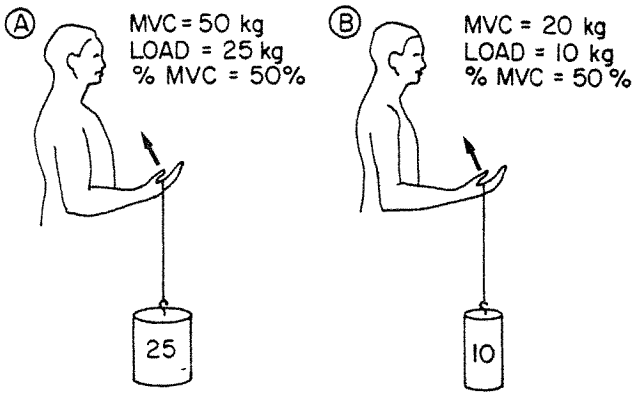

Figure 3. The hemodynamic response to isometric exertion is proportional, in part, to the percentage of maximal voluntary contraction (% MVC) of the muscle group involved. The heart rate and blood pressure response depends on the tension exerted relative to the greatest tension possible in the muscle group (MVC). A high degree of tension exerted by a stronger person (A) will produce approximately the same heart rate and blood pressure response as a low tension representing an equivalent relative tension (% MVC) developed by a weaker person (B), if all other factors are equal.

TABLE I
EFFECTS OF STRENGTH TRAINING ON MAXIMUM OXYGEN UPTAKE ($\dot{V}O_{2MAX}$) AND ENDURANCE DURING BICYCLE AND TREADMILL EXERCISE

	$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)		Endurance (sec)	
	Treadmill	Bicycle	Treadmill	Bicycle
Before training	47.8	44.0	291	278
After training	48.8	44.6	325*	407*

*Before training vs after training, $P < 0.01$.
Adapted from Hickson RC, Rosenkoetter MA, Brown MM: *Med Sci Sports Exerc* 1980;12:336-339.

is largely based on studies that evaluated training effectiveness with treadmill or cycle ergometer testing. When comparing hemodynamic responses with a standardized lifting or isometric test before and after isometric strength training, improvement has been noticed.[37] Such findings strongly support the specificity of measurement and specificity of fitness concept.

There are also intriguing data to suggest that strength training can increase muscular endurance capacity without an accompanying increase in $\dot{V}O_{2max}$. Hickson and associates[38] examined the effects of heavy resistance training on $\dot{V}O_{2max}$ and endurance time during cycle ergometry and treadmill exercise. Nine men ($\bar{x}$ age = 23 yr) participated in the training program (5 days a week for 10 weeks) that consisted of parallel squats, knee flexions and extensions, leg presses, and calf raises designed to strengthen the quadricep muscles. Thigh girth increased significantly, and muscle strength increased by 40% with training. Although $\dot{V}O_{2max}$ during treadmill and cycle ergometry remained essentially unchanged, endurance time to exhaustion increased while cycling (47%) and while running (12%) (Table I). These findings indicate that endurance is not a function of aerobic exercise alone, but can be significantly enhanced by increased muscle strength and/or girth. This is a further argument for the complementary use of weight training with aerobic exercise.

Regular progressive resistance exercise training may reduce blood pressure in hypertensive patients, and improve self-efficacy, glucose tolerance, insulin sensitivity, and lipid and lipoprotein levels in cardiac patients.[7-12] The last effect is, however, controversial.[39]

SAFETY OF RESISTANCE TRAINING

Although cardiac exercise programs have traditionally emphasized lower extremity dynamic aerobic exercise (*i.e.*, walking, stationary cycle ergometry), recent research studies suggest that resistance training programs are safe for selected patients with CAD. Zohman and Kattus[40] found that there was no detrimental effect when cardiac patients used resistance exercise machines, training at 40% of their maximum possible lift. Similarly, Saldivar and co-workers[41] reported that a low-weight, low-repetition strength training program was not associated with symptomatology, ST segment depression, or dysrhythmias in patients with heart disease. Kelemen et al.[2] also reported no sustained arrhythmias, abnormal hemodynamics or cardiovascular problems during a 10-week circuit weight training program in cardiac patients. More recently, Butler and associates,[42] using two-dimensional echocardiography in cardiac patients training at loads corresponding to 40% to 60% of one-repetition maximum, observed a worsening of wall motion in five of 61 LV segments during aerobic exercise, but in only one segment during resistance exercise. Others, however, suggest that resistance training be used with caution in cardiac patients with poor LV function because they may develop further wall motion abnormalities during isometric or isodynamic efforts,[16,43] threatening arrhythmias,[44] or both.

Vander and associates[45] studied the acute hemodynamic and electrocardiographic responses to Nautilus (Nautilus, DeLand, FL) resistance exercise in cardiac patients. Weight loads (range = 29–103 lb) during resistance exercise training (Figure 4) were estimated at 40% to 60% of maximal voluntary contraction. Subjects performed 12 repetitions at each of

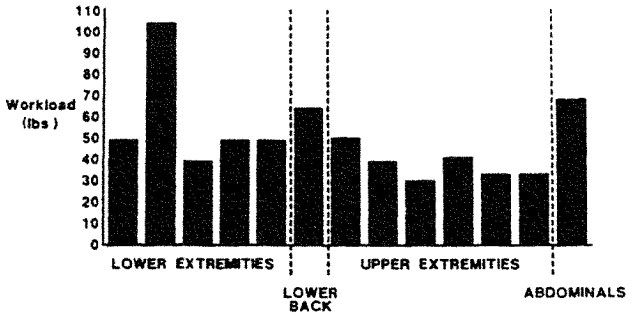

Figure 4. Workload (lb) during Nautilus exercise Lower extremity stations included (left to right) leg extension, duo-squat, leg curl, abduction, and adduction machines Upper extremity stations included (left to right) pullover, lateral raise, overhead press, decline press, multicurl, and multitricep machines (Reproduced from Vander LB, Franklin BA, Wrisley D, et al, *Annals of Sports Medicine* 1986;2·165–169, with permission)

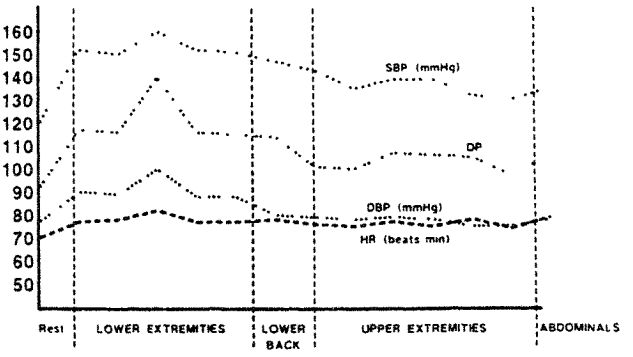

Figure 5. Cardiovascular response at rest and during peak Nautilus exercise. SBP systolic blood pressure (mm Hg), DP double product (beats • min^{-1} X mmHg X 10^{-2}), DBP diastolic blood pressure (mm Hg), HR heart rate (beats • min^{-1}) (Reproduced from Vander LB, Franklin BA, Wrisley D, et al, *Annals of Sports Medicine* 1986,2.165–169, with permission)

13 stations. Mean cardiovascular responses for all subjects (n = 21) at rest and during peak resistance exercise are shown in Figure 5. Upper extremity exercise elicited small increases (range) in heart rate, 3 to 7 beats • min^{-1}, double product, 8 to 17 beats • min^{-1} X mm Hg X 10^{-2}, and systolic/diastolic blood pressure, 6 to 16/2 to 4 mm Hg from rest. Lower extremity Nautilus exercise elicited the following greater increases: 5 to 10 beats • min^{-1}, 8 to 50 beats • min^{-1} X mm Hg X 10^{-2}, and 25 to 36/11 to 24 mm Hg. In contrast to the abnormalities documented on 16 of 21 subjects during graded exercise testing, no significant arrhythmias, ST segment depression, abnormal hemodynamics, or symptoms occurred during resistance exercise. The investigators concluded that Nautilus exercise using light-to-moderate loads is relatively safe in selected cardiac patients.

Ghilarducci and co-workers[4] assessed the safety and efficacy of a high-intensity strength training program in cardiac patients undergoing cardiac rehabilitation. Nine stable, aerobically trained, male subjects met three times a week for 30 minutes of strength training in addition to 30 minutes of stretching, calisthenics, and aerobic activity. The strength training program comprised lifting 80% of maximum voluntary contraction at five stations. No symptoms of ischemia, abnormal heart rate, or blood pressure responses were observed during the training sessions.

The absence of cardiovascular complications in these investigations has led to the inference that resistance exercise is safe for cardiac populations. This notion is based on a limited number of studies that, for the most, have involved small numbers of patients over a relatively short duration (*e.g.*, ≤12 weeks). Two recent reports[3,46] have evaluated the safety of resistance exercise in cohorts of patients who participated for several years. In 1986, Stewart and associates[3] assessed three-year changes in strength, self-efficacy, body weight, and skinfolds in 17 trained cardiac men who regularly performed cardiovascular endurance exercise and circuit weight training. The lack of cardiovascular and orthopedic complications in their program was largely attributed to proper preliminary screening and careful supervision. Our long-term experience with resistance training, using strict prescriptive guidelines and participation criteria, further attests to its safety and efficacy in low-risk cardiac patients. To date, 73 patients have completed 7,422 circuit weight training sessions uneventfully.[46]

Methodologic Limitations

The contention in these studies that resistance exercise evokes acceptable hemodynamic responses should be interpreted with caution because blood pressure was often measured immediately after and not during the actual weight lifting, when values may have been higher. MacDougall and co-workers,[47] using direct arterial measurements of blood pressure, found that blood pressures that were significantly increased during heavy resistance exercise normalized within 10 seconds of recovery. Our recent studies, using cuff-occlusion techniques (Figure 6) to obtain blood pressure values in the legs of exercising patients, indicate that systolic blood pressures taken by the standard cuff method immediately after arm exercise are likely to underestimate true physiologic responses.[48,49] Similarly, Wiecek et al.[50] compared indirect (*i.e.*, auscultation) vs direct measures of arterial pressure during weight lifting in cardiac patients and concluded that the former are inaccurate due to the rapid drop in pressure that occurs after exercise. Nevertheless, measured intra-arterial blood pressures

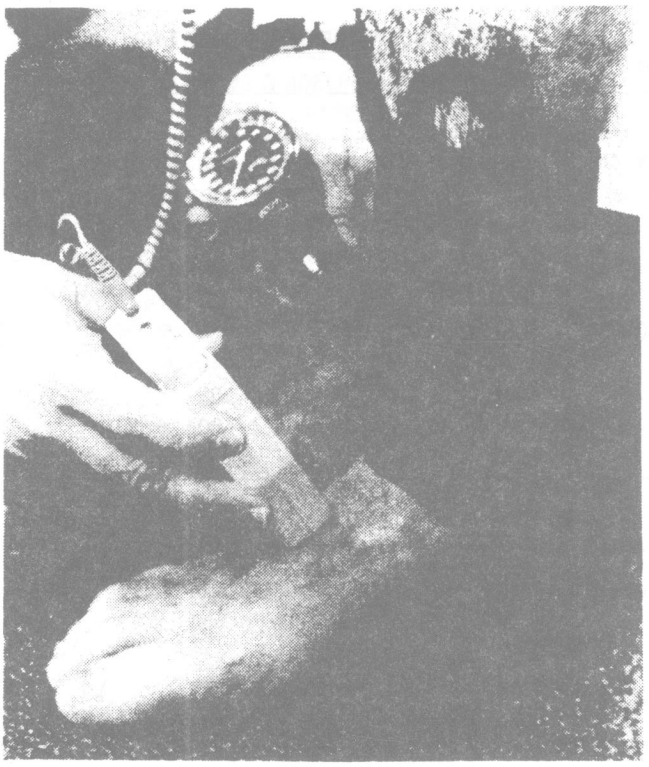

Figure 6. Technique for ankle systolic blood pressure measurements using a pneumatic cuff and an ultrasound Doppler stethoscope.

during weight lifting have been found to be within a clinically acceptable range at 40% of one-repetition maximum.[51]

TRAINING STUDIES

Several investigators have reported on the efficacy of resistance training in patients with CAD.[2–5] In these studies (Table II), circuit weight training was added to the physical conditioning regimens of cardiac patients who were already involved in regular aerobic exercise, generally for 3 months or more. Subjects averaged 56 years of age and had documented histories of myocardial infarction, coronary artery bypass surgery, or angina. Three 30-minute to 60-minute exercise sessions were offered each week. The duration of the training programs varied from 10 to 156 weeks; intensity prescriptions ranged from 30% to 80% of one-repetition maximum (1RM).

Kelemen and co-workers[2] found that peak treadmill time increased by 12%, from 619 to 694 seconds, in their circuit weight group, but remained unchanged in their control group (*i.e.*, volleyball participants). Both groups followed the same warm-up and 20-minute walk/jog at 85% HR_{max}, but the resistance group did an additional 20 minutes of circuit weight training instead of volleyball. Thus, it appeared that weight training, per se, had a favorable effect on cardiovascular endurance.

Strength gains in these studies were comparable to the increases previously reported in healthy individuals undergoing circuit weight training;[36,52] improvements ranged from 13% to 40% for the arms and legs, respectively. The latter increase, however, occurred over a 3-year training period.[3] Although 10 weeks of resistance training at 80% of 1RM elicited a 29% increase in overall strength,[4] training at 30% to 40% of 1RM over the same period resulted in a similar relative improvement (24%).[2] These findings suggest that heavy resistance training, which may potentially increase the hemodynamic response to and risk of circuit weight training, offers little additional benefit in strength to this population.

PATIENT ELIGIBILITY

Cardiac patients who wish to initiate mild-to-moderate training should have participated in a traditional aerobic exercise program for at least 3 months.[53] This time period permits sufficient observation of the patient in a supervised setting and allows for the cardiorespiratory and musculoskeletal adaptations needed to progress to more intense exercise. Although conventional participation guidelines have suggested that surgical and myocardial infarction patients wait at least 4 to 6 months after the event,[5,53] recent studies indicate that many men can safely perform static-dynamic activity equivalent to carrying up to 30 pounds by 3 weeks after myocardial infarction.[54] Thus, it is possible that resistance training could be initiated sooner, if low-weight programs are used.

Exclusion criteria for resistance training are similar to or slightly more cautious than those used for any outpatient, non-electrocardiographically monitored cardiac exercise program (*i.e.*, Phase III–IV). Patients are generally excluded from participation for any of the following reasons: unstable angina, uncontrolled hypertension (systolic blood pressure greater than 160 mm Hg or diastolic blood pressure greater than 100 mm Hg), uncontrolled arrhythmias, a recent history of congestive heart failure, or an aerobic capacity of less than 6 to 7 METS (1 MET = 3.5 $ml \cdot kg^{-1} \cdot min^{-1}$) during symptom-limited exercise testing.[2,5,42,53] It should be emphasized that these contraindications have been developed as guidelines, and that a patient's participation in circuit weight training should be contingent on approval of the medical director and his or her personal physician. In our cardiac rehabilitation program, we have adopted a more conservative approach. Because patients with poor LV function may develop wall-motion abnormalities

TABLE II
DESCRIPTION OF EXERCISE PROGRAMS, SUBJECTS, AND RESULTS FOR STUDIES INVOLVING RESISTANCE TRAINING IN CARDIAC PATIENTS

	Exercise Program				
Study	Duration (wk)	Frequency (sessions/wk)	Sessions (min)	Type of exercise description	Intensity
Kelemen et al.[2]	10	3	50	CWT W, J	40% of 1RM 85% HR_{max}
Sparling et al.[5]	26	3	30-50	CWT W, J, S, C	30-40% of 1RM 70-85% HR_{max}
Stewart et al.[3]	156	3	50	CWT W, J	40% of 1RM 85% HR_{max}
Ghilarducci et al.[4]	10	3	60	CWT W, J, C	80% of 1RM 60-80% $\dot{V}O_{2max}$

	Patients			
	n	$\bar{x}$ age (yr)	Clinical status	Results
Kelemen et al.[2]	20	55 ± 9	MI, CABGS, A	24% increase in strength 12% increase in treadmill time
Sparling et al.[5]	16	56 ± 7	MI, CABGS, HR	22% increase in strength
Stewart et al.[3]	17	58 ± 8	MI, CABGS, A	13% increase in arm strength 40% increase in leg strength
Ghilarducci et al.[4]	9	57	MI, CABGS, A	29% increase in strength 33% increase in sit-ups (1 min)

CWT: circuit weight training; W: walking; J: jogging; S: swimming; C: stationary cycle ergometry: HR: heart rate; 1RM: one repetition maximum; $\dot{V}O_2$: oxygen uptake: MI: myocardial infarction; CABGS: coronary artery bypass graft surgery; A: angina; HR: high risk.

or significant arrhythmias during isometric or isodynamic exertion,[16,43,44] we include moderate to good LV function (ejection fraction ≥45%) and cardiorespiratory fitness (*i.e.*, completion of Stage III, Bruce protocol) without ischemic ST segment depression as additional prerequisites for participation.[46] Such characteristics are compatible with low-risk status after an acute coronary event.[30]

PRESCRIPTIVE GUIDELINES

Resistance training should be considered an adjunct to the patient's aerobic exercise regimen rather than the primary mode of conditioning. Guidelines for circuit weight training, involving series of five to twelve exercise stations that are performed sequentially, are provided in Table III, with specific reference to appropriate sets/repetitions, progression, technique, and safety considerations. Stations may include biceps curl, military press, bench press, leg press, triceps pressdown, lateral pulldown, back extensions, double leg curl, and double leg extensions. Machine weights such as Nautilus, Universal (Downsview, Ontario, Canada), Cybex Eagle (Ronkonkoma, NY), and Hydra-Fitness (Belton, TX) are preferable to free weights (barbells, dumbbells). Such equipment is conductive to circuit weight training, safer to use, and more time-efficient for the patient.

Initial testing of the patient is recommended to establish weight loads for training. Lifts should involve smooth, controlled movements with no breath holding or isometric straining. As an alternative to having the patient perform a 1RM, Sparling and associates[5] suggest that a light weight be chosen and progressively incremented as few times as possible to determine the greatest load that the patient can lift at least twice, but no more than three times. This weight load is estimated to be 90% of 1RM.[56] Using this 90% value, a 1RM (100%) is calculated and used to establish the training weights.

Orientation to the strength training program should be done individually for each patient and include instruction and demonstration on setting the resistance, proper body mechanics, optimal range of motion, and

TABLE III
WEIGHT TRAINING GUIDELINES FOR LOW-RISK* CARDIAC PATIENTS†

- To prevent soreness and injury, initially choose a weight that will allow the performance of 12 to 15 repetitions comfortably, corresponding to approximately 30% to 50% of the maximum weight load that can be lifted in one repetition. (Note: Selected stable, aerobically trained cardiac patients may eventually use loads corresponding to a more traditional program of weight training [*i.e.*, 60%-80% of 1RM])
- Perform one to three sets of each exercise.
- Avoid straining. Ratings of perceived exertion (6-20 scale) should not exceed fairly light to somewhat hard during lifting.
- Exhale (blow out) during the exertion phase of the lift. For example, exhale when pushing a weight stack overhead and inhale when lowering it.
- Increase weight loads by 5 to 10 pounds when 12 to 15 repetitions can be comfortably accomplished.
- Raise weights with slow, controlled movements; emphasize complete extension of the limbs when lifting.
- Exercise large muscle groups before small muscle groups. Include devices (exercises) for the upper and lower extremities.
- Weight train at least 2 to 3 times per week.
- Loosely hold hand grips when possible; sustained, tight gripping may evoke an excessive blood pressure response to lifting.
- Stop exercise in the event of warning signs or symptoms, especially dizziness, arrhythmias, unusual shortness of breath, and/or angina pectoris.
- Allow minimal rest periods between exercises (*e.g.*, 30-60 sec) to maximize muscular endurance and aerobic training benefits.[52,55]

*Arbitrarily defined as individuals with good left ventricular function (*i.e.*, ejection fraction ≥45%) and reasonable cardiorespiratory fitness (≥7 METS) without ischemic ST segment depression, hypotensive or hypertensive blood pressure responses, serious ventricular arrhythmias, or symptoms.
†Adapted from the American Association of Cardiovascular and Pulmonary Rehabilitation: Guidelines for Cardiac Rehabilitation Programs. Champaign, Illinois: Human Kinetics Publishers, 1991.

breathing pattern. After this session, a staff member should calculate and record training weight loads on the patient's strength conditioning log.

Supervision and Monitoring

A staff person should be designated to supervise the strength training area, periodically monitoring the patient's blood pressure and heart rate. Ensuring that the patients are using proper lifting technique and weight loads, while avoiding the hemodynamic consequences of expiratory strain, is also important. These concerns can be addressed by posting instructional signs in the strength training area and by systematically reviewing the strength training logs that are completed by the patient. Intensity violators can be identified at this time.

SUMMARY AND CONCLUSIONS

The patient with CAD requires a minimum level of strength for daily living, similar to persons without heart disease. Many cardiac patients lack the physical strength to perform common tasks like carrying luggage, opening windows, or doing yard work. Of those patients who possess the requisite physical capacity, many lack the confidence to attempt activities involving even low levels of muscular exertion.

Mild-to-moderate resistance training can provide an effective method for improving strength and cardiovascular endurance, modifying coronary risk factors, and enhancing psychosocial well-being in cardiac patients. These adaptations should serve to maximize the crossover of training benefits to real-life situations, enabling the patient to better perform occupational and leisure activities.

The safety of resistance exercise is attributed, in part, to the fact that heart rate and blood pressure responses are not exacerbated beyond clinically acceptable levels. Nevertheless, proper preliminary screening, appropriate prescriptive guidelines, and careful supervision are also important.

Because long-term compliance remains a problem for exercise-based cardiac rehabilitation programs, resistance training can provide a means for maintaining interest and increasing diversity. It should serve as a supplement to, rather than a replacement for, the patient's aerobic exercise prescription.

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Diagnostic and functional exercise testing: Test selection and interpretation

Exercise stress tests are useful in many areas of medical practice and research. The results extend the clinical significance of information obtained from other sources (ie, detailed history, thorough physical examination, resting electrocardiogram, chest radiograph, and basic laboratory analyses) and serve as a diagnostic, prognostic, and therapeutic guide. Leg or arm ergometry is commonly used to assess a patient's functional status, diagnose relative myocardial ischemia, and investigate physiologic mechanisms of cardiac symptoms. The responses may also be used to determine the effects of interventions such as coronary artery bypass surgery, percutaneous transluminal coronary angioplasty, medications, or exercise training. Key words: *aerobic, electrocardiogram, ergometer, maximal oxygen consumption, myocardial ischemia, risk stratification.*

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ACCORDING TO a World Health Organization (WHO) Expert Committee on Rehabilitation, "the primary purpose of an exercise test is to determine the responses of the individual to efforts at given levels and from this information to estimate probable performance in specific life and occupational situations."[1(p270)] Exercise testing of the cardiac patient permits evaluation of the aerobic capacity of the body (ie, the peak or maximal oxygen uptake [$\dot{V}O_2max$]); hemodynamics, assessed by the heart rate and systolic/diastolic blood pressure responses; limiting clinical signs or symptoms; and associated changes in electrical functions of the heart, especially supraventricular and ventricular dysrhythmias and ST segment displacement.[2]

This article addresses the physiologic basis and rationale for exercise testing in the assessment of cardiac patients after hospital discharge, with specific reference to indications and contraindications, test selection and interpretation, diagnostic versus functional testing, risk stratification, and the conduct and supervision of these studies.

INDICATIONS

Exercise stress testing is generally recommended for one or more of the following reasons:

- to aid in the diagnosis of hidden or occult coronary artery disease (CAD) in asymptomatic or symptomatic individuals;
- to evaluate cardiopulmonary fitness;
- to assess the efficacy of interventions such as coronary artery bypass surgery, percutaneous transluminal coronary angioplasty, medications, or physical conditioning;
- to assess the safety of vigorous physical exertion;

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- to formulate an effective exercise prescription; and
- to assess work-related capabilities.

However, the need for exercise testing as a routine screening procedure for asymptomatic adult exercisers has been questioned.[3] One solution to this dilemma is to categorize patients according to age, coronary risk factors, and the presence or suspicion of CAD and to screen them according to the category in which they are placed. Accordingly, the American College of Sports Medicine[4] recommends maximal exercise stress tests for the following individuals:

- apparently healthy men older than 40 and women older than 50 who want to begin a program of vigorous exercise (activity performed above 60% of aerobic capacity [$\dot{V}O_2$max]);
- asymptomatic men and women with two or more major coronary risk factors (ie, elevated cholesterol, hypertension, cigarette smoking, sedentary life style) who wish to take up vigorous exercise; and
- individuals who have—or have symptoms suggestive of—cardiac, pulmonary, or metabolic disease.

Informed consent obtained prior to the test, safety precautions, trained personnel, a defibrillator, and emergency equipment are essential prerequisites when using exercise testing to evaluate the patient.

CONTRAINDICATIONS

Although the complications associated with exercise testing are relatively low (0.5 deaths per 10,000 tests [0.005%] and 8.86 complications per 10,000 tests [0.0886%]),[5] the ability to maintain a high degree of safety depends on knowing when not to perform the test (ie, absolute and/or relative contraindications) and when to terminate the test, and being prepared for any untoward event that may arise. Common contraindications to exercise testing include unstable angina, uncontrolled atrial or ventricular dysrhythmias that may compromise cardiac function, acute congestive heart failure, severe aortic stenosis, acute infection, third-degree heart block (without pacemaker), active myocarditis or pericarditis, and a recent significant change in the electrocardiogram (ECG).[4] However, some of these traditional contraindications to exercise stress testing now appear to be unwarranted. For example, the safety of low-level and symptom-limited exercise testing soon (3 to 14 days) after "uncomplicated" myocardial infarction has been documented.[6] These data are used to assess a patient's functional status and as a diagnostic, prognostic, and therapeutic guide.

END POINTS FOR TESTING

Commonly used criteria for discontinuing an exercise test include attaining a predetermined end point of submaximal performance (eg, ≥70% or 85% of age-predicted maximal heart rate, perceived exertion ≥ "somewhat hard"), evidence of peak or maximal performance, or emergence of abnormal signs or symptoms.[2] Abnormal clinical signs include marked dyspnea, pallor, and central nervous system dysfunction (ataxia, staggering, failure to respond to questions). Abnormal physical signs include exertional hypotension, excessive increase of systolic blood pressure (> 250 mm Hg), and ECG abnormalities (ie, serious ventricular dysrhythmias, development of left bundle branch block, or marked ST segment displacement [> 2 mm]).[4] Limiting symptoms include increasing chest pain or discomfort (with or without ECG changes), lightheadedness, dizziness, or other indications that the patient can no longer continue. The patient's rating of perceived

exertion can be especially helpful in this regard. Ratings ≥ 17 (very hard) or 7 (very strong) on the category and category-ratio scales, respectively, signify near-maximal to maximal exertion (Table 1).[7]

LEG ERGOMETRY

Standard lower extremity exercise tests, using either the cycle ergometer or the treadmill, have the advantage of reproducibility and quantitation of physiologic responses to known external work loads.

Cycle ergometry

Cycle ergometry is an alternative to treadmill testing for those patients who have musculoskeletal, peripheral vascular, or neurologic limitations that restrict weight bearing. Mechanically braked ergometers require that a specified pedaling rate be maintained to keep the work rate, expressed as kilogram meters per minute ($kg \cdot m \cdot min^{-1}$) or watts ($1 W \sim 6 kg \cdot m\ m^{-1}$), whereas electronically braked ergometers automatically adjust internal resistance (in kilograms) to maintain designated work rates according to the pedal speed (in revolutions per minute [rpm]). Regardless of the type of stationary cycle ergometer used for testing, the seat height should be set so that the knee is slightly flexed at full leg extension.

The cycle ergometer has several advantages. It is portable, requires less space, makes less noise, and generally costs less than the treadmill It also minimizes movement of the torso and arms, which may facilitate better quality ECG recordings and easier blood pressure measurements.[8] Its main disadvantage is that it is an unfamiliar method of exercise for many Americans and often results in limiting localized leg fatigue.[9]

Physiologic and clinical responses to exercise on a cycle ergometer may differ from those obtained on a treadmill. For

Table 1. Perceived exertion scales with descriptive effort ratings

Category scale		Category-ratio scale	
6		0	Nothing at all
7	Very, very light	0.5	Very, very weak
8		1	Very weak
9	Very light	2	Weak
10		3	Moderate
11	Fairly light	4	Somewhat strong
12		5	Strong
13	Somewhat hard	6	
14		7	Very strong
15	Hard	8	
16		9	
17	Very hard	10	Very, very strong (almost maximum)
18			
19	Very, very hard		Maximum
20			

Note: Shaded areas represent near-maximal to maximal exertion.
Source: Borg G. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982;14:377–381.

Physiologic and clinical responses to exercise on a cycle ergometer may differ from those obtained on a treadmill.

example, maximal oxygen uptake is typically 80% to 95% of that obtained during treadmill testing.[4] Furthermore, it remains unclear whether there is a significant difference between the two modalities in evaluating myocardial ischemia. Chest pain and ischemic ST segment depression are reported to occur less frequently with cycle ergometer exercise than with treadmill testing.[10] Wicks et al, however, reported no difference in the magnitude of ST segment depression evoked by these modalities.[11] In addition, the aerobic requirements of treadmill exercise are independent of body weight, whereas the oxygen cost of cycle ergometry is weight dependent.[4] Accordingly, a given work load on the treadmill (eg, 3 mph, 0% grade) requires approximately 3 metabolic equivalents (METs), or 10.5 mL kg^{-1} min^{-1} for all persons, regardless of body weight. However, a cycle ergometer work rate of 900 kg m $\cdot$ min^{-1} requires approximately 2,100 mL O_2 min^{-1} for all persons, corresponding to 21.0 mL $\cdot$ kg^{-1} $\cdot$ min^{-1}, or 6 METs, for a 100-kg person, and 35.0 mL kg^{-1} min^{-1}, or 10 METs, for a 60-kg person (Table 2).[4]

Treadmill testing

Treadmill testing provides a more common form of physiologic stress (ie, walking) in which subjects are likely to attain a slightly higher maximal oxygen uptake and heart rate than that obtained in cycle ergometry. The treadmill should be electrically driven, accommodate a variety of body weights up to at least 157.5 kg (350 lb), have a wide range of speeds (1 to 8 mph), and offer a progressive incline or grade (from no grade to 20% grade).[12] Side platforms are recommended to allow the patient to adapt to the moving belt before fully stepping onto it.

The treadmill protocol should generally last 8 to 12 minutes for patients limited by fatigue, and all patients should reach their peak performance by 15 minutes.[13] Fig 1 shows three commonly used multistage treadmill exercise protocols. Exercise stages are progressive in intensity; a duration at each stage of 2 minutes or more ensures that most cardiorespiratory variables reach a "steady-state" value. These protocols in-

Table 2. Approximate energy expenditure in METs* during cycle ergometry

Body weight		Work rate (kg m min^{-1})						
kg	lb	300	450	600	750	900	1,050	1,200
50	110	5.1	6.9	8.6	10.3	12.0	13.7	15.4
60	132	4.3	5.7	7.1	8.6	10.0	11.4	12.9
70	154	3.7	4.9	6.1	7.3	8.6	9.8	11.0
80	176	3.2	4.3	5.4	6.4	7.5	8.6	9.6
90	198	2.9	3.8	4.8	5.7	6.7	7.6	8.6
100	220	2.6	3.4	4.3	5.1	6.0	6.9	7.7

*1 MET = 3 5 mL kg^{-1} min^{-1}

Source American College of Sports Medicine *Guidelines for Exercise Testing and Prescription* 4th ed. Philadelphia, Pa: Lea & Febinger, 1991

volve a constant walking speed (range, 2.0 to 3.4 mph) and standardized increases in grade or incline; nevertheless, the increments in aerobic requirements for each stage are identical (ie, 1 MET).

The conventional Bruce[14] treadmill protocol (Fig 2) is perhaps the most familiar and widely used because it offers a rapid and safe exercise progression for which normative values for heart rate, blood pressure, and oxygen uptake have been established. The protocol, however, has several limitations. The initial work load (stage I), corresponding to 1.7 mph, 10% grade, has an aerobic requirement of 4 to 5 METs, which exceeds the aerobic capacity of many cardiac patients. Because the work load progression involves simultaneous increases in both speed and grade, it is sometimes difficult for patients to adapt to the large work increments (typically 2.5 to 3.0 METs) between stages. Consequently, patients often fail to demonstrate a leveling off or plateau in physiologic responses, and delineation of the precise MET level at the ischemic or anginal threshold is difficult. In addition, the Bruce protocol is a walk-jog test with a variable transition point from walking to jogging at stage IV (9th to 12th minute), resulting in a variety of mechanical efficiencies at this stage, with varying oxygen costs. Finally, the ECG may be distorted by artifact caused by muscle movement and foot impact during jogging.

A recent advance in test methodology that can overcome many of the limitations of multistage exercise tests is ramping.[15] Ramp protocols involve a nearly continuous and uniform increase in aerobic requirements that replaces the "staging" used in conventional exercise tests. With ramping, the gradual increase in demand allows a steady increase in cardiorespiratory and hemodynamic responses. Protocols have been developed for use with both the cycle ergometer and the treadmill that provide

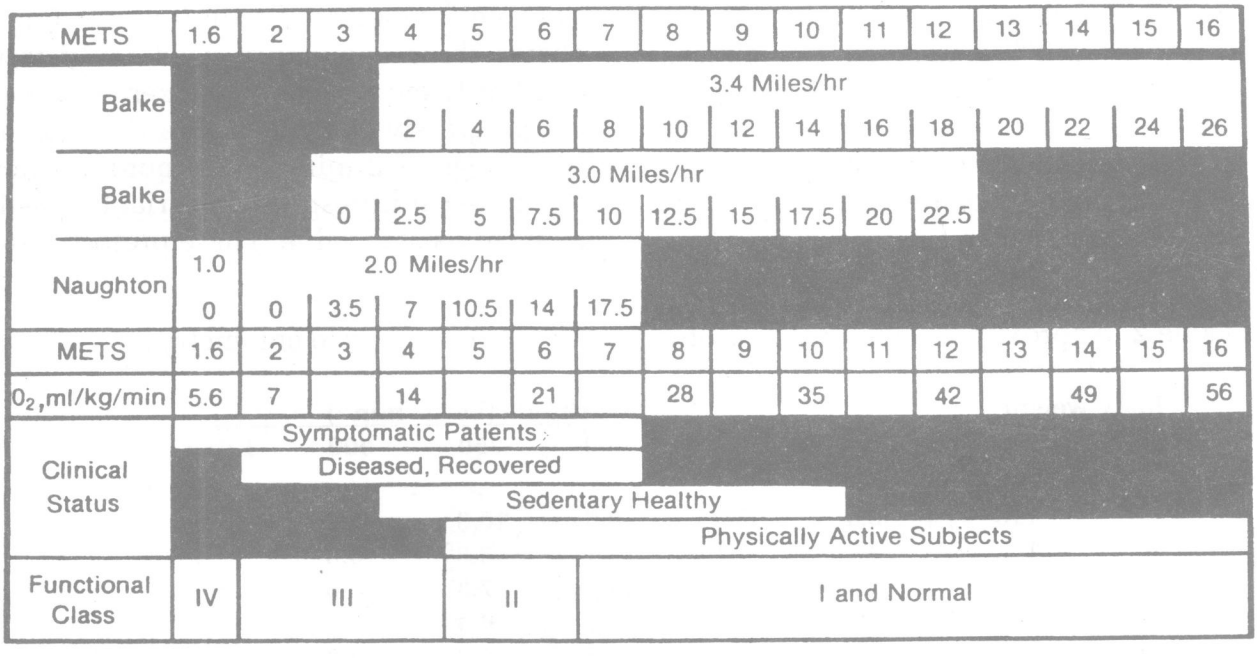

Fig 1. Metabolic cost of three common treadmill protocols; 1 MET signifies resting energy expenditure, equivalent to approximately 3.5 $mL \cdot kg^{-1} \cdot min^{-1}$. Unlabeled numbers refer to the treadmill grade, expressed as a percentage. The patient's clinical status and functional class (I–IV) for the peak attained work load are also shown.

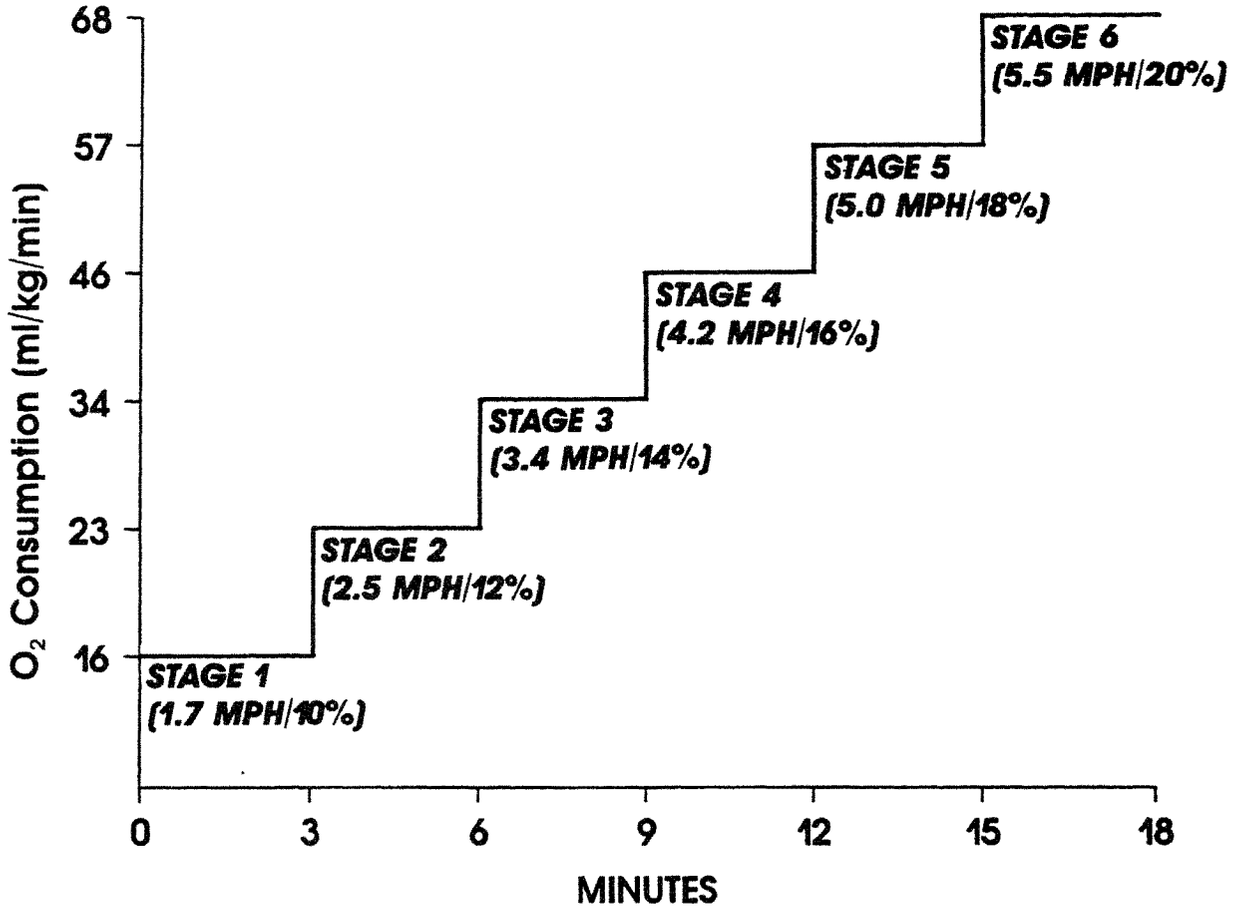

Fig 2. The standard Bruce treadmill protocol showing progressive stages (speed, percentage grade) and the corresponding aerobic requirement, expressed as $mL \cdot kg^{-1} \cdot min^{-1}$.

for ramping increments appropriate to the wide range of patient exercise capacities.

ARM ERGOMETRY

Arm ergometry provides a reproducible, noninvasive method of evaluating cardiovascular function in patients who are unable to perform treadmill or cycle ergometer exercise owing to neurologic, vascular, or orthopedic limitations (Table 3). In addition, arm exercise testing appears to be the functional evaluation of choice for persons whose occupational and recreational physical activity is dominated by upper extremity efforts.[16]

Dynamic arm exercise testing has been shown to offer a satisfactory but perhaps less sensitive alternative to leg ergometry for the detection of ischemic ST segment depression, the provocation of angina pectoris, or both.[17] This may be attributed, at least in part, to the fact that maximal heart rate and systolic blood pressure are generally greater during leg exercise than during arm exercise. As a result, the maximal rate pressure product and myocardial oxygen demand are probably lower during arm cranking. Nevertheless, arm exercise, coupled with thallous (thallium) chloride-210 scintigraphy, appears to be an effective method of detecting myocardial ischemia

and assessing prognosis in patients at increased risk for CAD.[18]

Methods and protocols

We have used a modified leg cycle ergometer for arm cranking, as previously described.[19] The arm ergometer is mounted on a table so that the patient can perform arm cranking while seated upright, with the feet flat on the floor. The ergometer should be positioned so that the midpoint of the sprocket wheel is at shoulder level. During cranking, the arms are alternately extended at right angles to the body, allowing for a slight bend at the elbow at maximal reach.

Selection of an appropriate protocol must consider that submaximal arm exercise is performed at a greater physiologic cost than leg exercise, but maximal responses are generally lower during arm exercise. Because a smaller muscle mass is used in arm cycle ergometer testing, and because most persons are not physically conditioned for sustained upper extremity exercise, low initial work loads (≤200 $kg \cdot m \cdot min^{-1}$) and small work load increases per stage (100 to 150 $kg \cdot m \cdot min^{-1}$) are recommended.[16] The testing protocol may consist of continuous or intermittent progressive exercise, with each exercise stage lasting 2 to 3 minutes; intermittent protocols typically allow 1 to 2 minutes of rest between stages. Peak effort is defined as the power output at which the patient is no longer able to maintain the designated pedal speed (generally 40 to 60 rpm) or the work rate at which significant clinical signs or symptoms develop.

Although the methodology of arm crank ergometry is well established, certain technical limitations remain. Because of motion artifact, satisfactory diagnostic ECGs are best obtained by having the patient pause briefly between stages. Blood pressure measurements are generally obtained by measuring pressure in the inactive arm

Table 3. Indications for upper extremity exercise tests

Type of test	Equipment	Objectives/evaluations	Applications
Rhythmic, isotonic, upper extremities	Modified Arm-Crank Ergometer, Monarch Rehab Trainer, Schwinn Air-Dyne Ergometric Exerciser	To determine submaximal and maximal cardiorespiratory and hemodynamic responses to sustained upper extremity exertion	*Occupations:* Sawing, machine operation, manual labor, ditch digging, landscaping *Recreation:* Swimming, canoeing, cross-country skiing, paddleball *Clinical:* Patients with intermittent claudication, orthopedic/arthritic limitations, paraplegia

while the patient continues cranking with the other, or having the patient crank with both arms and then measuring blood pressure while the patient pauses briefly between stages. Unfortunately, both methods have potential drawbacks. High work rates may be difficult to maintain with single-arm cranking, whereas systolic blood pressures taken by the standard cuff method immediately after arm cranking are likely to underestimate true physiologic responses.[20]

Aerobic requirements of arm ergometry

Previous studies in men[21] showed that the regression of oxygen uptake ($\dot{V}O_2$) on power output during arm ergometry was $y = 3.06\ x + 191$ ($y = \dot{V}O_2$ in mL min^{-1}; x = power output in $kg \cdot m \cdot min^{-1}$). Absolute arm $\dot{V}O_2$ ($mL \cdot min^{-1}$) at a given work rate demonstrated the smallest variability between patients; Table 4 was developed to predict arm $\dot{V}O_2$ in $mL \cdot kg^{-1} \cdot min^{-1}$, based on a constant absolute $\dot{V}O_2$ with increasing body weight (50 to 110 kg). More recently, Balady et al[22] reported separate regression equations for predicting oxygen consumption during arm ergometry for healthy men and women.

FUNCTIONAL EXERCISE TESTING

Although many clinicians view the exercise test primarily as a source of diagnostic (ECG) information, exercise testing also yields valuable data regarding the patient's functional capacity for occupational or recreational activities. This assessment is based on the oxygen cost required to perform a given external work rate, most accurately determined from direct measurements of minute ventilation and expired gas composition (O_2 and CO_2) at peak exercise. The most widely recognized measure of cardiopulmonary fitness is the maximal oxygen consumption ($\dot{V}O_2max$).[23] This variable is defined physiologically as the highest rate of oxygen transport and use that can be achieved at peak physical exertion.

Table 4. Aerobic requirements of arm ergometry*

Work rate* ($kg \cdot m \cdot min^{-1}$)		150	300	450	600	750
$\dot{V}O_2$ ($mL\ min^{-1}$)		648	1,104	1,562	2,079	2,431
Body weight						
lb	**kg**	**Oxygen consumption ($mL\ kg^{-1} \cdot min^{-1}$)**				
110	50	13.0	22.1	31.2	41.7	48.7
132	60	10.9	18.6	25.9	34.7	40.6
154	70	9.1	15.8	22.4	29.8	34.7
176	80	8.1	13.7	19.6	25.9	30.5
198	90	7.4	12.3	17.5	23.1	27.0
220	100	6.7	11.2	15.8	20.7	24.2
242	110	6.0	10.2	14.4	18.9	22.1

*Table discontinued above 750 kg m min^{-1} owing to small sample size.
Source: Franklin BA, Vander L, Wrisley D, Rubenfire M. Aerobic requirements of arm ergometry: implications for exercise testing and training. *Phys Sportsmed.* 1983;11:81–90.

Somatic oxygen consumption ($\dot{V}O_2$) may be expressed mathematically by a rearrangement of the Fick equation:

$$\dot{V}O_2 = HR \times SV \times (CaO_2 - C\bar{v}O_2)$$

where $\dot{V}O_2$ is oxygen consumption in milliliters per minute, HR is heart rate in beats per minute, SV is stroke volume in milliliters per beat, and $CaO_2 - C\bar{v}O_2$ is the arteriovenous oxygen difference in milliliters of oxygen per deciliter of blood. Thus, both central and peripheral regulatory mechanisms affect the magnitude of body oxygen consumption.

Determination of the $\dot{V}O_2$max

Maximal oxygen consumption is generally determined by measuring the volume and analyzing the oxygen content of expired air, corrected to standard temperature and pressure dry, using the following equation:

$$\dot{V}O_2 = \dot{V}E\ (FIO_2 - FEO_2)$$

where $\dot{V}E$ is the expired ventilation per minute; FEO_2 is the directly measured concentration of oxygen in expired air; and FIO_2 is the concentration of oxygen in the inspired air, normally 0.2093. Traditionally, this variable has been measured using an open circuit or Douglas bag technique. However, several automated systems are currently available to measure $\dot{V}O_2$ and related cardiorespiratory variables during exercise testing.

It is inconvenient to measure $\dot{V}O_2$ directly, because it requires sophisticated equipment, technical expertise, and frequent calibration. As a result, clinicians have increasingly sought to predict or estimate $\dot{V}O_2$max from the treadmill speed and percentage grade or from the cycle ergometer work rate, expressed as kilogram meters per minute. When these variables are defined, it is generally acknowledged that the external work rate, if performed for 2 or more minutes to attain a steady state, can be translated to an aerobic cost that will not differ significantly from one individual to another. It should be emphasized, however, that the cardiac patient's oxygen uptake may be markedly overestimated when it is predicted from the exercise time or work rate. Several explanations have been offered to account for the discrepancy.[24] The $\dot{V}O_2$ values for treadmill exercise presented in previously published tables and nomograms were generally obtained in healthy young adults and apply only when the patient has achieved steady-state work (eg, ≥2 minutes at the exercise stage) without holding the handrails. It has also been suggested that left ventricular dysfunction may slow oxygen uptake kinetics, perhaps accounting for the fact that cardiac patients often demonstrate lower submaximal and maximal oxygen uptake values and a larger oxygen debt for standard work rates.[25] Finally, β-adrenergic blocking medications have been shown to result in a slower adaptation of oxygen consumption to steady-state submaximal work rates.[26]

The cardiac patient's oxygen uptake may be markedly overestimated when it is predicted from the exercise time or work rate.

AEROBIC CAPACITY AND IMPAIRMENT

Aerobic capacity

Maximal oxygen consumption may be expressed on an absolute basis in liters per minute, reflecting total body energy output and caloric expenditure, where each liter of oxygen consumed is equivalent to approximately 5 kcal. Because large persons usually have a large absolute oxygen con-

sumption simply by virtue of their large muscle mass, physiologists generally divide this value by body weight in kilograms to allow a more equitable comparison between individuals of different size. This variable, when expressed in milliliters of oxygen per kilogram of body weight per minute ($mL \cdot kg^{-1} \cdot min^{-1}$) or as METs (1 MET = 3.5 $mL \cdot kg^{-1} \cdot min^{-1}$), is widely considered the single best index of physical work capacity or cardiopulmonary fitness.[27]

Functional aerobic impairment

It is useful to express the $\dot{V}O_2max$ in milliliters per kilogram per minute as compared with normative values. Bruce et al,[14] developed the concept of functional aerobic impairment (FAI) for this purpose. The FAI is the percentage difference between a person's observed $\dot{V}O_2max$, either measured directly or estimated, and the $\dot{V}O_2max$ predicted for a healthy person of the same age, gender, and habitual activity status. Average predicted values of $\dot{V}O_2max$ according to age for active and sedentary men and women are shown in Table 5.[14]

FAI and functional aerobic capacity (FAC) can be calculated from the following formulas:

$$\% \text{ FAI} = \frac{\text{Predicted } \dot{V}O_2max - \text{Observed } \dot{V}O_2max}{\text{Predicted } \dot{V}O_2max} \times 100$$

$$\% \text{ FAC} = 100 - \text{FAI}$$

The normal value for the FAI is 0%; this indicates that the $\dot{V}O_2max$ is 100% of the age- and sex-predicted (FAC) value, and there is no functional impairment. Negative values for FAI signify above-average fitness (ie, FAC is more than 100%). The degree of FAI can be categorized as mild (27% to 40% FAI), moderate (41% to 54%), marked (55% to 68%), or extreme (more than 68% FAI), corresponding to 73% to 60%, 59% to 46%, 45% to 32%, and less than 32% FAC, respectively.

The concept of FAI is particularly useful when making serial evaluations of individuals as well as comparisons with peers. For example, a 50-year-old sedentary man with a $\dot{V}O_2max$ of 24.5 $mL \cdot kg^{-1} \cdot min^{-1}$ had an FAI of 31.2% ([35.6 – 24.5]/35.6} x 100 =

Table 5. Average $\dot{V}O_2max$ values ($mL \cdot kg^{-1} \cdot min^{-1}$) for healthy active and sedentary men and women according to age*

Age (y)	Men		Women	
	Active 69.7 – 0.612 y‡	Sedentary† 57.8 – 0.445 y‡	Active 42.9 – 0.312 y‡	Sedentary† 42.3 – 0.356 y‡
20	57.5	48.9	36.7	35.2
30	51.3	44.5	33.5	31.6
40	45.2	40.0	30.4	28.1
50	39.1	35.6	27.3	24.5
60	33.0	31.1	24.2	20.9
70	26.9	26.7	21.1	17.4

*$\dot{V}O_2max$ for any age can be predicted using the above-referenced regression equations from Bruce RA, Kusumi F, Hosmer D. Maximal oxygen intake and nomographic assessment of functional aerobic impairment in cardiovascular disease. Am Heart J. 1973;85:546–562.

†Defined as subjects who do not exert themselves sufficiently to develop sweating at least once a week.

‡Regression formulas to predict $\dot{V}O_2max$ from age.

31.2%). In other words, his FAC was only 68.8% of the average normal expected value, corresponding to "mild" fitness impairment. Four years later his $\dot{V}O_2$max had increased to 30.6 mL·kg^{-1}·min^{-1} as a result of participating in a gymnasium-based exercise training program. Although the increase in $\dot{V}O_2$max was 6.1 mL·kg^{-1}·min^{-1}, equivalent to 24.9% (6.1/24.5 x 100), the age-corrected FAI had improved from 31.2% to 9.4%. Accordingly, his FAC had increased from 68.8% at age 50 to 90.6% at age 54.

DIAGNOSTIC EXERCISE TESTING

Exercise stress testing is one of the most common evaluations performed in the assessment of persons with known or suspected CAD. The test is based primarily on the ECG response to exercise, with 1 mm or more ST segment depression at 80 ms beyond the J point as an indicator of myocardial ischemia (Fig 3). Although monitoring of 12 or more leads is recommended by some clinicians, we have found that recording three leads (V_1, V_5, and a VF) is usually adequate for most clinical situations. Furthermore, a single precordial lead (V_5) has been shown to reveal ST segment depression in about 80% of all instances detected with a multiple lead system.[28]

Unfortunately, the conventional exercise ECG has significant limitations in the diagnosis of occult CAD, with an approximate sensitivity and specificity of 75% and 85%, respectively. In some persons, exercise-induced ST segment depression may suggest myocardial ischemia and underlying heart disease when, in fact, no disease is present. This scenario, termed a *false-positive response*, occurs predominantly in populations with a low pretest likelihood of CAD (eg, young adults, asymptomatic women). Conversely, when a patient is found to have significant CAD and fails to demonstrate exercise-induced ST segment depression, his or her test is classified as a *false-negative test.*

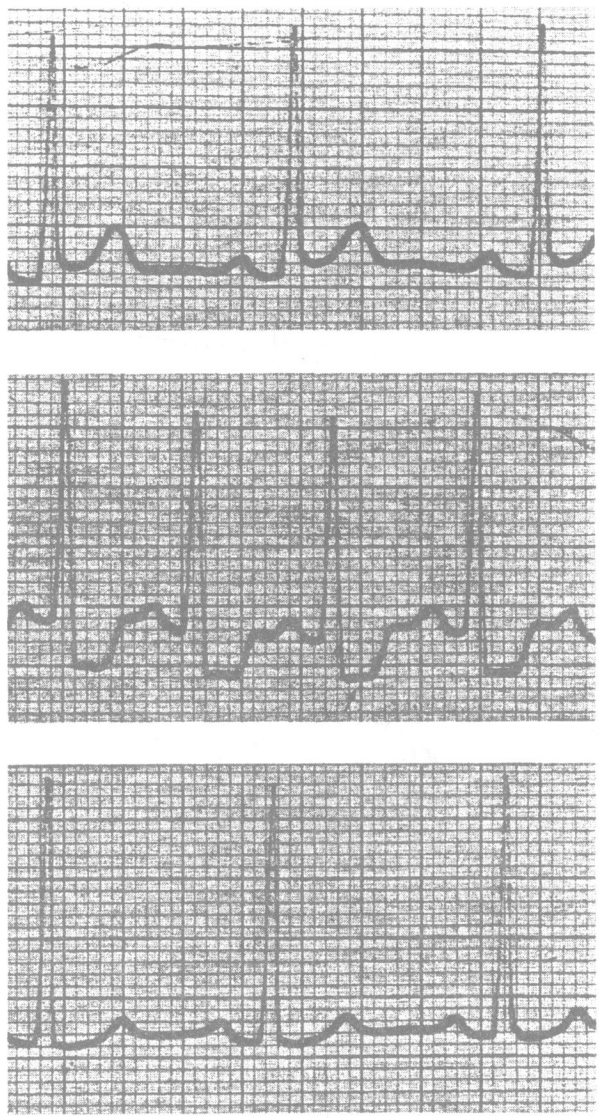

Fig 3. (Top) A patient's resting ECG (lead V_5) taken before exercise testing. **(Middle)** ECG obtained after several minutes of an exercise test, showing significant ST segment depression (*arrow*). **(Bottom)** Resting ECG recorded 6 minutes after exercise, representative of a normal configuration.

Pre- and posttest likelihood of heart disease

Clinicians currently use three variables—age, gender, and symptoms—to estimate a

person's pretest likelihood of CAD.[29] However, the risk may be even further defined by the major coronary risk factors, including blood pressure, smoking status, sedentary life style, and lipid-lipoprotein profile. As can be seen in Table 6, an asymptomatic 45-year-old woman has only a 1% chance of having significant CAD, whereas a 65-year-old man with typical angina (ie, substernal chest discomfort during heavy physical exertion) has a high (94%) pretest risk of heart disease.

The posttest likelihood of CAD is determined by the pretest risk and the results of the exercise test. When the pretest risk of CAD is either very high or very low, as in the previous examples, a normal or abnormal exercise ECG response has little influence on the posttest likelihood of disease. Exercise testing has the greatest diagnostic impact in persons with an intermediate likelihood of CAD (ie, in the 30% to 70% range of pretest probability). For example, a 55-year-old man with atypical angina has a 59% likelihood of significant CAD before exercise testing. After an exercise ECG, his posttest likelihood of CAD is either 90% or 30% according to the presence or absence, respectively, of significant ST segment depression (Fig 4).[30] Thus, by applying Bayesian analyses, the need for additional diagnostic studies (eg, exercise testing with myocardial perfusion imaging) can be defined more intelligently.

Radionuclide methods for CAD detection

Limitations in the conventional exercise ECG have led to the development of two noninvasive radionuclide methods for CAD detection: those that assess myocardial perfusion and those that evaluate ventricular function. These tests, which are superior to the conventional exercise ECG in terms of sensitivity, specificity, and predictive accuracy, are often used as a follow-up when patients demonstrate exercise-induced ST segment depression and/or angina pectoris. Such tests may also be of particular value in patients taking diuretics, digitalis preparations, or estrogen, as these compounds may make the exercise ECG uninterpretable with respect to evidence of myocardial ischemia. Additional variables that may contribute to spurious ST segment depression during exercise include baseline ECG abnormalities (eg, ST segment depression, left ventricular hypertrophy, left bundle branch block, the preexcitation [Wolff-Parkinson-White {WPW}] syndrome), and mitral valve prolapse.

One of the most useful evaluations is the myocardial perfusion scan, in which con-

Table 6. Pretest likelihood of CAD in patients by age, gender, and symptoms

Age (y)	Asymptomatic		Nonanginal chest pain		Atypical angina		Typical angina	
	Men	Women	Men	Women	Men	Women	Men	Women
35	1.9	0.3	5.2	0.8	21.8	4.2	69.7	25.8
45	5.5	1.0	14.1	2.8	46.1	13.3	87.3	55.2
55	9.7	3.2	21.5	8.4	58.9	32.4	92.0	79.4
65	12.3	7.5	28.1	18.6	67.1	54.4	94.3	90.6

Source Diamond GA, Forrester JS Analysis of probability as an aid in the clinical diagnosis of coronary artery disease *N Engl J Med* 1979,300 1,350–1,358

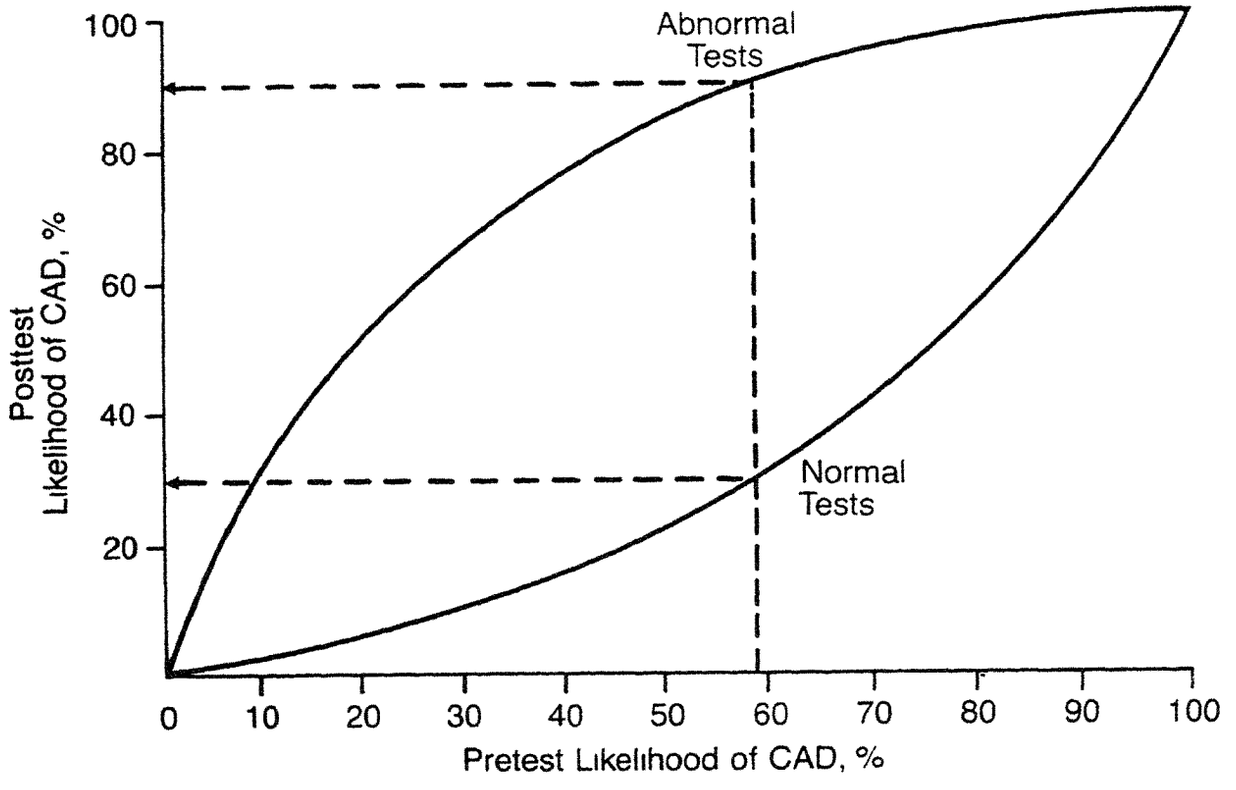

Fig 4. Impact of 59% likelihood of CAD on posttest likelihood of disease when exercise ECG is normal (30%) or abnormal (90%). The sensitivity of the exercise ECG is 75%, and its specificity is 85% Modified from Epstein SE. Implications of probability analysis on the strategy used for non-invasive detection of coronary artery disease. *Am J Cardiol.* 1980;46:491–499.

ventional exercise testing is combined with the injection of thallous (thallium) chloride-201 or technetium Tc99m sestamibi (Cardiolite) with post–stress test scintillation imaging. A set of resting images taken before or after exercise stress helps to differentiate regions of decreased isotopic uptake as being either a manifestation of exercise-induced ischemia or scar tissue from a previous myocardial infarction (Fig 5). Alternatively, clinicians can use radionuclide ventricular angiography to evaluate cardiac function. Exercise responses that are highly sensitive for the presence of CAD include the development of new regional wall motion abnormalities and the inability to augment ejection fraction by at least 5%.[31]

EXERCISE TESTING FOR RISK STRATIFICATION

Identification soon after myocardial infarction of patients who are at increased risk for subsequent cardiac events offers two major benefits: patients at moderate to high risk can be evaluated for more intensive pharmacotherapy, interventional cardiac catheterization, or revascularization, and patients at low risk can be spared immediate cardiac catheterization and unwarranted restriction of their vocational and leisure-time activities.[32] Stratification of patients into three main risk categories—low, moderate, and high—with associated first-year mortality rates of <2%, 10% to 25%, and >25%, respectively, is largely

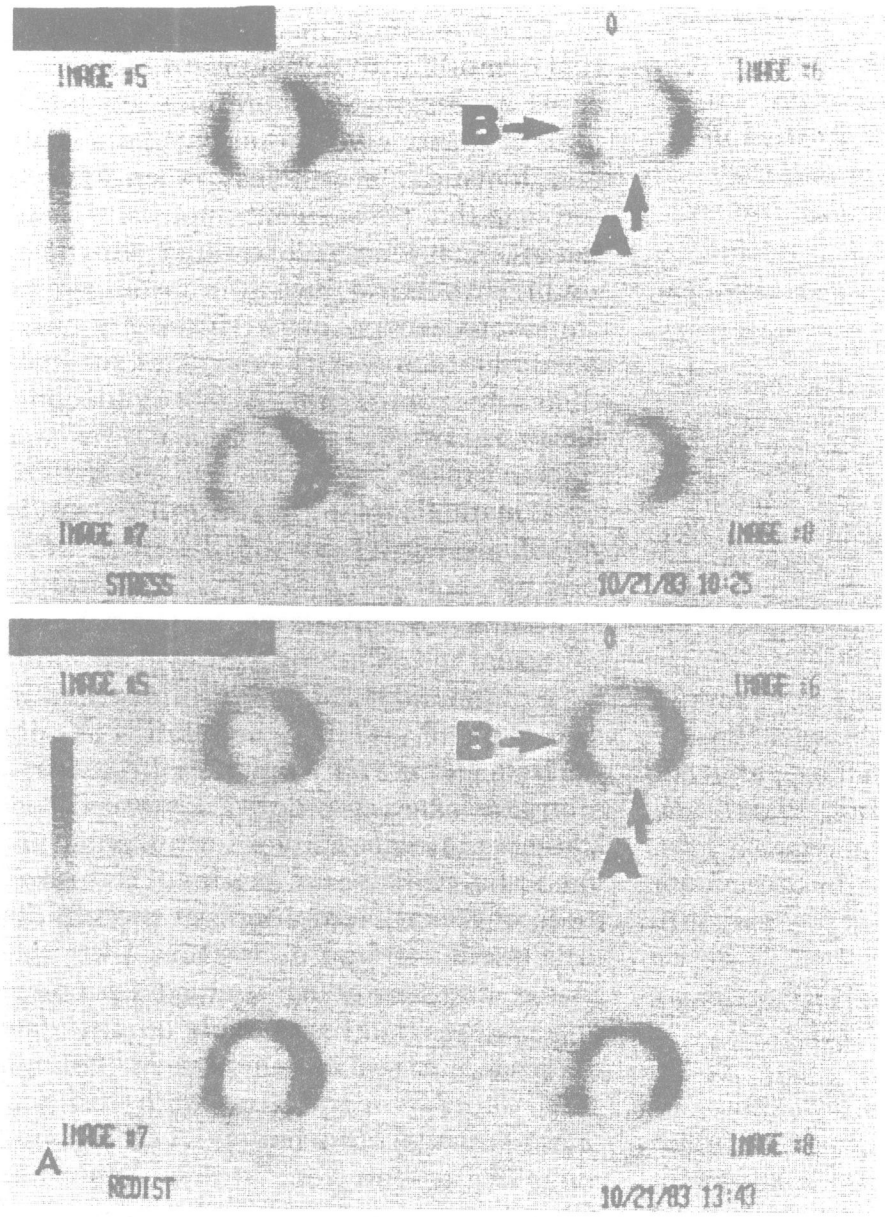

Fig 5. Thallium stress **(top)** and redistribution **(bottom)** images in a patient who had had a previous myocardial infarction in the inferior wall (A) with ischemia in the anteroseptal wall (B). After the patient rests, blood flow, and therefore thallous chloride uptake, increases in the ischemic area.

based on the extent of residual myocardial ischemia, manifested as resting or exertional angina pectoris, ST segment depression, or both, and the degree of left ventricular dysfunction, characterized by the ejection fraction.[33] Patients at moderate risk (ie, those demonstrating signs or symptoms of additional myocardium in jeopardy) are most likely to experience a reduction in mortality from coronary revascularization.

Although nearly half of the patients who will have reinfarction or die within the first year after acute myocardial infarction can be identified on the basis of severe ischemia or pump failure during hospitalization, there is a need to identify additional patients at increased risk who do not demonstrate these abnormalities. A variety of clinical observations and evaluations may be used to detect these abnormalities at

ECG responses to exercise tests should be interpreted according to the magnitude and configuration of ST segment displacement and the presence of supraventricular and ventricular dysrhythmias.

various times after acute myocardial infarction. For example, the Duke treadmill[34] score uses exercise time (minutes), ST segment displacement (millimeters), and an angina index (0, none; 1, occurring during treadmill test; 2, reason for terminating test):

Treadmill score = Exercise time – (5 x ST displacement) – (4 x Angina index)

The treadmill score added independent prognostic information to that provided by clinical data, coronary anatomy, and left ventricular ejection fraction, identifying patients at low (≥5 points), moderate (–10 to +4), and high (≤ –11) risk of subsequent cardiac events.

ECG RESPONSES TO EXERCISE TESTING

ECG responses to exercise tests should be interpreted according to the magnitude and configuration of ST segment displacement and the presence of supraventricular and ventricular dysrhythmias. Such information is useful in evaluating patients' clinical status as well as the efficacy of selected interventions, including coronary artery bypass surgery, percutaneous transluminal coronary angioplasty, medications, or exercise training.

ST segment depression

Traditionally, exercise-induced ST segment depression (especially horizontal or downsloping) has been considered diagnostic of CAD and quantitatively prognostic of morbidity and mortality. In such cases it presumably represents an imbalance between myocardial oxygen supply and demand. Nevertheless, recent reports indicate that ST segment depression does not localize ischemia to an area of myocardium.[15] Moreover, negative tests may be considered inconclusive when the peak heart rate achieved is <85% of the predicted maximum, because of inadequate cardiac stress.[8]

ST segment depression can be quantitated manually or, more precisely, with the aid of a computer. Although several methods are commonly used to evaluate an abnormal ECG response, we have found the ST segment index[35]—that is, the algebraic sum of the STJ segment depression in millimeters and the ST slope in millivolts per second—to be a valid and reliable method to assess exercise-induced myocardial ischemia. A negative index (ie, <0) is considered abnormal, assuming that the magnitude of ST segment depression (from the baseline to the J point) is at least 1.0 mm. For example, slow upsloping ST segment depression would be considered abnormal if the depression and slope were –2.0 mm and 1.0 mV/s, respectively. The ST segment index would be –1.0 in this case, suggesting an ischemic response (Fig 6).

ST segment elevation

In contrast to ST segment depression which is commonly induced by exercise in patients with CAD, ST segment elevation is an infrequent and often disregarded ECG response. Nevertheless, it is considered an ominous finding. In patients with a history of myocardial infarction and/or ECG Q waves in the lead corresponding to the ST segment elevation, the finding usually (in >75% of

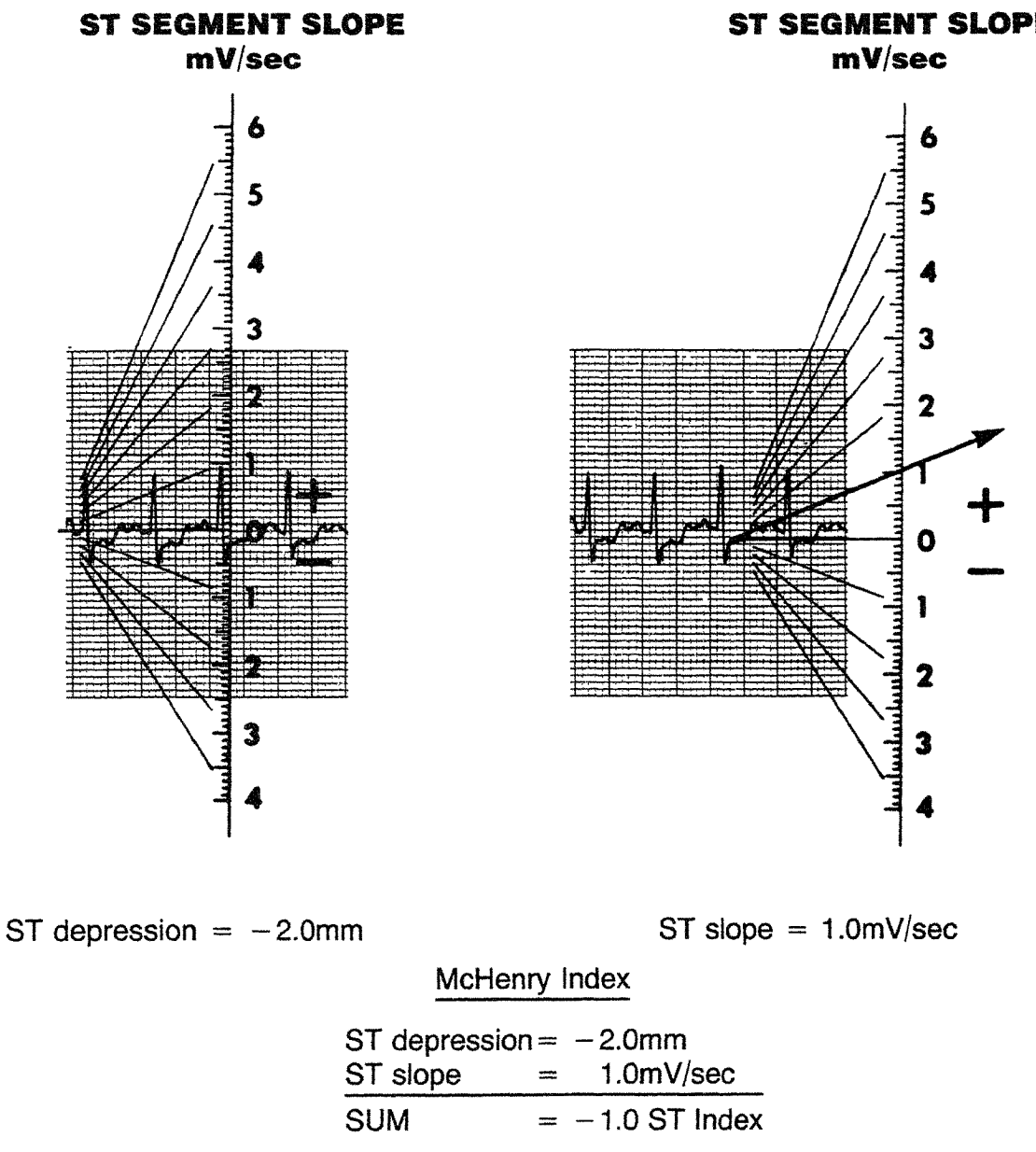

Fig 6. Example showing calculation of the ST segment index by using a transparent overlay to determine the ST segment slope and depression. An abnormal ECG response with upsloping ST segment depression is shown.

patients) reflects ventricular aneurysm, with associated poor ventricular function and poor patient prognosis.[36,37] However, the development of ST segment elevation during exercise in the absence of previous myocardial infarction suggests either severe coronary artery spasm or a fixed, high-grade coronary stenosis corresponding to the site of ischemia seen electrocardiographically.[38] The latter may represent more severe ischemia than ST segment depression, reflecting transmural rather than subendocardial ischemia.[39]

Supraventricular and ventricular dysrhythmias

Infrequent atrial or ventricular ectopic beats and short runs of supraventricular tachycardia commonly occur during exercise testing and do not appear to have diagnostic or prognostic significance for CAD. However, the suppression of resting ventricular dysrhythmias during exercise *does not* exclude the presence of underlying CAD; conversely, premature ventricular beats that increase in frequency, complexity, or both, do not necessarily signify underlying ischemic heart disease.[40] Threatening forms of ventricular ectopy, including paired or multiform ventricular premature beats, salvos, and ventricular tachycardia, are even more likely to be associated with significant CAD and a poor prognosis if they occur with ischemic ST segment depression, angina pectoris, or both.[8,41]

HEMODYNAMIC RESPONSES

Although ST segment displacement and angina pectoris have been the primary, and often sole, criteria for assessing effort-induced myocardial ischemia, the evaluation of hemodynamic responses (ie, heart rate and blood pressure) during exercise has been shown to enhance the predictive value of exercise testing.

Heart rate

The normal heart rate response to progressive exercise is a relatively linear increase, corresponding to 10 ± 2 beats/MET for sedentary subjects.[42] A patient with a markedly blunted heart rate response to exercise who is not taking β-blockers or related cardiac medications is said to have *chronotropic incompetence*, signified by a peak exercise heart rate that is two standard deviations or more (>20 beats/min) below the age-predicted maximal heart rate for normal subjects.[43–45] This finding during exercise, even in the absence of significant ST segment depression, has been associated with left ventricular dysfunction, multivessel CAD, and a higher subsequent morbidity and mortality rate.[46]

The age-predicted maximal heart rate can be estimated in one of the following two ways: 220 – age in years or 215 – (0.66 x age in years).[2] Because the maximal heart rate normally decreases with age, chronotropic impairment owing to heart disease or medications can be calculated by the following formula:

$$\text{Percentage chronotropic impairment} = \frac{a-b}{a} \times 100$$

where a = age-predicted maximal heart rate and b = heart rate attained at peak or maximal effort.

Blood pressure

Systolic blood pressure

The normal hemodynamic response to exercise is a progressive increase in systolic blood pressure, typically 10 ± 2 mm Hg/MET, with a possible plateau at peak exercise.[42] Commonly used criteria for discontinuation of exercise testing include systolic blood pressure values above 250 mm Hg.[4] It should be emphasized, however, that clinicians have arbitrarily established this value, and no studies exist to support it as an end point. Moreover, there are virtually no reports of hypertensive-related cardiovascular complications that have resulted when subjects exceeded this level.[47]

Exertional hypotension (systolic blood pressure at the end of the test falling below the baseline standing level and/or systolic blood pressure decreasing 20 mm Hg or more during exercise after an initial rise)

has been shown to correlate with myocardial ischemia, left ventricular dysfunction, and an increased risk of cardiac events during follow-up.[48] In a cohort of 1,586 male cardiac patients, Irving and associates[49] showed a negative correlation between the maximal systolic blood pressure during exercise and the annual rate of sudden cardiac death (Table 7). Men with a maximal exercise systolic blood pressure < 140 mm Hg had a 15-fold increase in the annual rate of sudden death compared with those whose pressures exceeded 200 mm Hg.

Diastolic blood pressure

Normal hemodynamic responses to progressive exercise include no change or a decrease in diastolic pressure. Moreover, a diastolic blood pressure response >120 mm Hg is generally considered an end point for exercise testing.[4] An increase of more than 15 mm Hg in diastolic pressure during treadmill testing may suggest severe CAD, even in the absence of ischemic ST segment depression.[50] In contrast, diastolic hypertension may occur normally during cycle ergometer testing if tight gripping of the handlebars is permitted.

SYMPTOMS

It is important for the clinician to note all symptoms that occur during and after the exercise test, especially substernal pressure, which may radiate across the chest and/or down the left arm. However, anginal symptoms may also masquerade as back, jaw, abdominal, and lower neck pain or discomfort. Such symptoms can be rated by the patient on a scale of 1 to 4, corresponding to perceptible but mild, moderate, moderately severe, and severe, respectively.[4] Ratings of more than 2 (moderate) should generally be used as end points for exercise testing.

Table 7. Relation between maximal exercise systolic pressure and annual rate of sudden cardiac death

Maximal systolic pressure (mm Hg)	Annual rate of sudden death per 1,000
<140	97.0
140–199	25.3
>200	6.6

Source: Irving JB, Bruce RA, DeRouen TA. Variations in and significance of systolic pressure during maximal exercise (treadmill) testing: relation to severity of coronary artery disease and cardiac mortality. *Am J Cardiol.* 1977;39:841–848.

Although patients with exercise-induced ST segment depression are often asymptomatic, when concomitant angina occurs, the likelihood that the ECG changes are due to CAD is significantly increased. In addition, angina pectoris *without* ischemic ECG changes may be as predictive of CAD as ST changes alone.[51] Both are currently considered independent variables that identify patients at increased risk for subsequent coronary events.

SUPERVISION OF EXERCISE TESTING

The use of exercise testing has expanded greatly to help guide decisions regarding medical management and surgical therapy in a broad spectrum of patients. Contemporary cost containment issues and time constraints on physicians have encouraged the use of specially trained (eg, Advanced Cardiac Life Support certified) nurses, exercise physiologists, and physical therapists to directly supervise exercise stress tests, with a physician immediately available. Proponents of this practice argue that such specially trained health care professionals can accurately interpret ancillary signs

and symptoms, enabling termination of exercise tests at an appropriate intensity level.[52–54] This premise is further supported by data showing that the incidence of cardiovascular complications is no higher with experienced paramedical personnel than with physician supervision of exercise tests.[55,56] The American Association of Cardiovascular and Pulmonary Rehabilitation has recommended direct physician supervision for all initial exercise tests and tests for patients classified as high risk.[57] It was suggested that specially trained nonphysician personnel may directly supervise follow-up exercise tests for low- and intermediate-risk patients, with approval of the program's medical director.

• • •

Exercise testing is warranted whenever it is clinically important to obtain quantitation of cardiovascular function, from the earliest stages of illness (the first few days after uncomplicated myocardial infarction) to later in convalescence and recovery. Multistage exercise-tolerance testing provides invaluable information in assessing the safety of physical exertion and the effects of medical or surgical interventions. The results also have long-term prognostic significance with regard to morbidity and mortality. Although the exercise ECG has traditionally received the primary emphasis, evaluation of hemodynamics, symptoms, and aerobic fitness can enhance the predictive value of stress testing. The test methodology and modality, however, should be adapted to the patient, rather than the patient to the protocol.

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Appendix

Common terminology for interpreting diagnostic and functional exercise stress tests

Arteriovenous oxygen difference: The difference between the oxygen content of arterial blood and that of venous blood.

Coronary artery disease: A progressive atherosclerotic narrowing of the coronary arteries with a reduction in the blood and oxygen supply to the heart muscle.

Ejection fraction: A measure of contractility of the left ventricle, normally 65% ± 8%; lower values indicate ventricular dysfunction: the difference between left ventricular end-diastolic volume and left ventricular end-systolic volume divided by the former. A critical index of long-term prognosis in coronary artery disease.

Ergometer: An apparatus or device, such as a treadmill, a stationary bicycle, or steps, used for measuring the physiologic responses to exercise.

False-negative: A test or examination suggesting that a condition or disease is not present when in fact it is present.

False-positive: A test or examination suggesting that a condition or disease is present when in fact it is not.

Kilocalorie (kcal): The heat required to raise the temperature of 1 kg of water 1°C under specified conditions; a key index of metabolic rate.

Maximal oxygen uptake ($\dot{V}o_2max$): The maximal rate at which oxygen can be consumed per minute; the best physiologic index of total body endurance. This is also referred to as *aerobic power, maximal oxygen intake, maximal oxygen consumption,* and *cardiovascular endurance capacity.*

Metabolic equivalent unit (MET): A resting metabolic unit, the equivalent of 1.2 kcal/min or 3.5 to 4.0 mL/kg/min; 1 MET equals the amount of energy expended at rest.

Rate-pressure product: An index of myocardial oxygen consumption. There is a close correlation between myocardial oxygen consumption and the product of heart rate and systolic blood pressure. This is sometimes called double product.

Risk stratification: Classification of patients with coronary artery disease into three main risk categories—low, moderate, and high—based primarily on the extent of myocardial ischemia and left ventricular dysfunction.

REVERSING HEART DISEASE WITH DIET, DRUGS, AND EXERCISE– FACT OR FICTION?

Certificate Course in
Coronary Artery Disease
and Cardiac Rehabilitation 96
February 3-7, 1996
Barry A. Franklin, Ph.D.

Learner Objectives

1. To describe the natural history of atherosclerosis, with specific reference to evidence linking lipid abnormalities and other coronary risk factors to the disease process.

2. To review selected trials to date (drug and/or lifestyle changes) aimed at halting and reversing coronary artery disease.

Lecture Highlights

That heart disease might be reversible has been suggested for years. Atherosclerotic deposits shrink, for example, when laboratory animals are put on very low fat diets. During the two world wars, when many people subsisted on near-starvation rations, pathologists found during autopsies that people's arteries were remarkably free of cholesterol clogging deposits.

Perhaps the first convincing evidence that cholesterol deposits might actually regress came from a post-mortem study of Nathan Pritikin's heart (of Pritikin Diet fame). At the time of his mild heart attack in 1955, Pritikin's blood cholesterol was 280 mg/dl. Over the next 30 years, he followed an exercise program, running several miles weekly, and adopted a high carbohydrate, low fat, low cholesterol diet. One year before his death from cancer in 1985, his cholesterol had dropped to 94 mg/dl. At autopsy, his coronary arteries were found to be remarkably free of atherosclerotic deposits.

Several major studies in humans have now shown that lowering blood cholesterol levels can retard the progression of heart disease and, in some cases, even reduce the degree of coronary artery blockages. Selected investigations are briefly summarized below:

The Leiden Intervention Trial involved 39 patients with stable angina and documented coronary disease (≥ 50 percent obstruction) in at least one vessel. The intervention consisted of a two-year vegetarian diet, and less than 100 mg of cholesterol per day. Arteriography was performed before the intervention and after 24 months. Coronary lesion growth correlated with total/HDL cholesterol ($r = 0.50$, $p = 0.001$). Disease progression was significant in patients whose total/HDL cholesterol had a median value >6.9.

The Cholesterol-Lowering Atherosclerosis Study (CLAS) was a 2-year randomized, placebo-controlled angiographic trial of the effects of combined colestipol-niacin therapy on coronary atherosclerosis. The study subjects included 162 nonsmoking men aged 40-59 years with previous coronary artery bypass surgery. Total cholesterol and LDL decreased 26% and 43% respectively, whereas HDL increased 37%. Drug treatment reduced the progression of atherosclerosis. Regression occurred in 16.2% of colestipol-niacin treated versus 2.4% of placebo treated patients ($p = 0.002$).

Dr. Dean Ornish and colleagues reported even more impressive improvements without drugs after one year. Twenty-eight cardiac patients were assigned to a comprehensive lifestyle change program that included a low-fat vegetarian diet, stopping smoking, stress management training, and moderate exercise. These patients were

compared to a usual-care control group. Using computer-enhanced x-rays of the coronary blood vessels, Ornish found that the average degree of stenosis decreased from 61 percent to 56 percent. In the control group, blockages progressed from 62 percent to 64 percent. Overall, 82 percent of the experimental-group patients had an average change towards regression.

Whitney and co-workers reported on the reversibility of fixed atherosclerotic lesions with aggressive risk factor modification (7 case reports). Seven patients with documented coronary artery disease underwent a 2-year walking program and combination drug therapy. Mean weight loss was 24.7 pounds; average cholesterol decreased from 297 to 167 mg/dl, whereas HDL increased from 33 to 55 mg/dl. In 6 of the 7 patients, there was a mean increase in luminal area; 4 of these showed evidence of improved coronary blood flow.

The St. Thomas Atherosclerosis Regression Study (STARS) was a randomized, controlled, end-point-blinded trial based on coronary angiograms in patients with angina or previous myocardial infarction. Ninety men with coronary heart disease who had elevated plasma cholesterol levels were randomized to receive usual care, dietary intervention, or diet plus cholestyramine, with angiography at baseline and at 39 ± 3.5 months. Almost half (46%) of the controls, who received usual medical treatment, showed progressive narrowing and only one (4%) showed luminal widening. In contrast, subjects advised to follow a lipid-lowering diet, and those on diet and cholestyramine, showed a 66% fall in the incidence of progression, a 10-fold increase in the frequency of luminal widening, and fewer cardiac events; patients in the diet plus cholestyramine group

also showed widening of mean coronary diameter and of stenoses, compared with controls.

Schuler and associates studied the effects of regular physical exercise and a low-fat diet on the progression of coronary artery disease. Patients were recruited after routine coronary angiography for stable angina pectoris; they were randomized to an intervention group (n = 56) and a control group (n = 57). Treatment included intense physical exercise and a low-fat, low-cholesterol diet (AHA, phase 3). After 12 months of participating, repeat coronary angiography was performed. Patients in the intervention group demonstrated decreased stress-induced myocardial ischemia and improved coronary morphology, as compared to the control group.

Recently, Brown et al. reviewed the arteriographic outcomes in nine lipid-lowering trials. As a generalization of the composite of results, 8% of the control group patients were judged to have improvement in arterial obstruction ("regression") during the study period. By contrast, about one fourth of the treated patients were found to have improved (a threefold increase).

The Stanford Coronary Risk Intervention Program (SCRIP) evaluated whether 4 years of participation in a comprehensive cardiac intervention program of lifestyle modification and lipid-lowering medications could reduce angiographic progression of coronary disease compared with a control group that was randomly assigned to usual care. SCRIP demonstrated that multiple-risk-factor intervention can slow angiographic progression of coronary stenoses and reduce hospitalizations for cardiac events in patients with baseline LDL-cholesterol levels in the borderline-high range.

In summary, research indicates that partial regression of coronary artery disease can occur with intensive dietary, drug, and exercise therapy, or combinations thereof, but only in some people. These findings are encouraging, since even small decreases in coronary stenoses are associated with significant increases in perfusion.

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Outcome Measurement in Cardiac and Pulmonary Rehabilitation

by the AACVPR Outcomes Committee

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Reliable, valid, and responsive measures of outcome are essential in the evaluation of the effectiveness of cardiac and pulmonary rehabilitation. Objectively measured information of this type not only assesses the progress of the individual, but helps evaluate the effectiveness of the rehabilitation program and the discipline as a whole.

In the past, although administrators have rarely considered cardiac and pulmonary rehabilitation as a cost center, they have acknowledged the value-added service of these programs to their organizations. In the current health-care environment, even relatively inexpensive services such as cardiac and pulmonary rehabilitation will require documentation and accountability for quality and cost. Both payors and patients are demanding this information to make intelligent decisions about if and where to seek care and to spend their limited health-care dollars. Additionally, the incorporation of outcome measures into rehabilitation programs will help determine long-term, global benefits and the costs of intervention.

Recognizing the importance of outcome measurement, the American Association of Cardiovascular and Pulmonary Rehabilitation established an Outcomes Committee in 1992 with the following tasks: (1) to identify outcome areas for cardiac and pulmonary rehabilitation; (2) to establish an operational definition for each outcome; (3) to suggest methodology or tools to measure each outcome; and (4) to encourage outcomes research.

As a prelude to this paper, a survey was distributed at the 1993 American Association of Cardiovascular and Pulmonary Rehabilitation (AACVPR) annual meeting in Orlando, Florida. The following is a synopsis of the results reported in the Summer 1994 issue of *News and Views*. The questionnaire was designed to identify which, if any, outcome measures were being used. Three domains were of particular interest: functional capacity, quality of life (QOL), and health-related behaviors. A total of 210 surveys were returned representing 199 different programs. The programs were located in 39 states, plus the District of Columbia, Puerto Rico, Canada, and Mexico. This represents at least 20% of the rehabilitation programs listed in the AACVPR programs directory in 1993.

Fifty-two percent of the respondents reported assessing all three domains in their programs. The majority of programs evaluated functional capacity and health-related behaviors (85% and 84%, respectively) in some manner. As expected, the graded exercise test was the method used most frequently to evaluate func-

tional capacity. Health-related behaviors were assessed most often by measuring cholesterol levels, and less often by assessing smoking status or performing a dietary evaluation. Half of the programs responding reported that the determination of success of their education programs was assessed by testing patient knowledge or attitudes. A surprisingly high number of programs evaluated some form of QOL (62%), primarily by program developed questionnaires. Only 22% of those programs measuring QOL used published instruments, and no single tool was used with great frequency.

Although a majority of the programs staff responding to this survey engage in some form of outcome measurement, there is much inconsistency related to the areas evaluated and the types of measurements used, especially regarding QOL and health-related behaviors. Although the results of the survey are encouraging, they represent a limited sample of those engaged in rehabilitation programs, and may overstate the extent to which outcomes are being assessed. The purpose of this paper is to develop a conceptual model for outcomes, to discuss mechanisms for outcome measurement, and to make recommendations regarding the implementation of outcome assessment instruments.

Section I: Outcome Domains and Their Measurement

To achieve a comprehensive approach to measuring outcomes in cardiac and pulmonary rehabilitation, the committee identified three outcome domains based on Green's PRECEDE Framework used in health education.[1] These domains, which include health, clinical, and behavioral outcomes, influence one another, thus the overlap illustrated in Figure 1. Although a positive health outcome is the goal of cardiac and pulmonary rehabilitation, it is through clinical and behavioral interventions that we attempt to influence this primary outcome.

The health domain represents general or primary indicators of health outcomes that include morbidity, mortality, and QOL. Although a major goal of individual programs is to reduce morbidity and mortality, it is beyond the scope of most rehabilitation programs to measure their effects on these components of the health domain. This should be reserved for large outcome studies and would require multi-center, longitudinal designs. Quality of life, however, is a component that should be measured by all programs.

Measurement of QOL requires input from the patient to ensure an accurate assessment of perception of personal well-being and overall satisfaction with life. Its many indices can be divided among three dimensions: (1) physical function; (2) psychological well-being; and (3) social functioning.[2] Self- or interviewer-administered questionnaires can be used to measure differences in QOL between patients at one point in time, or in any given patient before and after an intervention.

Two basic approaches to QOL measurement are available: (1) general instruments that provide a summary of QOL; and (2) specific instruments that focus on problems associated with a single disease state, age group, or area of function.[3] One of the most widely used general instruments in the cardiac population is the Medical Outcomes Study Short Form (MOS SF-36). It contains 36 items that measure aspects of physical, psychological, and social functioning.[4] A more specific tool used to measure physical functioning in the cardiac population is the New York Heart Association Classification containing only four items measuring ability to perform physical activity.[5] One advantage of employing a general instrument is that different disease states can be compared. An advantage of using a disease specific measure is that selected changes unique to a specific patient population can be detected with greater sensitivity to change.[3] Examples of cardiac and pulmonary specific tests are described in Sections II and III of this paper.

Health outcomes are impacted by clinical outcomes. These outcomes include physiological, psychosocial, and medical utilization indices, some of which are listed in Table 1. Medical utilization indices are often measured in terms of cost. Many programs already measure one or more of these clinical outcomes.

To a large extent, clinical outcomes are influenced by patient behaviors. These are the behaviors generally

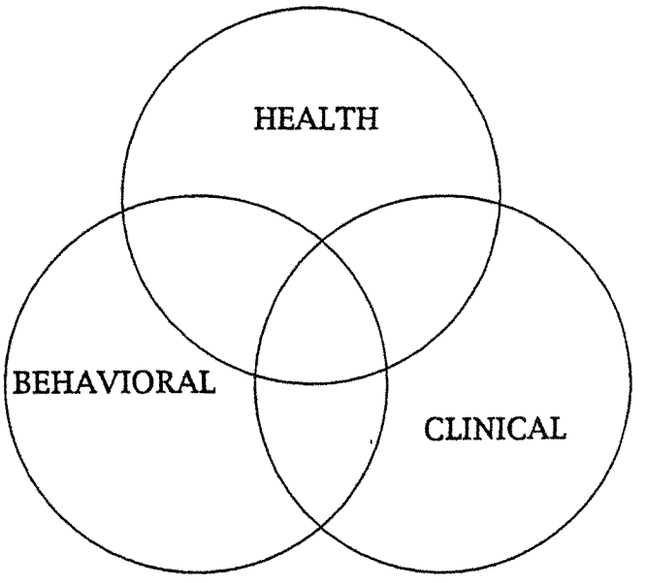

Figure 1. Outcome dimensions.

Table 1. *Some Measures of Clinical Outcomes*

Weight
Blood pressure
Oxygenation
Lipids
Exercise capacity
Functional status
Blood nicotine levels
Blood medication levels
Theophylline
Digoxin
Symptom management
Cough
Dyspnea
Angina
Interpersonal function and dysfunction
Psychological status
Return to vocational and avocational independent living
Medication usage
Hospitalizations
MD/ER visits

MD physician, ER. emergency room.

targeted by program interventions (Table 2). Adherence to these behaviors influences the clinical outcomes that ultimately affect the QOL and reduces the risk of subsequent morbidity and mortality. Traditionally, because of the influence of the medical model, rehabilitation programs have focused on clinical outcomes for ease of measurement. However, the life-long adoption of health-promoting behaviors may represent the most important contribution of cardiac and pulmonary rehabilitation.

Collection of outcome measurements should be integrated into routine clinical practice. Some data related to the outcome domains exist in medical record documentation or databases within rehabilitation programs. However, if these are used as outcome measures, there must be a systematic approach to organizing, collecting, and reporting this information. Program resources such as staff, finances, and time will influence the selection of specific outcomes to be measured.

For both clinical and research programs, accurate collection of data is important. The same test or questionnaire should be used before and following therapeutic intervention. Although it is ideal for the same clinician to administer the test each time, this is not crucial as long as standardization is strictly maintained. Special provisions should be made for individuals who have difficulty completing a questionnaire.

As a rule, outcome tools chosen should be:

1. *Clinically relevant.* Do the tools give meaningful results?
2. *Reproducible.* Do the tools produce the same results when administered repeatedly for stable characteristics? Do the tools produce the same results when administered by different staff members?
3. *Valid.* Do the tools measure the characteristics desired?
4. *Responsive.* Are the tools sensitive to change resulting from an intervention?
5. *Easy to administer and understandable.*

Staff can refer to a number of basic texts to learn how to collect and interpret data once a specific tool is selected.[6–8]

At a minimum, health, clinical, and behavioral outcomes should be measured at entry and discharge from rehabilitation programs. Because maintenance of gains resulting from rehabilitation is a measure of program success, follow-up is recommended. Six months to 1 year after completion of the program is a reasonable time period. Whenever possible, pre-morbid measures are desirable, (e.g., graded exercise test results or cholesterol measured before myocardial infarction [MI]).

Goals for the individual program as well as time and budget constraints will dictate the type and amount of tests employed. Simple self- or group-administered tests may be helpful when resources are limited. Although limitations in resources are to be expected, we recommend the measurement of at least one outcome from each of the three domains for each patient entering into a rehabilitation program. Information about selected valid and reliable tools is found in Table 3.

Table 2. *Some Measures of Behavioral Outcomes*

Compliance with:
Medical regimen
Diet
Exercise
Smoking cessation
Breathing retraining
Relaxation skills
Social skills
Recognition of impending complications

Table 3. *Outcome Measurement Tools*

Quality-of life measures (reference)	*Acronym*	*Items*	*Self*	*Time per Patient*	*Time per Staff*	*What is measured?*
Medical Outcomes Study Short Form (4)	SF-36	36	Yes	5–10	5	Physical, psychological and social functioning
Nottingham Health Profile (21)	NHP	45	Yes	10	10	Energy, pain, emotion, sleep, mobility, social isolation, and ADL
Sickness Impact Profile (19)	SIP	136	Yes	30	20	Physical, psychosocial, and five independent factors
Quality of Well-Being Scale (22, 63)	QWB	18–62	No	10–15	20	Mobility, physical activity, social activity, self care, and symptoms
Illness Effects Questionnaire (20)	IEQ	20	Yes	20	25	Biologic, psychological, and social aspects
Chronic Respiratory Disease Questionnaire (54)	CRQ	20	No	25–30	35	Dyspnea, fatigue, emotional function, and feeling of mastery over disease
St. George's Respiratory Questionnaire (60)	SGRQ	76	Yes	15	10	Symptoms, activity, and impact
Pulmonary Functional Status Scale (58)	PFSS	56	Yes	15–30	10 to 25	ADL in respiratory patients
Pulmonary Functional Status and Dyspnea Questionnaire (59)	PFSDQ	164	Yes	15	15	ADL and dyspnea
Minnesota Living with Heart Failure Questionnaire (26)	LHFQ	21	Yes	10–15	3 to 5	Physical, socio-economic, and psychological impairment
Functional activity and exercise–related measures (reference)						
The New York Heart Association functional classification (5)	NYHA	4	No	NA	1	Physical activity performance
The Specific Activity Scale (24)	SAS	5	No	5	1	Activities of daily living
Baseline Dyspnea Index/ Transitional Dyspnea Index (77)	BDI/TDI	15	No	5–10	5–10	Functional impairment, magnitude of task, and magnitude of effort
Duke Activity Survey Index (78)	DASI	12	Yes	5	2	Functional status
Borg Scale (68)		NA	No	Seconds	Seconds	Perceived exertion
Visual Analogue Scale (69)		NA	No	Seconds	1	Severity of dyspnea during exercise
Progressive multistage exercise test maximal and submaximal	GXT	NA	No	45–60	45–60	Changes in exercise capacity
Progressive Multistage Exercise Test With Metabolic Analysis		NA	No	45–60	45–60	Changes in exercise capacity by direct measure
Six or 12 minute walk (79)		NA	No	20–30	20–30	Exercise endurance
Human Activity Profile (Formerly ADAPT) (80)	HAP	105	Yes	15–30	20–50	Activity level

(continued)

Table 3. *Outcome Measurement Tools (Continued)*

Psychological measures (reference)	*Acronym*	*Item*	*Self*	*Time per*		*What is measured?*
				Patient	*Staff*	
Hopkins Symptom Checklist-revised (34)	SCL-90-R	90	Yes	20	30*	9 subscales, i.e., somatization, depression, anxiety, and hostility
Minnesota Multiphasic Personality Inventory-2 (35)	MMPI	567	Yes	180	60*	Depression, anxiety, psychosis, and personality disorders
Cook-Medley Hostility Inventory (37)		50	Yes	15	15*	Hostility
Beck Depression Inventory (39)	BDI	21	Yes	5–10	15*	Depression and mood state
Center for Epidemiological Studies—Depression Inventory (40)	CES-D	20	Yes	10	15	Depression and mood state
State-Trait Anxiety Inventory (41)	STAI	40	Yes	20	5*	Anxiety
COPD Self Efficacy Scale (81)		34	Yes	5–10	30*	Confidence level
COPE Inventory (82)	COPE	52	Yes	20	20*	Different ways in which people respond to stress
Jenkins Activity Survey (83)	JAS	20	Yes	20	15*	Four common components of Type A Behavior
Profile of Mood States (84)	POMS	65	Yes	15	20*	Various mood states (e.g., tension, anger, depression, confusion)
Sociological measures (reference)						
Marital Adjustment Scale (45)	MAS	16	Yes	5	5	Marital adjustment
MOS Social Support Survey (85)		20	Yes	10	5	Four functional support scales
Other health-related behavior measures (reference)						
Harvard-Willett Food Frequency Questionnaire (47)		116	Yes	45–60	†	Type, quantity and frequency of food ingested
Block Food Questionnaire (49)		128	No	30–45	45	Calories, fat, fiber, protein, carbohydrates, linoleic/oleic acids, vitamins, and minerals
Diet Habit Survey (86)		32	Yes	30	20–30	Cholesterol-saturated fat intake; complex carbohydrates, and salt
Quick Check (87)		74	Yes	15	3–5	Fat, saturated fat, and cholesterol
Health Knowledge Test (88)		40	Yes	15–20	5–10	Knowledge of disease management skills in pulmonary patients

Self: yes = self administered; no = structured interview; Time per patient: time in minutes for patient to complete; Time per staff: time in minutes for staff to administer, score, and interpret; ADL: Activities of Daily Living; NA: not applicable.
*Psychologist must interpret.
†Must be mailed out for computer scoring.

Section II: Outcomes in Cardiac Rehabilitation

Benefits of comprehensive cardiac rehabilitation (CR) demonstrated in randomized, controlled clinical trials include an improved work capacity,[9-11] improved coronary risk factor profiles,[12,13] and increased return to work rates.[14,15] Pooled data from randomized trials in post-MI patients have documented a 20% decrease in cardiac and overall mortality over a 2- to 3-year follow-up period.[16,17] The effects of CR as defined by the individual patient has received less study. These include both primary health outcomes, such as QOL, and secondary clinical and behavioral outcomes, such as lipid levels and smoking status, which mediate changes in the health-related outcomes. This section will focus primarily on the QOL, clinical, and behavioral outcomes that are important to measure in individuals and in programs of cardiac rehabilitation. It is designed to provide a general overview of the many elements that can be measured with suggestions for limited measurement in each domain.

Health-related Outcome

Quality of Life. Quality of life outcome measures before and after CR should include the assessment of physical function, psychological well-being, and social functioning. The Medical Outcome Study (MOS) questionnaire[18] is a general health profile that includes a physical dimension and a psychosocial dimension, and exists in a time-saving short form (SF-36).[4] Other well-known general health QOL instruments include the Sickness Impact Profile (SIP),[19] the Illness Effects Questionnaire,[20] the Nottingham Health Profile,[21] and the Quality of Well-Being Scale.[22]

The SF-36 is a 36-item scale providing 8 separate multi-item subscales assessing physical functioning, physical role functioning, emotional role functioning, social functioning, bodily pain, mental health, vitality, and general health perceptions, which takes approximately 16 minutes to complete.[4] The SIP is a much lengthier measure, requiring as much as 45 minutes to complete, but providing more detailed information. It addresses physical functioning (ambulation, mobility, body care, and movement), four psychosocial dimensions (social interaction, alertness, emotional behavior, communication), and five independent categories (sleep and rest, eating, work, home management, recreation).[23] The SIP can be either self-administered or interviewer-administered.

Specific measures for cardiovascular disease are designed to address selected changes unique to cardiac patients. The New York Heart Association functional classification was an early, disease-specific scale used to assess limitations of ability to perform physical activity in a variety of cardiovascular diseases.[5] The Specific Activity Scale may be more precise and correlates well with measures of maximal oxygen uptake at exercise testing.[24] The Medical Outcome Study has a disease specific instrument for angina and specific instruments have been developed for patients with congestive heart failure,[25,26] and MI.[27]

Clinical Outcomes

Some measures of clinical outcome in CR are functional capacity, smoking, lipids, and blood pressure.

Functional capacity. Traditionally, physical function has been assessed by the performance of a continuous, graded exercise test performed before and after an exercise conditioning program. It is the standard tool by which functional capacity is assessed and provides an accurate assessment of exercise-induced symptoms such as angina or exertional dyspnea. Additionally, physical performance questionnaires or testing batteries that relate to the patient's work and/or home activities can be employed. A number of physical performance batteries have been developed, some of which focus on specific patient groups such as the elderly and chronic heart failure patients.[28-30] However, these have not been specifically developed for the coronary patient.

Smoking. Smoking cessation and/or relapse prevention is an important goal of CR. Smoking outcomes are measured through self-report or through the use of physiologic and/or biochemical measures. Physiologic measures include serum thiocyanate, expired carbon monoxide, or salivary, urine, or plasma cotinine levels. Carboxyhemoglobin can also be measured with or without a blood sample. Thiocyanate, a metabolite of hydrogen cyanide, remains in the plasma for up to 14 days.[31] Cotinine, a metabolite of nicotine, has a long half-life of 16 to 20 hours, and remains constant in individuals who habitually smoke. The latter may be the best biochemical indicator of smoking status because of its longer half-life. Although self-report measures through interviews, questionnaires, or daily logs are often used to confirm smoking status, there is concern about patient misreporting, which has been reported as high as 20% in some studies. At a minimum, self-report should be obtained at the end of program participation on all patients classified as ever-smokers. Spousal or family confirmation of smoking status is helpful where biochemical confirmation is not available.

Lipids. A fasting complete lipid profile is recommended before and after the CR intervention to include total

cholesterol, triglycerides, high density lipoprotein (HDL) cholesterol, and low density lipoprotein (LDL) cholesterol. Because of laboratory variability and intraperson biologic variability, which combined range from 6% to 10%, accuracy is significantly improved with the averaging of two values at any given point in time. Additionally, if lipid profiles are drawn within the first few weeks after a cardiac event such as MI or coronary artery bypass graft (CABG), may not accurately reflect the patient's lipid profile because the event has an impact on the substances measured.[32]

Blood pressure. Systolic and diastolic blood pressure measured in the seated position after a 5-minute rest is the standard by which the effect of therapies is generally measured. More sophisticated measures such as the ambulatory monitoring of blood pressure responses over a 24-hour period may add a higher degree of accuracy and provide insight into the effects of numerous variables such as time of day, effect of physical activity, and mental stressors.

Psychological. Assessing and monitoring changes in psychological functioning is critical to facilitating the long-term well-being of the patient in CR. Depression, low morale, and distress are significant predictors of mortality among patients after MI.[33] The assessment of psychological outcomes can be complex because of the multiplicity of measures. Global measures of psychological functioning are available, as well as specific indicators of distress, such as measures of depression and anxiety. The choice of outcome measures may be dictated by the interests of the particular rehabilitation site. However, a global measure of psychological well-being will be helpful in providing at least a summary indicator of the potential areas of concern for patients, as well as providing an outcome measure at the conclusion of rehabilitation.

A commonly used measure of psychological functioning is the Hopkins Symptom Checklist-revised (SCL-90-R).[34] The SCL-90-R is a 90-item scale providing 9 clinical subscales (e.g., somatization, depression, anxiety, hostility) and a summary score. A more detailed summary of psychological functioning can be assessed with the Minnesota Multiphasic Personality Inventory-2 (MMPI-2),[35] a 567-item scale measuring depression, anxiety, psychosis, and personality disorders.

Because recent research suggests the importance of the hostility component of the Type A behavior pattern,[36] a useful psychological measure in CR is the Cook-Medley Hostility Inventory,[37] a 50-item True-False scale derived from the MMPI-2. Elevated scores have been associated with a greater incidence of coronary disease.[38]

Psychological functioning of patients in CR can also be evaluated with well-validated measures of specific mood states. These include the Beck Depression Inventory,[39] the Center for Epidemiological Studies—Depression Inventory,[40] and the State-Trait Anxiety Inventory.[41] In addition, there are numerous other aspects of psychological functioning that have been evaluated with cardiac patients, including loneliness, self-concept, locus of control, and cognitive functioning.[42] However, for most CR programs, a general measure such as the SCL-90-R may provide a good overview of psychological functioning. Measures of psychosocial functioning, as described below, may provide additional relevant outcome data.

Social. Social changes among patients in CR are common, with patients frequently experiencing changes in social relationships, sexual functioning, and vocational status.[36] Usually the cardiac patient must re-evaluate roles and responsibilities in the home, workplace, and interpersonal relationships.

Marriage, in particular, is an important influence on psychosocial functioning in patients in CR. Past research suggests that married post-MI patients have a better chance of survival than nonmarried patients.[43] However, the roles of the patient and spouse in the marriage often may need to be altered, at least temporarily. In addition, sexual activity is often reduced among post-MI patients.[44] This may be the result of medication side effects, fear of precipitating further cardiac problems, and depression subsequent to the cardiac event. Factors associated with psychological well-being, in combination with social, sexual, and general activity changes, contribute to the overall psychosocial functioning of the patient in CR. The Marriage Adjustment Scale (MAS) is a good tool for assessing marital factors.[45]

Behavioral Outcomes

The outcome of a number of behavioral-based interventions is important to assess before and after CR because of their impact on primary health outcomes and clinical outcomes. Dietary interventions, for example, play an important role in affecting positive clinical outcomes with regard to serum lipids, weight loss, and control of hypertension. Alternatively, an extremely restrictive diet may be viewed by the individual patient as having a negative effect on overall QOL.

Some of the behavioral interventions that may be used to improve outcomes in CR are listed in Table 2. Unlike health and clinical outcome domains, adherence to these interventions is most often assessed by self-report. Many of the techniques described to chart dietary adherence may be used to monitor and assess patient success in other areas of needed behavioral change.

Diet. Evaluating dietary content in macronutrients requires reliance on self-reported data that are subject to errors in perception, recall, and underreporting. The four most commonly used tools for collecting individual dietary intake information are diet histories, food records, 24-hour recall, and food frequency questionnaires. Food records are completed prospectively, whereas histories, recalls, and frequencies are completed retrospectively. The diet history involves an extensive interview by a trained nutritionist to determine the long-term dietary patterns of an individual. Food frequency questionnaires list a number of food items and patients indicate how often they eat each one. Several food questionnaires have been developed and tested in relation to measuring dietary fat, saturated fat, and cholesterol. These include the Quantitative Food Frequency Analysis,[46] The Willett Food Frequency Questionnaire,[47,48] and the Block Questionnaire,[49] which is a 13-item tool used as a screening device to detect high or low fat intake.

Exercise. Adherence to an exercise program may have a powerful effect on the clinical outcome of change in functional capacity. Although independent observation of all exercise behavior is the only direct measure of exercise adherence, this is impractical and rarely possible. Two indirect measures that provide a reasonable substitute include self-report of the exercise behavior and attendance at a program. Self-monitoring can be accomplished by asking the patients to keep track of their exercise using diaries or activity logs that can be returned to staff for review. Patients are usually asked to document the frequency, duration, and intensity of exercise. Studies have demonstrated the reliability of two self-report measures of exercise activity in the general population that may also be useful in patients with cardiac disease.[50,51]

Although attendance at a program is important, unless the patient follows the parameters of the exercise prescription, attendance is not a valid measure of adherence. Moreover, decreased attendance at a program does not always mean a patient has decreased exercise. Staff must consider the issue of outside activities and define a level of adherence for the patient.

Medication use. Adherence to medication regimens may affect clinical outcomes such as a change in lipid levels or a reduction in systolic or diastolic blood pressure. Although the use of pill counts, mechanical monitors, or clinical estimates are ways to monitor adherence to medications, they are generally not realistic in a rehabilitation setting. Patient's self-report of adherence by interview has also been shown to be effective and has shown a correlation of 0.75 with pill counts.[52] Simple questions about daily dose administration, timing, side effects, and use of patient reminders may enhance self-monitoring and facilitate adherence to medications.

Section III: Outcomes in Pulmonary Rehabilitation

The benefits of pulmonary rehabilitation (PR) are clearly multifactorial.[53–57] This is understandable given the devastating effect of advanced pulmonary disease on virtually all aspects of the individual's life and the comprehensive nature of most pulmonary rehabilitation programs. Because of the diverse gains from pulmonary rehabilitation, no single test is sufficient to determine its full effectiveness. It is recommended that PR programs consider evaluating the following areas of potential improvement within the health, clinical and behavioral outcome domains: (1) quality of life; (2) dyspnea; and (3) exercise ability.

Health-related Outcome

Quality of Life. Quality of life questionnaires specific for pulmonary disease have the advantage of greater applicability and responsiveness to therapeutic intervention. They are designed to assess symptoms, physical function, psychological well-being, and social function in areas affected by chronic obstructive pulmonary disease (COPD). Four recommended questionnaires of this type include: (1) the Chronic Respiratory Disease Questionnaire (CRQ),[54] (2) the Pulmonary Functional Status Scale (PFSS),[58] (3) the Pulmonary Functional Status and Dyspnea Questionnaire (PFSDQ),[59] and (4) Saint George's Respiratory Questionnaire (SGRQ).[60]

The CRQ is a widely used instrument that measures QOL in four dimensions: dyspnea, fatigue, emotional function, and the feeling of mastery over the disease. This 20-item questionnaire, which can be completed in approximately 20 to 30 minutes, requires training to administer. Responsiveness to pharmacologic intervention[54] and pulmonary rehabilitation[54,61] have been demonstrated.

The PFSS and the PFSDQ are two newly developed, self-administered instruments that measure functional status of individuals with lung disease. Both focus on activities of daily living and areas affected by chronic lung disease. Categories such as self-care, mobility, home management, recreation, social activity, and relationships are covered. Each questionnaire takes approximately 15 minutes to complete.

The SGRQ is a 76-item questionnaire that can be self-administered, but may require help from a staff member. It measures health status of individuals with

chronic lung disease in three areas: symptoms, activity limitation, and impact on well being. Scores from this questionnaire correlate with other indices of severity, including spirometry, respiratory symptoms, anxiety, depression, walking distance, and general health status.

General instruments can be used to assess QOL in the pulmonary patient, but are probably less responsive to therapeutic intervention. Because an instrument of this type is generic in nature, it can be used in different diseases. As such, it may be useful as a single tool in cardiopulmonary rehabilitation programs. Examples include the Sickness Impact Profile (SIP),[62] the Quality of Well-Being Scale (QWB),[63] and the Medical Outcomes Study short form survey (SF-36).[64]

Clinical Outcomes

Dyspnea. Dyspnea, the unpleasant sensation of breathlessness, is usually the overriding symptom of advanced pulmonary disease.[65] Its reduction is an important therapeutic goal for both standard medical therapy and PR. Although this symptom is totally subjective, it can nonetheless be measured. Dyspnea may be measured in two ways: overall dyspnea; and dyspnea for a specific task, such as stair climbing or walking.

The recommended measure of overall dyspnea is the Baseline Dyspnea Index (BDI)/Transitional Dyspnea Index (TDI), developed by Mahler and colleagues.[64] These can be readily completed by health professionals with minimal training. The BDI measures three components of dyspnea: functional impairment, magnitude of task, and magnitude of effort, whereas the TDI measures changes in these components. Results from the BDI correlate with other dyspnea scores[66] and health status. Improvement in dyspnea scores from the TDI has been associated with theophylline therapy, inspiratory muscle training, and pulmonary rehabilitation.[67]

The dyspnea dimension of the CRQ is another good measure of overall dyspnea. For this measure, the patient chooses five important activities that produce breathlessness in his/her daily activities. The degree of breathlessness for each activity is then graded by the patient using a 7-point scale. The dyspnea score from this instrument is highly patient-specific.

Dyspnea for a specified task can be quantitated using a category scale such as the Borg Scale,[68] or a visual analog scale.[69] For the latter, the individual indicates his or her level of breathlessness by pointing to a point on a line that is usually 100 mm long. The line is usually anchored at both ends with one end labeled, "no breathlessness" and the other labeled, "severe breathlessness." Using a category or visual analog scale to quantify exertional dyspnea as an outcome measure in PR obviously requires they be obtained at comparable external workloads before and after rehabilitation.

Exercise ability. Improvement in exercise ability resulting from PR usually reflects increases in both endurance and maximal exercise capacity. Exercise endurance usually improves to a greater degree than maximal capacity.[56] Although exercise conditioning is important in producing these gains, other factors, such as increased motivation, desensitization to dyspnea, and improved pacing may also be significant.[70]

The minimum standard test of exercise capacity recommended is the 6- or 12-minute walk to determine changes in exercise ability with PR. For this, the distance walked in an enclosed area, such as a hallway or the perimeter of an auditorium, is recorded. Because the 6-minute walk test correlates well with the 12-minute test[71] and requires less time, it is preferred.

In addition to its ease of administration and relevance to ordinary daily exercise, performance on the walk test correlates with degree of airway obstruction, dyspnea, and QOL,[54,72] and is very responsive to pulmonary rehabilitation.[53,73] However, this test is significantly affected by variables such as encouragement and test repetition.[74] For this reason, standardization is essential, especially with regard to encouragement and the use of practice trials before taking the actual measurement.

The graded exercise test, performed either to a submaximal or maximal workload and using either a cycle ergometer or treadmill, is the preferred method to measure changes in exercise capacity. Although costly, it is responsive to change with PR,[75] and can be used to provide objective physiologic data, such as minute ventilation, oxygen consumption, and oxygen desaturation. In addition, unexpected cardiac ischemia, arrhythmias, or hypertensive responses to exercise may be detected. Finally, results from graded exercise testing may prove useful in determining the exercise prescription for the rehabilitation patient.

The ability to climb stairs is also a useful test of exercise capacity in pulmonary patients. The maximum number of steps or flights achieved during this simple and inexpensive testing correlates well with maximal exercise capacity as determined by standard cycle ergometry.[76] Whether this test is responsive to therapeutic intervention remains to be determined.

For all of the above tests of exercise capacity, dyspnea can be measured using a Borg-type categorical scale[68] or visual analog scale.

Behavioral Outcomes

There is a great deal of overlap in the behavioral outcomes addressed in PR and CR. For PR, the most important behavioral outcome is smoking cessation.

This is so important to the process of PR that most programs require abstinence from cigarettes as an entrance criterion. Because undernutrition and weight loss are associated with increased mortality in chronic obstructive pulmonary disease (COPD) and obesity increases the work of breathing, behavioral intervention in the area of nutrition is important in PR. Dietary supplements for underweight or undernourished patients and calorie restriction diets for obese patients should be offered, and nutritional status should be considered as an outcome measure in PR.

The educational components of comprehensive PR are in large part aimed at improving disease management skills. This includes improved compliance with medications, properly metered dose-inhaler techniques, and breathing retraining. Increased communication between the patient and caregivers encourages the patient to assume greater responsibility for his or her own care. Although progress in these areas is difficult to quantify, PR staff should consider their evaluation and document changes.

Cost Effectiveness Outcomes

Cost effectiveness is a concept of growing importance in cardiac and pulmonary rehabilitation; however, the collection of sophisticated cost-analysis data is beyond the resources of most CR and PR programs. Patients and payors want information for selecting the best outcome for the least price. Cost effectiveness needs to be viewed as a "value index" specific to the measure attained as in Value = outcome/cost. For example: Value = change in METs/cost of providing the service.

Assessment of outcomes has been discussed in this paper. Cost is defined as the true cost of the rehabilitation intervention and therefore, includes not only staff salaries, but also other program expenses such as equipment, facility overhead, and supplies. Where possible, data collection should go beyond the costs for the CR and PR intervention and consider dropout rates, rehospitalization rates, return-to-work rates and a QOL or functional status measure for determination of cost per effect, or cost effectiveness.

At a given institution, charges can be converted to true costs through the use of the institutional cost/charge ratio. Your administrator, business office staff, or a financial consultant should be enlisted to assist you.

Conclusion

Outcome measurements need to be more consistently used in clinical CR and PR programs to better justify the resources used for cardiac and pulmonary rehabilitation. Benefits of patient's functioning need to be better documented to describe effects of CR and PR from the *patient's* point of view. Although limitations in personnel and resources of the individual program may limit the sophistication and depth of these measures, a comprehensive rehabilitation program should measure at least one QOL, one clinical, and one behavioral measure of outcome. Recommendations for cardiac and pulmonary programs are shown in Table 4.

It is the consensus of the authors of this position paper that the AACVPR should encourage outcomes research in cardiac and pulmonary rehabilitation with controlled, multicenter clinical trials both at academic and clinical institutions. However, before conducting clinical research trials, programs must recognize the importance of collecting outcome data to determine individual patient progress and program effectiveness.

Table 4. ***Suggestions for Minimal Outcomes Measurement***

Outcome	*Cardiac*	*Pulmonary*
QOL	SF-36, SIP, or IEQ	CRQ, PFSS, PFSDQ, or SGRQ
Clinical	GXT	GXT or 6-minute walk test
Behavioral	Patient report based on risk	BDI/TDI (overall dyspnea) Category or visual analog scale (exertional dyspnea)

QOL: quality of life; SF-36: Medical Outcomes Study Short Form; SIP: Sickness Impact Profile; IEQ: Illness Effects Questionnaire; CRQ: Chronic Respiratory Disease Questionnaire; PFSS: Pulmonary Functional Status Scale; PFSDQ: Pulmonary Functional Status and Dyspnea Questionnaire; SGRQ: St. George's Respiratory Questionnaire; GXT: Maximal and Submaximal Progressive Multistage exercise test; BDI/TDI: Baseline Dyspnea Index/Transitional Dyspnea Index.

The Outcomes Committee suggests the use of scientifically valid and reliable instruments to help begin the process of outcomes measurement. The instruments included in this paper are not all inclusive. A resource guide compiled by the Outcomes Committee is available from the AACVPR with a more extensive list of tools, their description, and their source. With input from the members, the Outcomes Committee will continue to research and evaluate outcome measurement in the future.

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A Consensus Approach to Predicting Angiographic Coronary Artery Disease: Applying Logistic Regression Equations Based on Clinical and Exercise Test Data

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Abstract

- ***Objective:*** To demonstrate that a consensus approach to applying probabilities generated from logistic regression equations based on clinical and exercise test variables is accurate, self-calibrating and thus portable to any clinical setting for cost-effectively diagnosing coronary artery disease.
- ***Design:*** Retrospective analysis of consecutive patients with complete data from exercise testing and coronary angiography referred for evaluation of possible coronary artery disease. One of the three logistic equation equations was developed in a training set then, along with two other equations developed by other investigators, validated in a test set.
- ***Settings:*** Two University affiliated Veteran's Affairs Medical Centers.

Presence of Disease Study

- ***Patients:*** 718 consecutive males studied between 1985 and 1995 who had coronary angiography within 3 months of the treadmill test. The population was randomly divided into a training set of 429 patients and a test set of 289 patients. Patients with previous myocardial infarction or coronary artery bypass surgery, valvular heart disease, left bundle branch block or any Q-waves present on their resting electrocardiogram were excluded from the study.
- ***Measurements:*** Recording of clinical and exercise test data along with visual interpretation of the ECG recordings on standardized forms and abstraction of visually interpreted angiographic data from clinical catheterization reports.
- ***Results:*** We demonstrated that by using simple clinical and exercise test variables, we could improve on the standard use of ECG criteria for diagnosing coronary artery disease. The sensitivity and specificity in the test set using standard ST criteria were 49% and 83%, respectively. Using the consensus approach divided the test set into thirds with low, intermediate and high risk for coronary artery disease. Since the patients in the intermediate group would be sent for further testing and would eventually be correctly classified, the sensitivity of the consensus approach is 87% and the specificity is 92%. The consensus approach appears to be unaffected by disease prevalence, missing data, how variables are defined and even by angiographic criterion.
- ***Conclusions:*** Requiring consensus has made these logistic regression equations portable to other populations. The consensus approach is best applied utilizing a

computer program to simplify the process of calculating the probability of coronary artery disease using the three equations. It effectively limits the usage of more expensive imaging modalities to one third of patients and excludes the need for cardiac catheterization in another one third of the patients. Excellent diagnostic characteristics are obtained using simple data and measurements permitting a cost-effective evaluation of patients with possible coronary artery disease.

Severity of Disease Study

- ***Patients:*** 1080 consecutive males studied between 1985 and 1995 who had coronary angiography within 3 months of the treadmill test. The population was randomly divided into a training set of 701 patients and a test set of 379 patients. Patients with previous coronary artery bypass surgery, valvular heart disease, left bundle branch block were excluded from the study.
- ***Measurements:*** Recording of clinical and exercise test data along with visual interpretation of the ECG recordings on standardized forms and abstraction of visually interpreted angiographic data from clinical catheterization reports.
- ***Results:*** We demonstrated that by using simple clinical and exercise test variables, we could improve on the standard application of ECG criteria for predicting severe coronary artery disease. The sensitivity and specificity in the test set for 1 mm ST depression criteria were 57% and 71%, and for 2 mm ST depression criteria were 36% and 89%, respectively. The consensus approach divided the test set into thirds with low, intermediate and high risk for coronary artery disease. Since the patients in the intermediate group would be sent for further testing and would eventually be correctly classified, the sensitivity of the consensus approach is 81% and the specificity is 90%. The consensus approach appears to be unaffected by disease prevalence, missing data, how variables are defined and even by angiographic criterion.
- ***Conclusions:*** Requiring consensus has made these logistic regression equations based on clinical and standard exercise test data portable to other populations. The consensus approach effectively limits the usage of more expensive imaging modalities to one third of patients and excludes the need for cardiac catheterization in another one third of the patients. Excellent predictive characteristics are obtained using simple data and measurements permitting a cost-effective evaluation of patients with possible severe coronary artery disease.

Believability Criteria for Evaluating Diagnostic Tests

Vic Froelicher, MD

Probability of Coronary Disease for Middle-Aged Males or Post Menopausal Females Pre/Post Any Non-Invasive Test

Chest Pain Character	Pre-Test	Post Abnormal Test	Post Normal Test
Typical Angina	90%	98%	75%
Atypical Angina	50%	90%	25%
Non-Angina	10%	45%	4%
None	2%	6%	<1%

Insert ~~4-5~~

Comparison of Diagnostic Tests

	Test (MI excld, WU bias)	(N)	Sens	Spec
Exercise Stress	ECG	147	68%	77%
	Thallium (planar/spect)	59	79%	87/74
	RNV	10	92%	65%
	ECHO	10	86%	86%
Non-Exercise Stress	Dobutamine Echo	5	88%	84%
	Persantine Thallium	11	85%	91%

Guyatt Rules for Believing an Evaluation of a Diagnostic Test

1. **Identification of Comparison Groups with One Group Free of Disease**
2. **Consecutive or Randomly Selected Patients for Whom the Diagnosis is in Doubt**
3. **Separate Analysis of Patients Likely to Have the Disease (ie, Post-MI patients)**
4. **Blind Comparison of the Test with a Reliable Standard**

..... If Believability Criteria met then the Test can be put into Practice

Work-up Bias

- In All Studies that Require the Patients to have a Cardiac Catheterization, the Patients are already selected by their physicians for the Cardiac Catheterization Using Clinical and Test Variables
- For the Selection of Variables from a Study to be Applicable to the Patient Presenting for a Work-Up, the Study must be Performed on an Un-selected Population
- Work-Up Bias can Only be Avoided by having the Patient Consent to Catheterization no Matter what the Test Results Are

Scores to Predict Severity and Prognosis in Coronary Artery Disease

Vic Froelicher, MD

Stable CAD

Who to Cath ?

- Quality of Life
  - Limitations to activities
  - Medication side effects
- Quantity of Life
  - Clinical Scores to Estimate CV annual mortality and probability of severe CAD
  - Angiographic Subsets

Prognosis

- Congestive Heart Failure
  - 10 to 25% Annual Cardiac Mortality
- Myocardial Infarction
  - Complicated: Shock, CHF, Ischemia...
  - 10% prior/10% in-hospital/10%1st year
- Angina Pectoris
  - Stable: 2% Annual Cardiac Mortality
  - Unstable: 4%

Meta Analysis of Prognosis Post MI

Exercise Test and Follow-Up (N=28)

CHF (Excluded from Test)	**2x**
Resting ST Depression	**3X**
Poor Exercise Capacity	**14/18***
Exercise ST Depression	**15/24**
(Non-Qwave only Studies)	**2/2***
Exercise SBP	**13/18***
Angina	**12/20**
PVCs (frequent)	**14/23**

Prognosis

Meta Analysis of Prognosis in Stable CAD

Exercise Test and Cath (N=9)

Poor Exercise Capacity	6/9
CHF	3/9
ST Depression	
Resting	2/9
Exercise	3/9
Exercise SBP	3/9

Work-up Bias

- In All Studies that Require the Patients to have a Cardiac Catheterization, the Patients are already selected by their physicians for the Cardiac Catheterization Using Clinical and Test Variables
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Clinical Scores

- Survival Analysis
  - Based on Follow-up and Censoring
  - Cox Hazard Function
  - Weighted Coefficents used to construct Equations for Scores and Nomogram
- Probability of Severe Disease
  - Based on Angiography
  - Multiple Logistic Regression
  - Coded Variables x Coefficents added then solved in Natural Log Equation to fit a Sigmoid Curve

Prognosis

Prognostic Scores

- DUKE SCORE
  METs - 5x[mm E-I ST Depression] - 4x[TM Angina Index]
  ***see nomogram
- VA SCORE
  5x[CHF/Dig] + [mm E-I ST Depression] + change in SBP score - METs
  E-I = Exercise Induced

Multiple Logistic Regression Equations to Predict Severe CAD

Christian, et al, Ann Intern Med 121:825-832, 1994: age, gender, symptoms, diabetes, peak double product and amount of ST depression.

Detrano, et al, Comp & Biomed Res 25:468-485, 1992: age, gender, symptoms, history of MI or Q waves, METs, peak HR, exercise induced angina, exercise induced hypotension, ST slope and amount of ST segement depression.

Morise, et al, JACC 20:1187-96, 1992: age, gender, symptoms, diabetes, hypertension, cholesterol, obesity, current cigarette use, estrogen, change in systolic blood pressure, ST slope, amount of ST segment depression and negative ST (< 1.5mm upsloping or < 1mm horizontal or downsloping).

Froelicher/Do (1995): age, cholesterol, LVH with strain, change in systolic blood pressure, peak heart rate, ST slope and amount of ST segment depression.

Problems with Prediction Equations

- Misclassification
- Follow-up Confounded by Interventions
- Work-Up Bias
- Skepticism that Simple Variables Can Be Better Than Imaging Technologies
- Differences between Studies as to Variables and Their Coding
- Requires Nomograms or Computers to Calculate

CONCLUSIONS

*ST segments exhibited abnormal depression during exercise and abnormal depression in recovery. (Abnormal ST response)
*The systolic blood pressure response is normal (27 mmHg increase).
*The patient achieved 164% of normal exercise capacity for age, and 110% of normal maximal heart rate for age.
*The patient has a moderate probability of having any clinically significant coronary artery disease and low probability of having severe coronary artery disease.
*Estimated prognosis from treadmill scores is as expected for age, gender and race.

PROGNOSTIC ADDENDUM

*The age expected annual mortality from any cause is 6.5% (National Center for Health Statistics, 1990).
*The Framingham score (Age, Cholesterol, Diabetes, Smoking, LVH) estimates a five year incidence of cardiovascular events (Angina, MI or Death) of 14%.
*The Froelicher score (METs, CHF, SBP rise, and ST depression) estimates an annual cardiovascular mortality of 1.3% (not greater than two times the age expected mortality).
*The Duke Score (METs, ST depression, and treadmill angina) estimates an annual cardiovascular mortality of 1.2% (not greater than two times the age expected mortality).
*The estimated operative mortalities for bypass surgery are 16% (Parsonnet, 1989), 3% (NY State Dept. of Health, 1992) and 1% (VA, 1993).
*The posttest probability for any clinically significant coronary artery disease are 73% (Detrano, 1992), 85% (Morise, 1994) and 32% (Do/Froelicher, 1995).
*The probabilities of having severe coronary artery disease are 18% (Detrano, 1992), 51% (Morise, 1992), 11% (Do/Froelicher, 1995) and 6% (Christian, 1994).

Disclaimer: This Report was computer generated and the results are dependent on rules and correct data entry. It must be overread by a physician

CONCLUSIONS

Simple Clinical and Exercise Scores can be used to Decide which Patients Need Interventions in order to improve their Prognosis.

These Scores could frequently obviate the need for Cardiac Catheterization.

RULES

- The treadmill protocol should be adjusted to the patient and one protocol is not appropriate for all patients
- Report exercise capacity in METs not minutes of exercise
- Hyperventilation prior to testing is not indicated
- ST measurements should be made at ST0 (J-junction) and ST depression should only be considered abnormal if horizontal or downsloping
- Patients should be placed supine as soon as possible post-exercise with a cool down walk avoided in order for the test to have its greatest diagnostic value
- The three minute recovery period is critical to include in analysis of the ST response.
- Measurement of systolic blood pressure during exercise is extremely important and exertional hypotension is ominous; at this point, only manual BP measurement techniques are valid.
- Age-predicted heart rate targets are largely useless because of the wide scatter for any age; a relatively low heart rate can be maximal for a patient of a given age and submaximal for another. Thus, a test should not be considered non-diagnostic if a percentage of age-predicted maximal heart rate (i.e., 85%) is not reached.

DANGERS

The most dangerous circumstances in the exercise testing lab are:

- Testing patients with aortic valvular disease because they can have a cardiovascular collapse (and it is extremely hard to resuscitate them because of the outflow obstruction);
- When patients exhibit ST segment elevation without diagnostic Q-waves (which is due to transmural ischemia); this can be associated with dangerous arrhythmias and infarction; it occurs in about one out of 1000 clinical tests.
- When a patient with an ischemic cardiomyopathy exhibits severe chest pain due to ischemia (angina pectoris); in this instance, a cool down walk is advisable since the ischemia can worsen in recovery;
- When a patient develops exertional hypotension accompanied by ischemia (angina or ST depression) or when it occurs in a patient with a history of CHF, cardiomyopathy or recent MI; and,
- When a patient with a history of sudden death or collapse during exercise develops PVCs which become frequent; in this instance, a cool down walk is advisable since the PVCs can increase in recovery.

Appreciation of these circumstances can help you avoid any complications in your exercise lab.

What are your issues?
Do you think you will be doing TM tests? Do you want to do them?
Do you think the TM test is old technology and that there are better tests?
Do you only want to interpret them and know which tests to order?
If you want to do them, what are your concerns? dangers, stopping