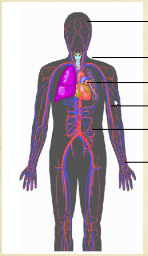
Role of exercise in Heart Failure

ผศ. นพ. วิศาล คันธารัตนกุล

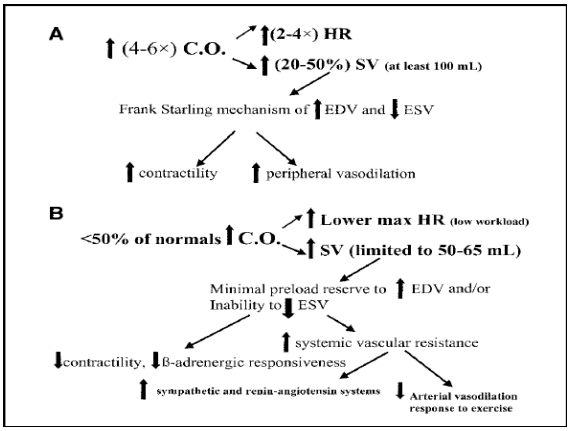
Limitations of people with heart failure

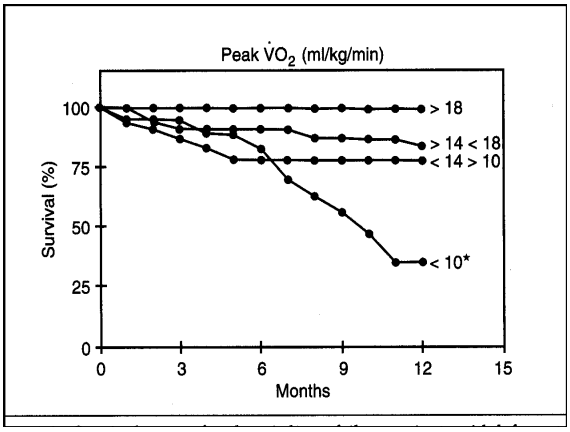


- Psychological — *reduced quality of life*
- Inc. Sympathetic act.
- Central — *dyspnea*
- Endothelial dysf. — *fatigue*
- Inc. RAS
- Peripheral — *fatigue*

reduced activity tolerance

↑Mortality ↑Morbidity ↑ Cost





Ventilatory and Heart Rate Responses to Exercise

Better Predictors of Heart Failure Mortality Than Peak Oxygen Consumption

Mark Robbins, MD; Gary Francis, MD; Fredric J. Pashkow, MD; Claire E. Snader, MA; Kathy Hoercher, RN; James B. Young, MD; Michael S. Lauer, MD

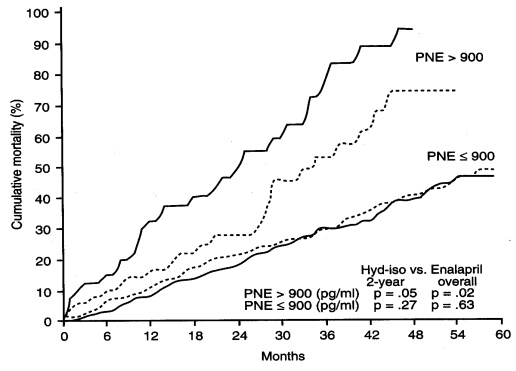
Background—An abnormally low chronotropic response and an abnormally high ventilatory response ($\dot{V}E/\dot{V}CO_2$) to exercise are common in patients with severe heart failure, but their relative prognostic impacts have not been well explored.

Methods and Results—Consecutive patients with heart failure referred for metabolic stress testing who were not taking β-blockers or intravenous inotropes (n=470) were followed for 1.5 years. The chronotropic index was calculated while peak $\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$ were directly measured. Chronotropic index and peak $\dot{V}O_2$ were considered abnormal if in the lowest 25th percentiles of the patient cohort, whereas $\dot{V}E/\dot{V}CO_2$ was considered abnormal if in the highest 25th percentile. For comparative purposes, a group of 17 healthy controls underwent metabolic testing as well. Compared with controls, heart failure patients had markedly abnormal ventilatory and chronotropic responses to exercise. In the heart failure cohort, there were 71 deaths. In univariate analyses, predictors of death included high $\dot{V}E/\dot{V}CO_2$, low chronotropic index, low $\dot{V}O_2$, low resting systolic blood pressure, and older age. Nonparametric Kaplan-Meier plots demonstrated that by dividing the population according to peak $\dot{V}E/\dot{V}CO_2$ and peak $\dot{V}O_2$, it is possible to identify low, intermediate, and very high risk groups. In multivariate analyses, the only independent predictors of death were high $\dot{V}E/\dot{V}CO_2$ (adjusted relative risk [RR] 3.20, 95% CI 1.95 to 5.26, $P<0.0001$) and low chronotropic index (adjusted RR 1.94, 95% CI 1.18 to 3.19, $P=0.0009$).

Conclusions—The ventilatory and chronotropic responses to exercise are powerful and independent predictors of heart failure mortality. (***Circulation.*** **1999;100:2411-2417.)**

Key Words: heart failure ■ mortality ■ exercise ■ heart rate ■ ventilation

below 900 pg/ml.



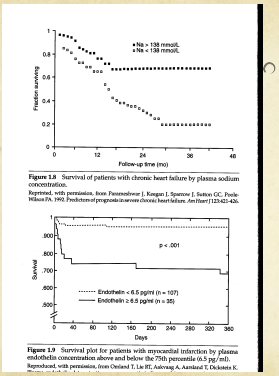


Figure 1.8 Survival of patients with chronic heart failure by plasma sodium concentration.
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Figure 1.9 Survival plot for patients with myocardial infarction by plasma endothelin concentration above and below the 75th percentile (6.5 pg/ml).
Reproduced, with permission, from Omland T, Lie RT, Aakvaag A, Aarsland T, Dickstein K.

- Neurohormonal activation
  - norepinephrine
  - renin
  - aldosterone
  - atrial natriuretic peptides
  - arginine vasopressin
  - sodium : a surrogate measyre for renin–angiotensin–aldosterone activation
  - endothelin

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Compensatory mechanisms

- Activate when left ventricular function is depress
- activate to maintain an adequate cardiac output
- benefit is the short term
- contribute to progressive ventricular and vascular dysfunction

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Compensatory mechanism

- Cardiac adjustment
  - Lt. Ventricular dilatation and concentric hypertrophy *
  - inc. end-diastolic volume resulting in inc. SV *
  - Ventricular remodeling from myocyte loss, interstitial fibrosis, myocardial slippage, and myocyte hyperthrophy
  - Inc. wall stress dec. systolic function

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Compensatory mechanism

- inc passive wall stiffness and slowed-energy dependent myocyte relaxation result in diastolic dysfunction
- impaired ability to regenerate high energy phophate
- direct effects from some substances: endothelin, angiotensin-II
- inc TNF, IL-6
- increase free fatty acid mobilization via neurohormonal stim.
- Blood ketones body elevated

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High risk cardiac patient

- Myocardial ischemia
- Infarct size
- Myocardial tissue at Risk
- Occurrence of ventricular fibrillation or tachycardia
- Neurohormonal factors
  - plasma con of various peptides: NE, renin, aldosterone, atrial naturetic peptide endothelin
  - Plasma Sodium concentrations
- Psychosocial factors

AHA Scientific Statement

Team Management of Patients With Heart Failure

A Statement for Healthcare Professionals From the Cardiovascular Nursing Council of the American Heart Association

Kathleen L. Grady, PhD, RN; Kathleen Dracup, DNSc, RN; Gemma Kennedy, PhD, RN; Debra K. Moser, DNSc, RN; Mariann Piano, PhD, RN; Lynne Warner Stevenson, MD; James B. Young, MD

Heart failure is estimated to affect 4 to 5 million Americans, with 550 000 new cases reported annually.[1] In the past 3 decades, both the incidence and prevalence of heart failure have increased.[1–3] Factors that have contributed to this increase are the aging US population and improved survival rates in patients with cardiovascular disease due to advancements in diagnostic techniques and medical and surgical therapies.[2,4–6] Heart failure is a chronic, progressive disease that is characterized by frequent hospital admissions and ultimately high mortality rates. Because of its high medical resource consumption, heart failure is the most costly cardiovascular illness in the United States.[7]

Advances in the treatment of heart failure and early intervention to prevent decompensation may delay disease progression and improve survival. After initial evaluation, further diagnostic testing, and implementation of standard

Development of an Integrated Approach to Heart Failure Management

Pathophysiology and Definition of Heart Failure

The syndrome of heart failure is a result of complex interactions among molecular, endocrine, and biodynamic systems. There are several pathophysiological mechanisms that are involved in the progression of heart failure; however, cardiac remodeling is more than likely a central feature in the progression of heart failure (Table 1).[8–10] We now recognize that many different injurious processes create altered pump performance and circulatory dynamics.[8] Injury can range from ischemia to myocardial toxins to volume or pressure overload. It can also be linked to genetic causes such as familial cardiomyopathies or even sporadic mutations in sarcomeric proteins. Even though these conditions are linked to different inciting extracellular stimuli (eg, myocardial

สาเหตุการเสียชีวิตฉับพลันในผู้ป่วยกลุ่มนี้ (6-9 เท่า)

- Ventricular tachycardia
- Ventricular fibrillation
- Bradyarrhythmia
- Pump failure
- Recurrent ischemia
- Electromechanical dissociation
- Strokes/embolic phenomenon
- Pulmonary/renal complication

Clinical stability

- Clinical cardiovascular criteria
- Stability of noncardiovascular disease
- Laboratory criteria
- Psychosocial criteria

Cardiovascular criteria

- improved activity since previous visit, walk > 1 city block
- no limitation during dressing
- freedom from evidence of congestion
- absent of angina or present in stable exertional patterns
- No syncope or recurrent symptomatic arrhythmias
- Stable fluid balance with an increase in diuretic dose of not more often than once a week

Cardiovascular criteria

- Stable vital signs

  Blood pressure
  - Systolic heart failure: Systolic pressure > 80 mmHg, no symptoms of postural hypotension, and proportional pulse pressure of > 25 %
  - Heart failure with "preserved EF": control of hypertension

  Heart rate
  - Sinus rhythm: generally < 85 BPM at rest
  - AF: generally > 85 BPM at rest,< 110 bpm with routine activity

Noncardiovascular disease

- control of pulmonary disease with lowest possible doses of systemic corticosteroids and inhaled beta-adrenergic agents
- control of hyperglycemia without hypoglycemia
- effective therapy of chronic blood loss or anemia

Laboratory criteria

- stable renal function, generally with serum Cr < 2.0 mg/dL and BUN < 50 mg/dL; may be higher in patients with known intrinsic renal disease
- stable serum sodium, generally > 134 mEq/L

Psychosocial criteria

- Compliance with medical regimen
- Social support
- absence of serious depression or dementia

Role of Exercise Training in HF

- Exercise Capacity **
- Catecholamines *
- Ventilatory Responses **
- Endothelial Function *
- Myocardial Adaptations ?
- Mortality ?
- Quality of Life ?*

Role of Exercise Training in HF

- Adaptations are mostly peripheral
- Activation of neuro-hormonal system
- Central adaptation

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

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Exercise and Heart Failure: A Statement From the American Heart Association Committee on Exercise, Rehabilitation, and Prevention

Ileana L. Piña, Carl S. Apstein, Gary J. Balady, Romualdo Belardinelli, Bernard R. Chaitman, Brian D. Duscha, Barbara J. Fletcher, Jerome L. Fleg, Jonathan N. Myers and Martin J. Sullivan

Circulation 2003;107:1210-1225
DOI: 10.1161/01.CIR.0000055013.92097.40
Circulation is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514

 Print ISSN: 0009-7322. Online ISSN: 1524-4539

Authors (Year of Publication)	No. of Patients	Exercise Program: Duration, wk	Exercise Program: Activity, Intensity, and Frequency	$\dot{V}O_2$ Increase, % (Outcome vs Controls)
Jette et al (1991)[84]	18	4	Mon–Fri. AM: Jog at 70% to 80% max HR for 5 min 3×/wk; calisthenics 30 min; cycle 15 min at 70% to 80% max HR. PM: Walk 30 to 60 min.	22 in group with EF <30%
Belardinelli et al (1992)[83]	20	8	Cycle at 40% peak $\dot{V}O_2$ 3×/wk.	20
Coats et al (1992)[87]	17	8	Cycle 20 min at 60% to 80% max HR 3×/wk.	18
Belardinelli et al (1995)[88]	55	8	Cycle 40 min at 60% $\dot{V}O_2$max 3×/wk.	12
Hambrecht et al (1995)[89]	22	3	Walk 10 min 6×/d at 70% $\dot{V}O_2$max 2×/wk.	31
Keteyian et al (1996)[90]	29	24	Rating of perceived exertion, 12–14. Treadmill, cycle, rowing, and arm ergometer at 60% exercise capacity for 33 min 3×/wk.	16.3
Radaelli et al (1996)[84]	6	5	Cycle 20 min 5 d/wk.	15
Dubach et al (1997)[87]	25	4	Walk 60 min 2×/d; cycle 40 min 4×/wk at 80% $\dot{V}O_2$max.	26
Tyni-Lenne et al (1997)[85]	16	8	Knee extensor: 60 repeats/min for 15 min for 8 wk (at 65% peak work rate for 4 wk and then 75% peak work rate for 4 wk).	14
Callaerts-Vegh et al (1998)[86]	17	8	Walk 1 h, 2×/d; cycle 45 min at 70% to 80% HR reserve 4×/wk.	30.9
Reinhart et al (1998)[67]	25	8	Cycle 40 min at 70% to 80% max capacity 4×/wk; walk 1 hour 2×/d.	29
Belardinelli et al (1999)[82]	99	8 (plus maintenance)	Cycle at 60% peak $\dot{V}O_2$ 3×/wk for 8 wk. Maintenance: 2×/wk for 12 mo.	18 at 2 mo; 23 at 14 mo
Taylor (1999)[69]	8	8	Train 30 min at 45% to 70% peak $\dot{V}O_2$ 3×/wk.	17.6
Sturm et al (1999)[89]	26	12 (plus maintenance)	Step aerobics and cycle at 50% capacity for 12 wk; then step aerobics 100 min/wk and cycle 50 min/wk.	23.3
Keteyian et al (1999)[70]	43	24	Treadmill, cycle, and arm ergometer at 60% to 80% max HR for 33 min 3×/wk.	14.3

Authors (Year of Publication)	Exercise Program	Improvement
Kiilavuori et al (1999)[78]	Cycle 30 min 3×/wk at 50% to 60% $\dot{V}O_2$max for 3 mo.	Plasma noreginephrine decreased at rest; no adverse effects on catabolic hormones
Coats et al (1992)[87]	Cycle 20 min 3×/wk at 60% to 80% max HR for 8 wk.	Improved HR variability; decreased ventilation
Belardinelli et al (1995)[88]	Cycle 40 min 3×/wk at 60% $\dot{V}O_2$max for 8 wk.	Improved indices of diastolic function
Hambrecht et al (1995)[89]	Walk 10 min 6×/d at 70% $\dot{V}O_2$max for 3 wk.	Increased muscle mitochondria volume density
Radaelli et al (1996)[84]	Cycle 20 min 5 d/wk for 5 wk.	Improved autonomic control of HR
Tyni-Lenne et al (1997)[85]	Knee extensor for 8 wk.	Increased citrate synthetase, lactate dehydrogenase; improved QOL
Belardinelli et al (1999)[82]	Cycle 3×/wk at 60% peak $\dot{V}O_2$ for 8 wk. Maintenance: 2×/wk for 12 mo.	Fewer hospital readmissions for HF and reduced mortality
Taylor (1999)[69]	Train 3×/wk for 8 wk.	Peak cardiac index increased by 10%
Keteyian et al (1999)[70]	Treadmill, cycle, and arm ergometer 33 min at 60% to 80% max HR 3×/wk for 24 wk.	Improved chronotropic response to exercise
Maiorana et al (2000)[71]	Circuit weight training for 8 wk; 1 h, 3×/wk; 55% MVC for 4 wk; 65% MVC after 4 wk.	Isotonic voluntary contractile skeletal muscle strength increased by 17.9%

HR indicates heart rate (bpm); max, maximum; and MVC, maximal voluntary contractile strength.

AHA/ACC Conference Proceedings

Evaluating Quality of Care for Patients With Heart Failure

Harlan M. Krumholz, MD, Chair; David W. Baker, MD, MPH, Cochair; Carol M. Ashton, MD; Sandra B. Dunbar, RN, DSN; Gottlieb C. Friesinger, MD; Edward P. Havranek, MD; Mark A. Hlatky, MD; Marvin Konstam, MD; Diana L. Ordin; Ileana L. Pina, MD; Betram Pitt, MD; John A. Spertus, MD, MPH

The First American Heart Association/American College of Cardiology Scientific Forum on Quality of Care and Outcomes Research in Cardiovascular Disease and Stroke was held from May 24 to May 26, 1999, in San Diego, Calif. In this report, the Heart Failure Working Group examines efforts to assess care for patients with heart failure (HF) and makes recommendations about quality measures. The working group includes healthcare professionals (cardiologists, general internists, and nurses) from academia. Representatives of governmental agencies, industry, and healthcare systems also have input. The group has expertise in the treatment of HF and/or quality improvement. Several panel members have participated in writing guidelines for management of HF, and several were involved in the 1994 effort by the Agency for Health Care Policy and Research (AHCPR) to develop guidelines for management of patients with HF and to remain without symptoms despite major LV dysfunction and (sometimes) a poor prognosis.

The pathophysiology of the cardiac dysfunction must be an integral part of the definition of HF because therapy and prognosis are greatly influenced by the specific abnormalities present. Cardiac pathophysiology is a critical feature in developing quality indicators and risk-stratification models for this complex and diverse syndrome. Echocardiography is best suited for evaluating pathophysiology to determine if HF is principally related to contractile dysfunction or filling abnormalities. This technique can determine systolic left or right ventricular function, ventricular hypertrophy, diastolic filling abnormalities, regional myocardial wall motion abnormalities, valvular abnormalities, and pericardial thickening or effusions. Echocardiography can also estimate pulmonary artery systolic pressure. In patients with suspected ischemic

TABLE 2. Proposed Future Process-of-Care Measures, Ratings of Evidence Supporting Measure, and Ability to Abstract Information From Charts

	Process-Outcome Link*			Abstraction*			
	Mortality	Quality of Life	Admissions	Reliability	Validity	Burden	Limitations
β-Blockers prescribed if LVEF ≤0.35	A	A	A	A	A	A	Applicable to outpatients with NYHA class I–III. Functional status may not be recorded.
Spironolactone prescribed if LVEF ≤0.35	B	?	B	A	A	A	Generalizability and target population (ie, "ideal candidates") not defined.
Patients with a history of past MI or current angina should be evaluated for ischemia	C	C	C	B	C	C	Does not apply to patients who are not candidates for revascularization or who refuse procedures. Difficult to assess by chart review.
Daily weight recorded on ≥50% of days in hospital	?	?	?	A	A	B	No data linking this measure to outcomes. Not recommended by an expert panel.
Blood pressure control (systolic blood pressure ≤120 mm Hg and diastolic blood pressure ≤80 mm Hg for patients with LVEF ≤0.40. Systolic blood pressure ≤140 mm Hg and diastolic blood pressure ≤90 mm Hg for patients with LVEF >0.40.)	?	?	?	A	A	A	No direct process-outcome link, although studies have shown that blood pressure control can prevent development of HF. Not recommended by an expert panel to date.
Exercise training	?	A	?	A	A	A	Unclear if formal training program superior to informal training program. No financing mechanism for most patients. Generalizability and target population (ie, ideal candidates) not defined. Many patients may refuse.

*Level of evidence for a process-outcome link: A indicates multiple randomized controlled trials; B, single randomized trial or nonrandomized trials; and C, consensus of experts. Question mark indicates contradictory studies or insufficient published data to provide a rating. Ratings for abstraction reliability, validity, and burden are subjective: A indicates excellent; B, good; and C, fair or poor.

Authors (Year of Publication)	Exercise Program	Improvement
Kiilavuori et al (1999)[78]	Cycle 30 min 3×/wk at 50% to 60% Vo_2max for 3 mo.	Plasma noreginephrine decreased at rest; no adverse effects on catabolic hormones
Coats et al (1992)[67]	Cycle 20 min 3×/wk at 60% to 80% max HR for 8 wk.	Improved HR variability; decreased ventilation
Belardinelli et al (1995)[58]	Cycle 40 min 3×/wk at 60% Vo_2max for 8 wk.	Improved indices of diastolic function
Hambrecht et al (1995)[59]	Walk 10 min 6×/d at 70% Vo_2max for 3 wk.	Increased muscle mitochondria volume density
Radaelli et al (1996)[64]	Cycle 20 min 5 d/wk for 5 wk.	Improved autonomic control of HR
Tyni-Lenne et al (1997)[66]	Knee extensor for 8 wk.	Increased citrate synthetase, lactate dehydrogenase; improved QOL
Belardinelli et al (1999)[62]	Cycle 3×/wk at 60% peak Vo_2 for 8 wk. Maintenance: 2×/wk for 12 mo.	Fewer hospital readmissions for HF and reduced mortality
Taylor (1999)[69]	Train 3×/wk for 8 wk.	Peak cardiac index increased by 10%
Ketoyian et al (1999)[70]	Treadmill, cycle, and arm ergometer 33 min at 60% to 80% max HR 3×/wk for 24 wk.	Improved chronotropic response to exercise
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HR indicates heart rate (bpm); max, maximum; and MVC, maximal voluntary contractile strength.

Exercise testing

- Due to limitation of FC < 5 METs
- Use Naughton protocol / ramp protocol with increment 0.2 – 0.4 METs per second or 1-2 METs/ stage
- Gas exchange measurements should be used when feasible

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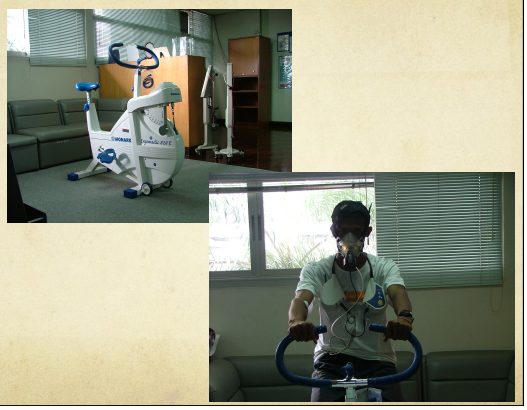

Exercise recommendation

- assessment and management of possible limitations to exercise
- optimal pharmacology
- nutritional support

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Exercise recommendation

- dyspnea management(supplemental oxygen, mobilization of secreations)
- Ventilatory breathing exercise
  purse lip breathing
  inspiratory muscle training
  diagphragmatic breathing
  inhibition of accessory muscle

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Risks

- Very low
- Activities performed with continuous ECG monitoring have the lowest rates of sudden cardiac arrest compared with those that are unmonitored or only intermittently monitored.
- High risk: MI, VF, Poor systolic response, survival of CPR

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Authors (Year of Publication)	No. of Patients	EF, %	Exercise Program: Duration, wk	Exercise Program: Intensity (% Peak HR or $\dot{V}O_2$)	Adverse Events
(1) Conn et al (1982)[132]	10	29	5 to 8	70% to 80% HR	None during training
(2) Sullivan et al (1988)[66]	12	24	16 to 24	75% $\dot{V}O_2$	Worsened HF (n=1); exhaustion (n=1)
(3) Jette et al (1991)[65]	7	24	4	70% to 80% HR	Worsened HF (n=3); ventricular tachycardia (n=1)
(4) Meyer et al (1991)[135]	12	23	6	70% to 80% HR	Worsened congestive HF (n=1)
(5) Coats et al (1992)[67]	17	19	8	70% to 80% HR	None during training
(6) Koch et al (1992)[80]	12	26	12	Individualized protocol	None during training
(7) Belardinelli et al (1995)[134]	16	31	8	40% $\dot{V}O_2$	None during training
(8) Belardinelli et al (1995)[68]	36	28	8	60% $\dot{V}O_2$	Atrial fibrillation (n=1); hypotension (n=2)
(9) Hambrecht et al (1995)[69]	12	26	24	70% $\dot{V}O_2$	Atrial arrhythmia (n=1)
(10) Keteyian et al (1996)[60]	15	21	24	60% to 80% HR	None during training
(11) Kavanagh et al (1996)[46]	15	22	52	50% to 60% $\dot{V}O_2$	None during training but worse HF (n=5) after training
(12) Killavuori et al (1996)[135]	12	24	24	50% to 60% $\dot{V}O_2$	Not reported
(13) Wilson et al (1996)[59]	32	23	12	60% to 70% HR	Extreme exhaustion (n=3)
(14) Demopoulos et al (1997)[136]	16	21	12	50% to 80% $\dot{V}O_2$	None during training
(15) Dubach et al (1997)[61]	12	32	8	70% to 80% $\dot{V}O_2$	None during training
(16) Meyer et al (1997)[137]	18	21	3	50% $\dot{V}O_2$	None during training
(17) European Heart Failure Training Group (1998)[74]	134	25	6 to 16	70% to 80% HR	None during training
(18) Hambrecht et al (1998)[77]	10	24	24	70% $\dot{V}O_2$	None during training
(19) Belardinelli et al (1999)[62]	50	28	52	60% $\dot{V}O_2$	None during training
(20) Hare et al (1999)[138]	9	26	11	Resistance training	None during training

HR indicates heart rate (bpm).

Contraindications or delay in initiating

- **Recent hospitalization for, or clinically unstable, heart failure**
- **Heart failure medication not optimized**
- **Severe stenotic or primary regurgitant valvular heart disease**
- **Significant intercurrent illness, not substantially resolved**
- **Recent procedure that requires delay in starting exercise training** (ICD)

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Contraindications or delay in initiating

- **Exercise testing results that would preclude safe exercise training**
- **Uncertain: New York Heart Association functional class IV, secondary severe mitral or tricuspid regurgitation, uncontrolled atrial fibrillation**

Exercise prescription

- **ไม่มีกฎแน่นอน โดยมากให้ใช้ระดับเบาคือ 40%–50% of Vo2 max** = 65% of HR max
- **ไม่มีระดับความหนักเบาขั้นต่ำ ผป. บางรายอาจจะเกิดประโยชน์จากการเคลื่อนไหวเล็กน้อย**
- RPE = 12-13
- Resistance training in stable cases

with low weight(1-2 lbs) and dynamic exercise

Exercise prescription

- special precaution and strict to criteria
- For diastolic dysfunction avoid resting vigorous heart rate
- For systolic dysfunction avoid isometric and heavy work load even the heart rate is not raise

ตัวอย่างโปรแกรมสำหรับ EC < 3 METs

Week	%EC	Total min	Min Ex	Min rest	Reps
1	50	10-15	3-5	3-5	3-4
2	50	12-20	5-7	3-5	3
3	50-60	15-25	7-10	3-5	3
4	50-60	20-30	10-15	2-3	2
5	60-70	25-40	12-20	2	2
6	60-70	30-45	15-25	2	1-2

ตัวอย่างโปรแกรมสำหรับ EC 3 - 5 METs

Week	%EC	Total min	Min Ex	Min rest	Reps
1	50-60	15-20	7-10	2-3	3
2	50-60	20-30	10-15	2	2
3	60-70	25-40	15-20	2	2
4	60-70	30-45	20-30	2	1-2

Exercise prescription

- status change very quickly, pt. Should reevaluate every exercise session
- warm-up and cool down should be prolonged
- use low intensity/long duration sessions
- PRE and dyspnea scales are very useful

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Exercise prescription

- avoid isometric exercise
- ECG monitors in Hx of V tach, cardiac arrest, or exertional hypotension
- consider investigation to developing exercise program, do not exceed work load that produces wall motion abnormalities, a drop in EF, a pulmonary wedge pressure ›20 mmHg, or above ventilatory threshold

Relative criteria for participation in an exercise program

- ability to speak without signs and symptoms of dyspnea(able to speak comfortably with RR < 30 beat/min
- not more than moderate fatigue by subjective clinical impression
- crackles present in less than one half of the lungs
- cardiac index > 2L/min/m2 or CVP < 12 mmHg

Indicators for modifying or termination of exercise in CHF patients

- marked dyspnea of fatigue (RPE > 3/10 or 15/20) RR > 40 BPM
- Development of S3 or pulmonary crackles
- Increase in pulmonary crackles
- Significant increase in the sound of the second component of the second heart sound (P2)

Indicators for modifying or termination of exercise in CHF patients

- Poor pulse pressure< 10 mmHg
- Decrease in HR or BP > 10 BPM or mmHg
- Increasing supraventricular or ventricular ectopy
- Diaphoresis, pallor, or confusion

Journal of the American College of Cardiology
© 2007 by the American College of Cardiology Foundation
Published by Elsevier Inc.

Vol. 49, No. 24, 2007
ISSN 0735-1097/07/$32.00
doi:10.1016/j.jacc.2007.04.013

EDITORIAL COMMENT

Exercise Training in Heart Failure

Contradictory or Conventional?*

Stanley A. Rubin, MD, FACC
Los Angeles, California

The paintings of the Belgian surrealist René Magritte (1898 to 1967) juxtapose 2 incongruous, illogical, or even contradictory objects: a steam locomotive projecting from a domestic fireplace (Time Transfixed, 1939); a bright daytime sky over a nighttime scene of a dimly lighted street (The Empire of Light, 1954); or a picture of a pipe with a caption on the canvas that reads, in French, "This is not a pipe" (The Treachery of Images, 1929). On the basis of conventional wisdom, the current palette of therapy for heart failure also would seem incongruous, illogical, or contradictory if it included exercise therapy. However, change is at hand.

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(3) that advocated limited exercise rehabilitation and training based on available evidence-based medicine perhaps marked the tipping point away from the conventional view. Subsequently, an American College of Cardiology/American Heart Association (ACC/AHA) guideline statement (2) recommended exercise training in patients with heart failure. A formalized recognition of this recommendation is part of the current ACC/AHA guidelines (4) for patients with current or prior symptoms of heart failure with reduced systolic function: exercise training is a Class I recommendation (Level of Evidence B). What is the evidence basis for this sea change in exercise training as therapy for heart failure, what are the limitations of the recommendations (both patient- and payer-centered), and what should clinicians now do with this information?

Studies of exercise training suggest some beneficial effects. The ACC/AHA statement (2), noted previously, reviewed 15 exercise trials in systolic dysfunction heart failure that included from 17 to 99 patients (total of 426) patients, the typical range of study patients in such trials. These trials used a wide range of training programs that differed by setting, types of activity, intensity, and duration, the latter ranging from 4 to 24 weeks. It found a modest improvement in Vo_{2max} that occurred after a few weeks (range 12% to 31%) that was sustained for a number of months if the training program continued. A Cochrane review of 29 studies with 1,126 patients similarly reported that exercise training resulted (in the subset of studies providing data) in modest increases of Vo_{2max} (range ~10%

- Conventional wisdom excluded the role of exercise training in the management of heart failure.
- Studies of exercise training suggest some beneficial effects.

Conclusions.

So, is heart failure and exercise training contradictory or conventional?

Looking forward to the likely beneficial effect of exercise training, as well as additional beneficial therapies available to treat heart failure, we may one day look upon such a patient and say,

"This is not a heart failure patient."

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Wrap Up messages

- HF is best candidate to exercise
- HF is safe to exercise
- Consider risk group & optimal management before start exercise program
- Exercise prescription is related to FC

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