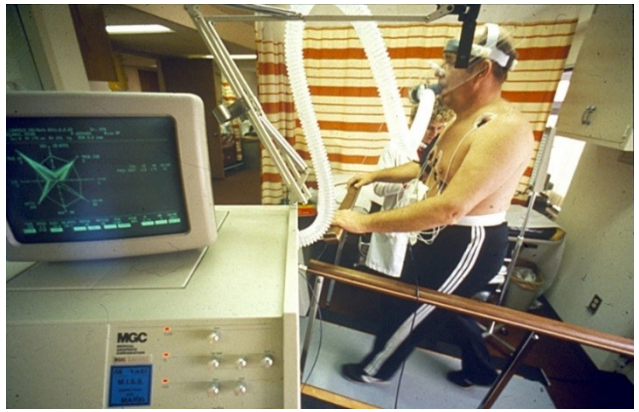
CLINICAL EXERCISE PHYSIOLOGY: What a Physician Should Know ?



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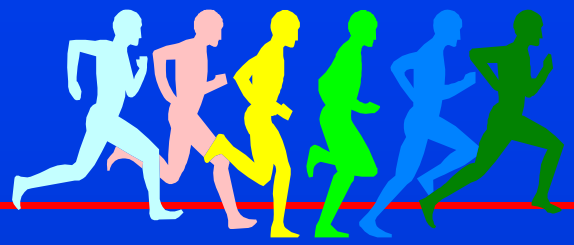
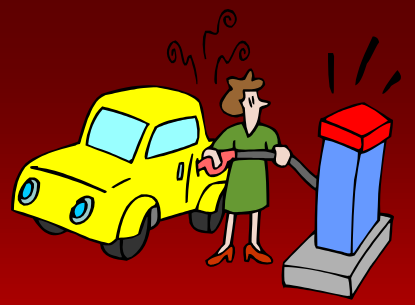
Disclosures: None

Outline

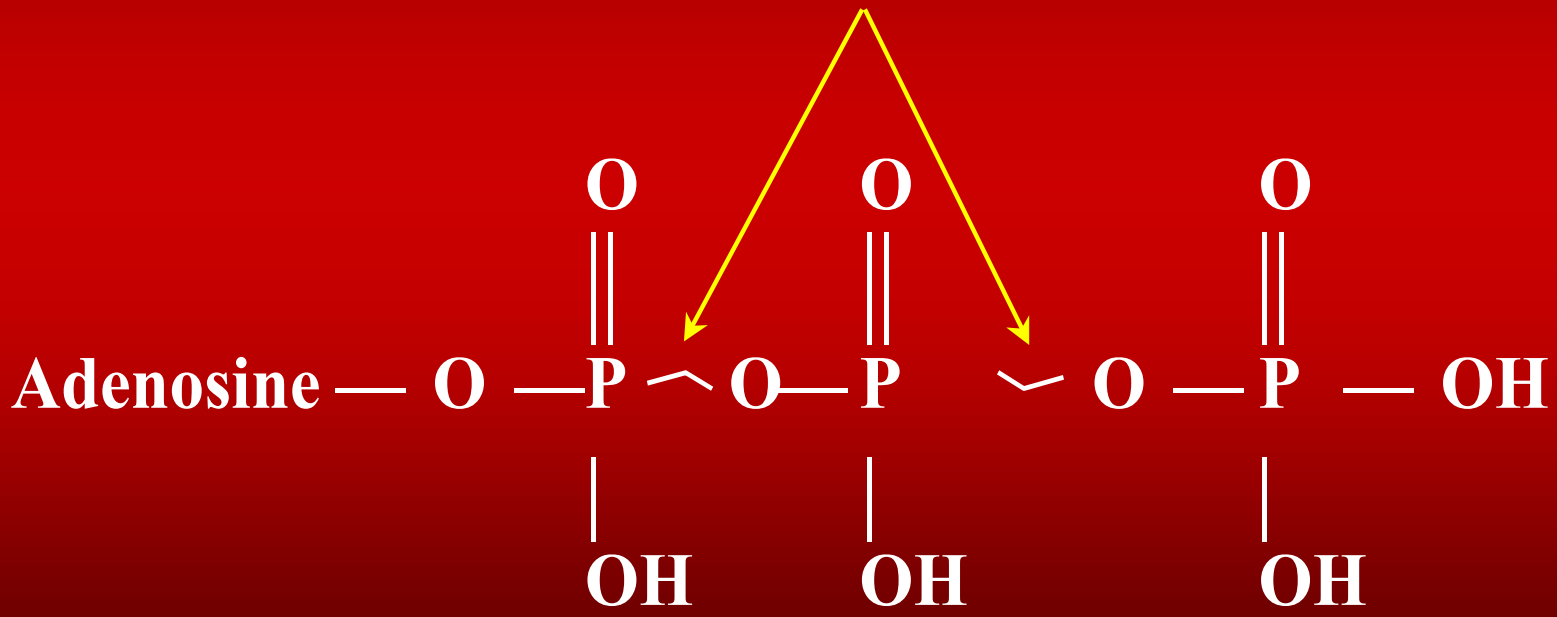


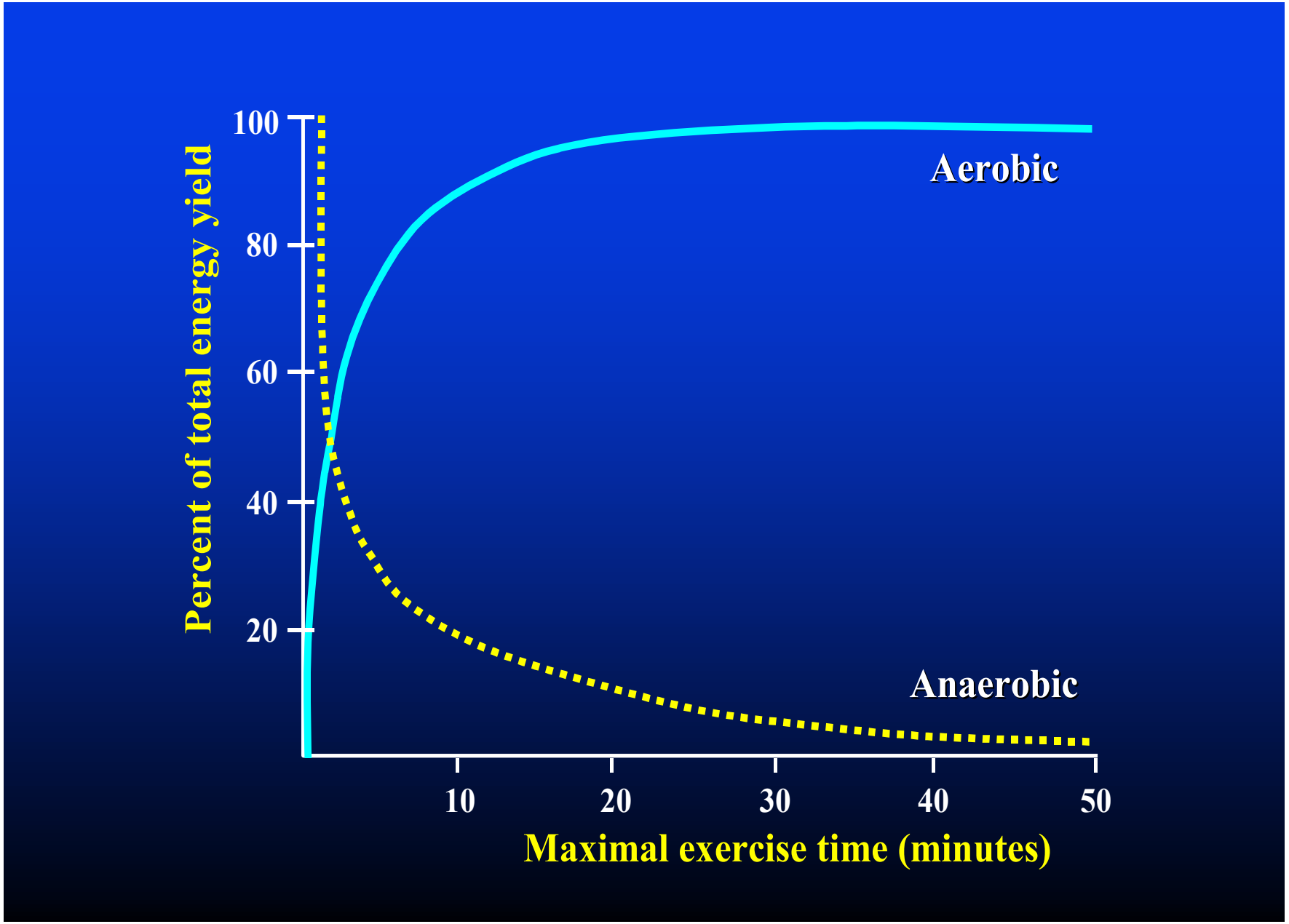
- Energy systems for exercise
- Acute cardiorespiratory responses ($\dot{V}O_2$ max)
- Metabolic equivalents (METs)
- Anaerobic (Ventilatory) Threshold
- Fitness and Mortality
- Fitness and Surgical Outcomes
- Clinical Considerations: CPX Testing

Triphosphate

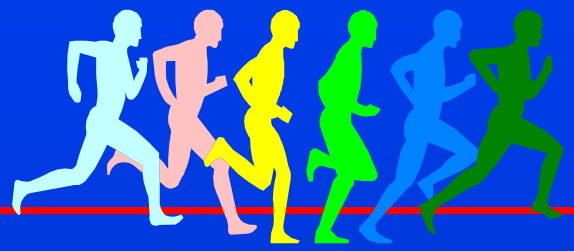


High-energy bonds




Percent of total energy yield
100
80
60
40
20
Aerobic
Anaerobic
10
20
30
40
50
Maximal exercise time (minutes)

Outline



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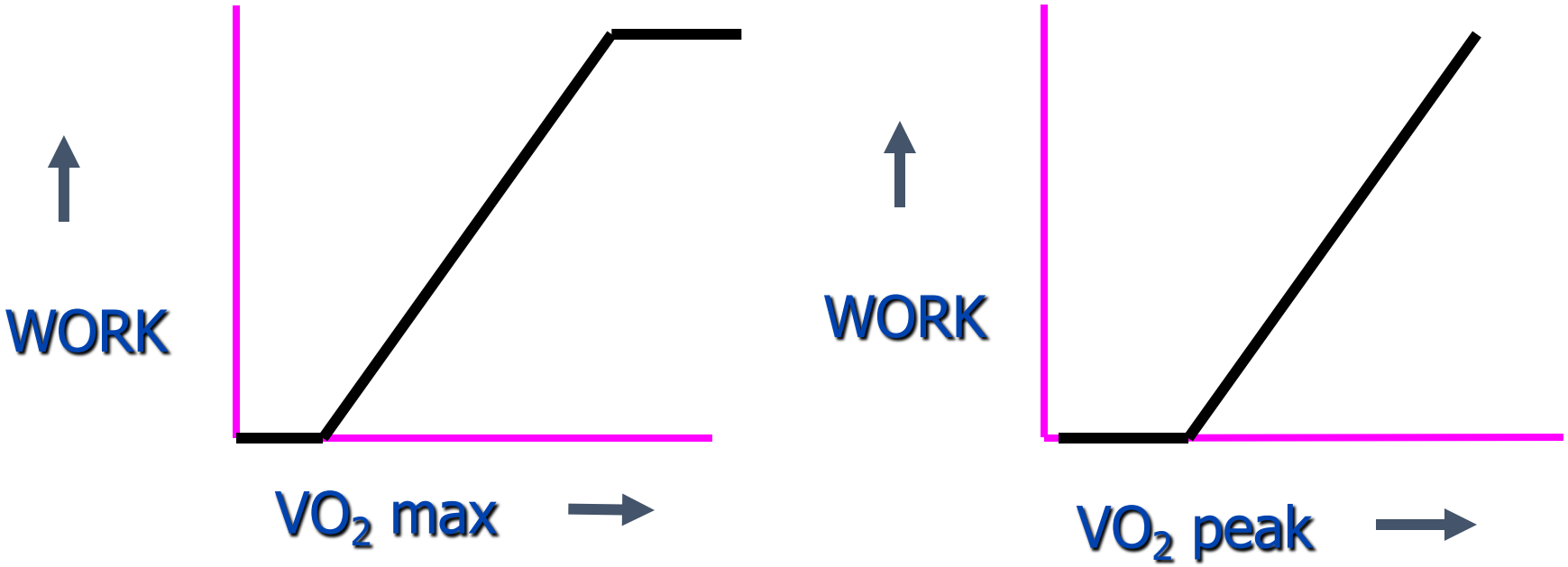
VO_2 max versus VO_2 peak

TREADMILL



Oxygen Consumption

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

Where $\dot{V}O_2$ is oxygen consumption in ml/min; HR is heart rate in bpm; SV is stroke volume in ml/beat; and $CaO_2 - C\bar{v}O_2$ is the arteriovenous oxygen difference in ml/dL of blood.

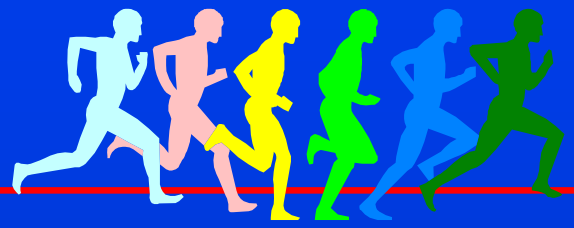
Key Players

Variable	Rest → Exercise	Relative Increase
Heart Rate		2.7 x ↑
Stroke Volume		1.4 x ↑
Cardiac Output		4 x ↑
a-$\bar{v}$ O_2 Difference		3 x ↑
Blood Pressure	SBP DBP	1.3 – 1.5 x ↑ ↔or ↓
Pulmonary Ventilation		15-25 x ↑

OXYGEN-CARRYING CAPACITY OF BLOOD: TRANSPORT MECHANISMS

- Dissolved in plasma
  (0.3 ml O_2 /100 ml plasma)
- Combined with hemoglobin (Hb)
  1 gm of Hb carries 1.34 ml O_2
  ~ 15 gm Hb/100 ml blood
  O_2 Capacity = 15 x 1.34 =
  20 ml O_2 /100 ml blood

Outline



- Energy systems for exercise
- Acute cardiorespiratory responses ($\dot{V}O_2$ max)
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Resting Metabolic Rate*

- 5,000 ml blood/min x 5 ml O_2/100 ml blood = 250 ml O_2/min = 1.25 Kcal/min
- 250 ml O_2/min ÷ 70 kg = 3.5 ml O_2/kg/min
- 3.5 ml O_2/kg/min = 1 MET

*70 kg man

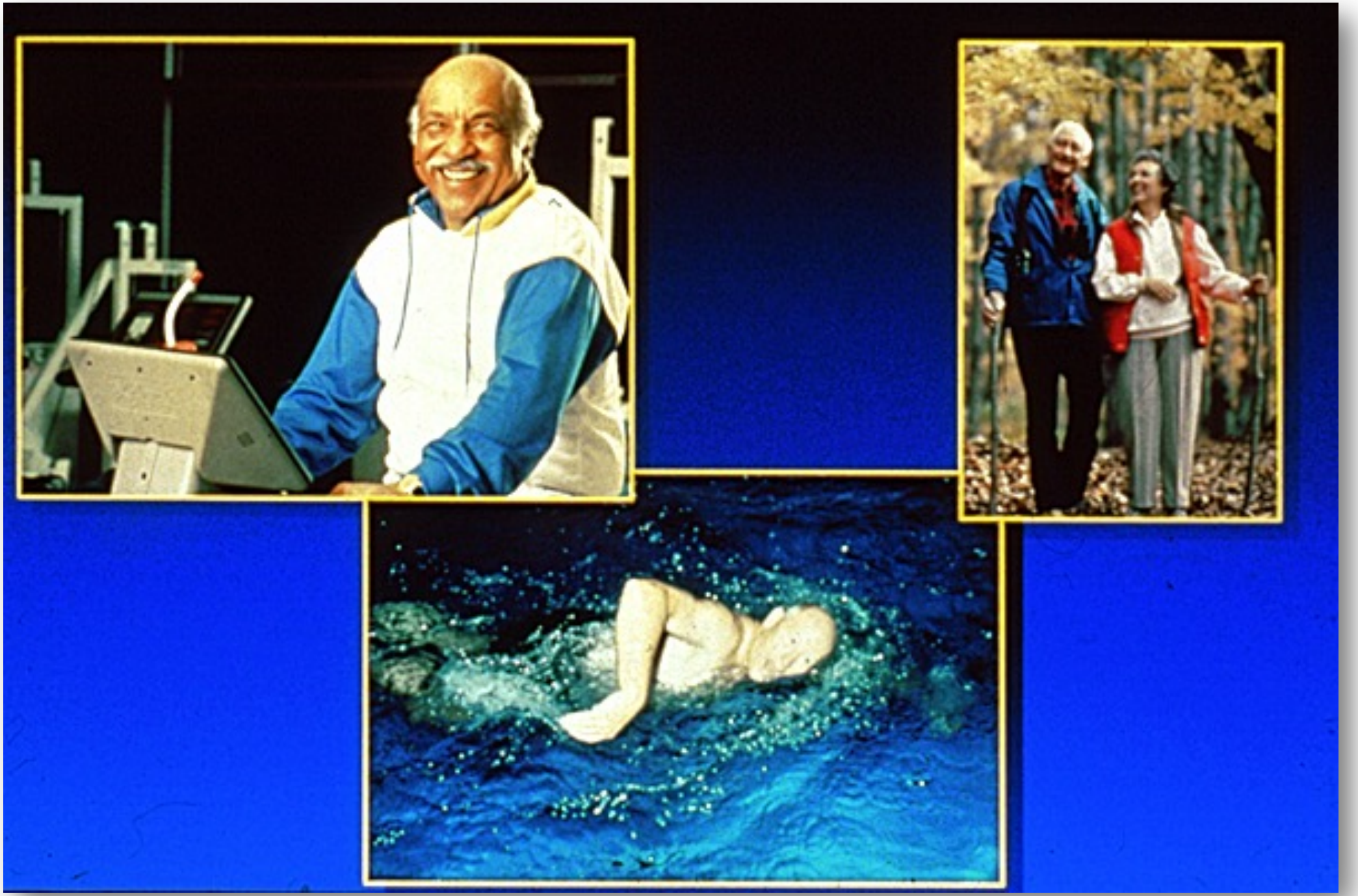
Exercise Metabolic Rate

- 20,000 ml blood/min x 15 ml O_2/100 ml blood = 3,000 ml O_2/min = 15 Kcal/min
- 3,000 ml O_2/min ÷ 70 kg = 42.9 ml O_2/kg/min
- 42.9 ml O_2/kg/min ÷ 3.5 = 12 METs

The typical 12-fold increase in oxygen transport and utilization achieved at maximal exercise is brought about by respective increases in the hemodynamic correlates of VO_2, that is, a 4-fold increase in cardiac output and a 3-fold increase in arterio-venous oxygen difference (4 x 3 = 12 METs)

Understanding Exercise Intensity Expressed as Metabolic Equivalents (METs) & Exercise Prescription

- 1 MET = Amount of oxygen consumed at rest
- 2 METs = 2 mph , 0% grade
- 3 METs = 3.0 mph, 0% grade or 2.0 mph, 3.5 % grade
- The ~ energy cost (METs) of varied activities has been measured directly or estimated. These include:
- Sexual Activity = 2-3 METs
- Table Tennis or Ping Pong = 2-3 METs
- Singles Tennis = 7 METs
- Jogging at 6 mph = 10 METs

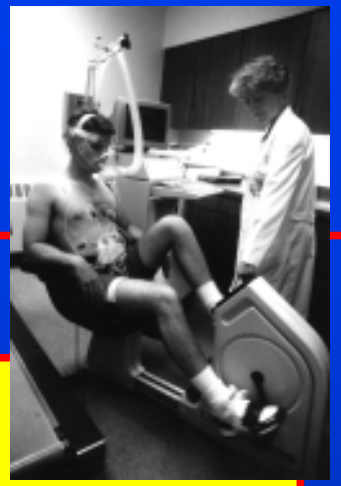


Maximal Oxygen Consumption for Varied Population Subsets

Group	METs
Normals	10-12
Cardiacs	6-8
Endurance Athletes	15-20+

The reduced aerobic (MET) capacity in the cardiac patient appears primarily due to decreased maximal cardiac output, secondary to reduced stroke volume and/or heart rate, rather than impairment in the peripheral extraction of oxygen.

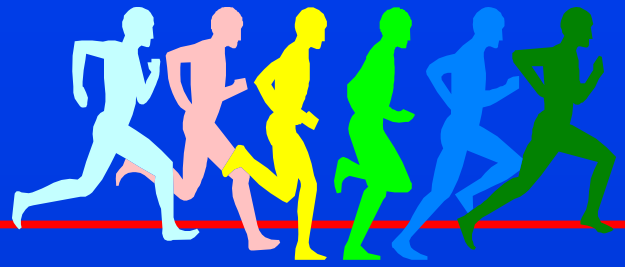
Measurement of $\dot{V}O_2$



$$\dot{V}O_2 = \dot{V}_E (F_I O_2 - F_E O_2)$$

Where $\dot{V}_E$ is the expired minute ventilation, $F_E O_2$ is the directly measured concentration of O_2 in the expired air, $F_I O_2$ is the concentration of oxygen in the inspired air, and normal room air is 0.2093.

Outline



- Energy systems for exercise
- Acute cardiorespiratory responses ($\dot{V}O_2$ max)
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- **Anaerobic (Ventilatory) Threshold**
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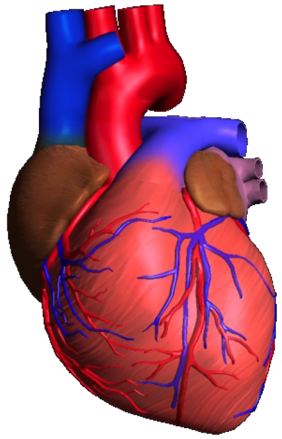
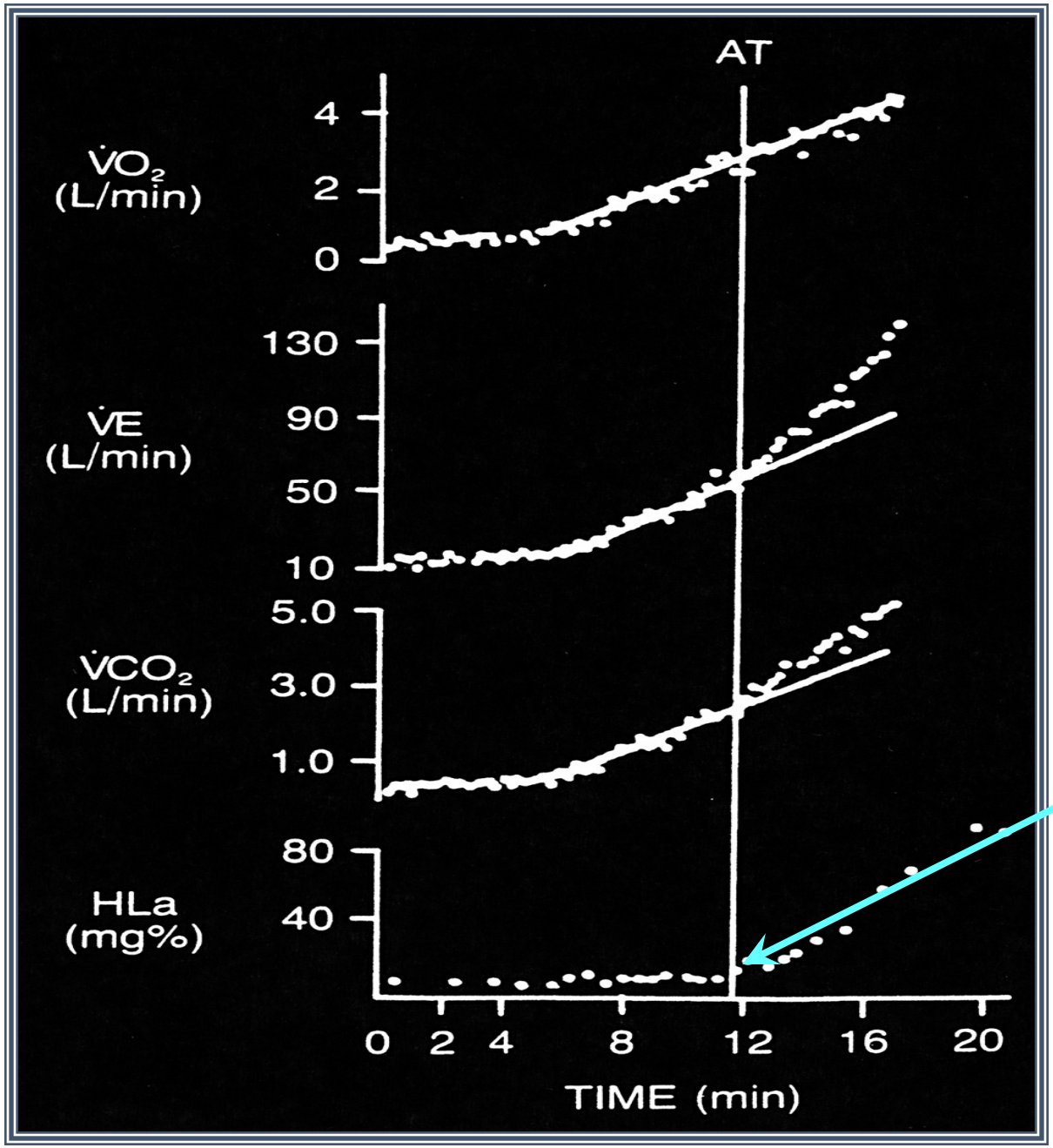


Anaerobic or Ventilatory Threshold

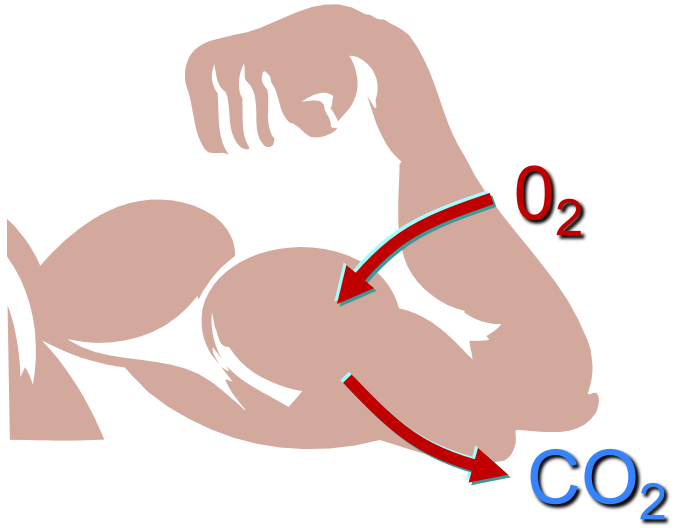

428
39
196
39



Anaerobic Threshold

J Appl Physiol 1976;41:544

Understanding the Ventilatory Threshold

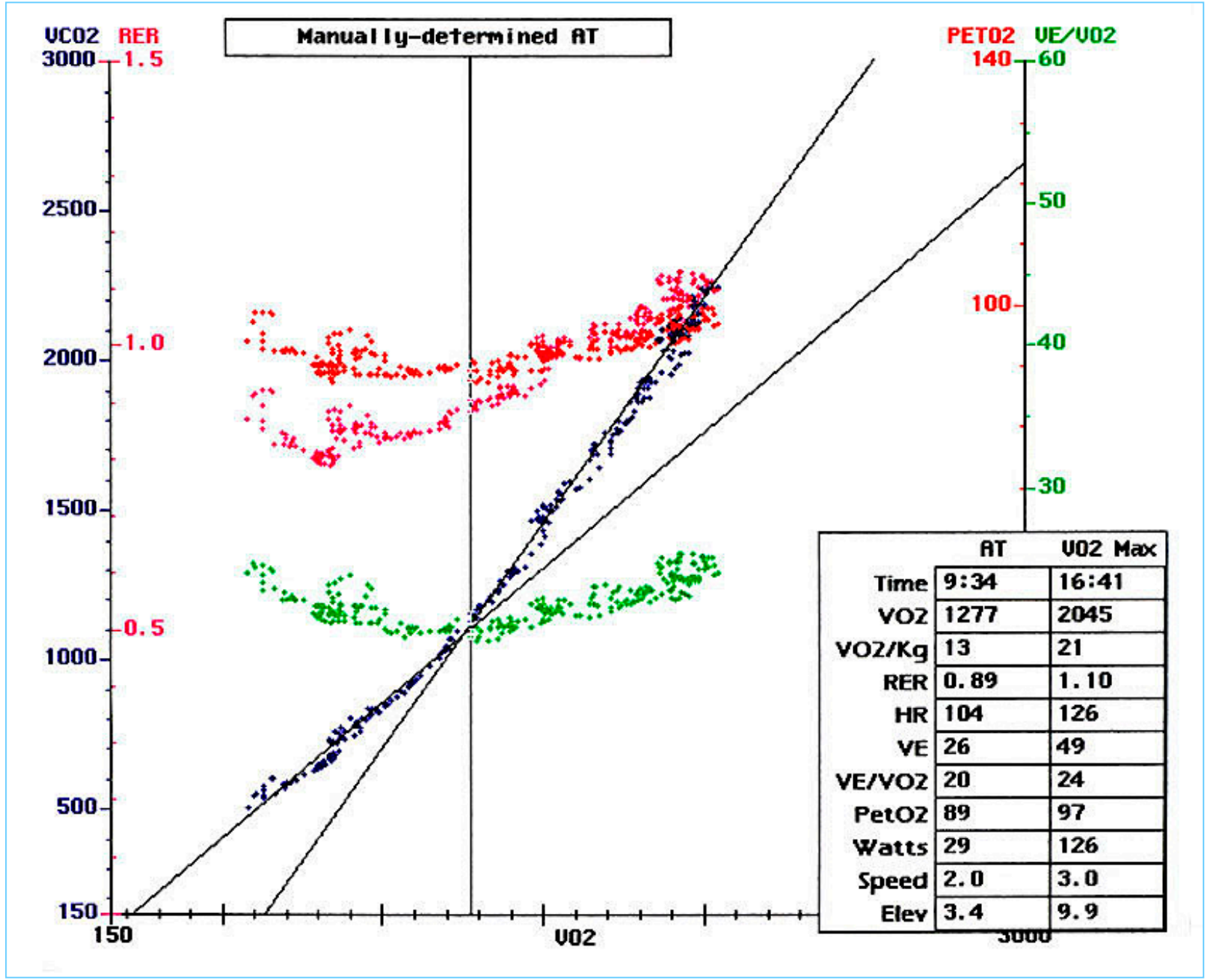


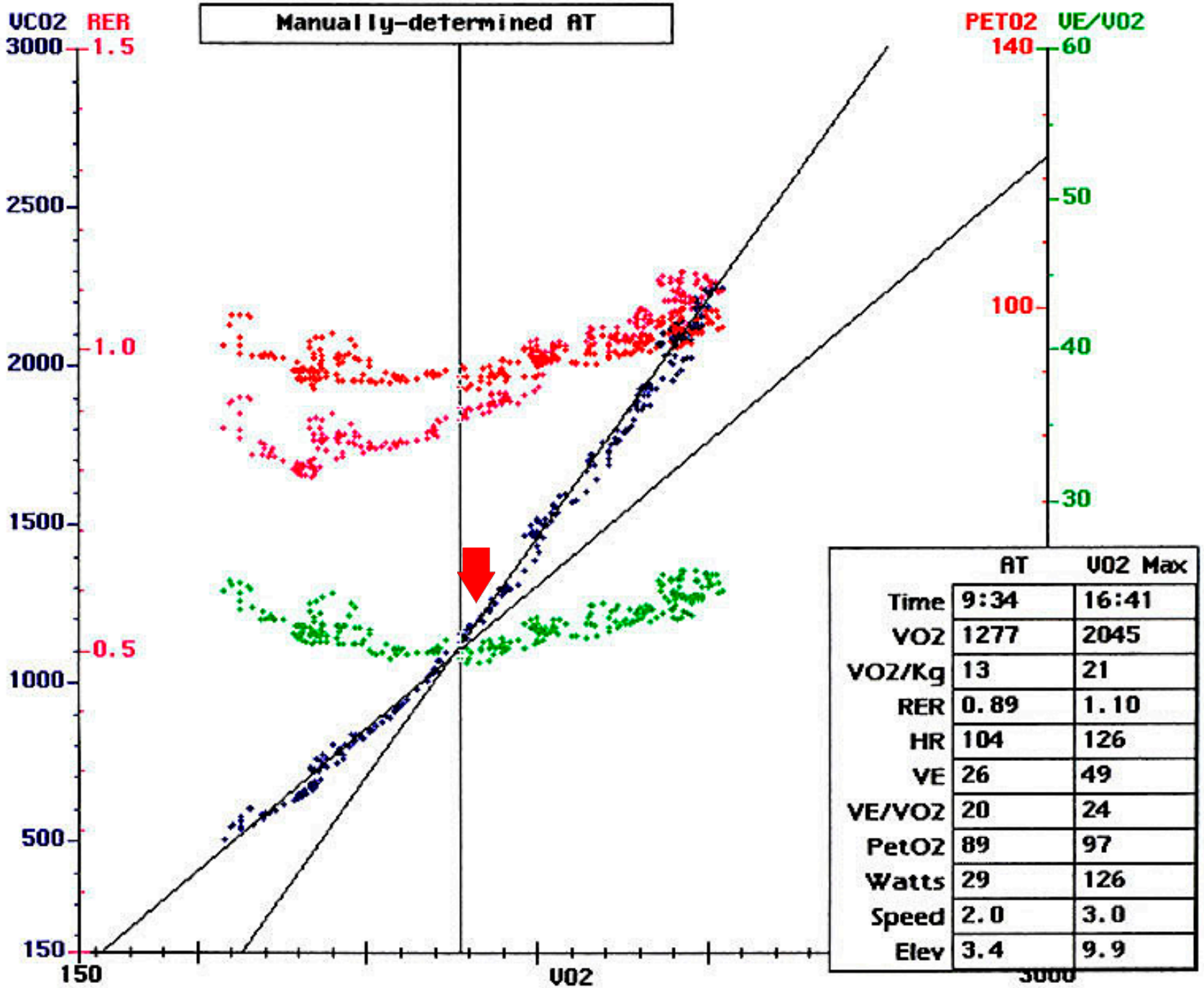
$$HLA + NaHCO_3 \rightarrow NaLa$$

(lactic acid)(sodium bicarbonate) (sodium lactate)

$$+ H_2CO_3 \rightarrow H_2O + CO_2$$

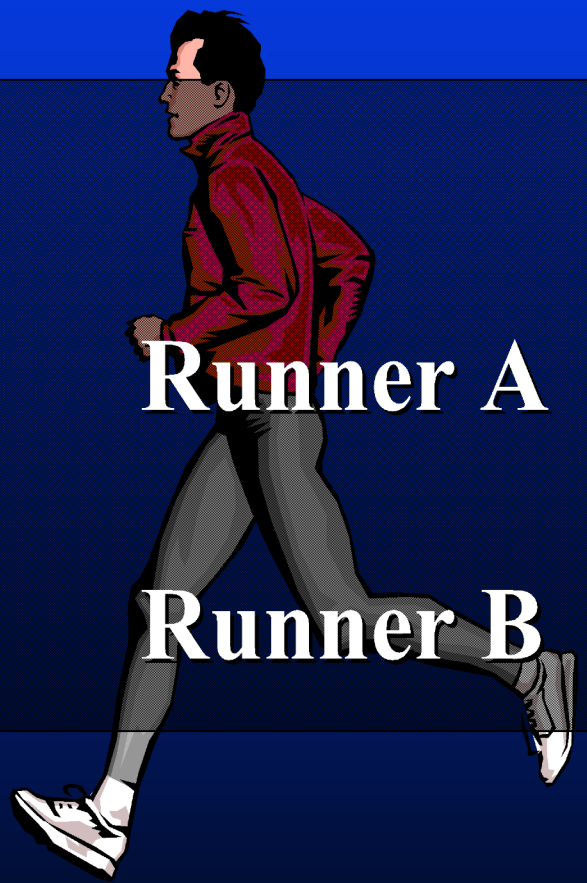
(carbonic acid)

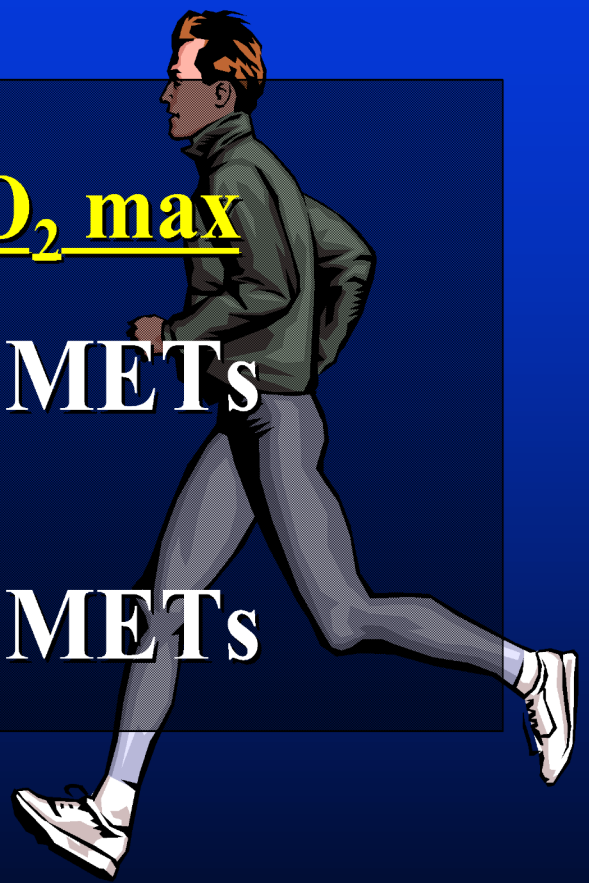

Manually-determined AT
VCO2
RER
PETO2
VE/VO2
3000
2500
2000
1500
1000
500
150
1.5
1.0
0.5
140
100
60
50
40
30
150
VO2
3000
AT
VO2 Max
Time 9:34 16:41
VO2 1277 2045
VO2/Kg 13 21
RER 0.89 1.10
HR 104 126
VE 26 49
VE/VO2 20 24
PetO2 89 97
Watts 29 126
Speed 2.0 3.0
Elev 3.4 9.9



	AT	VO2 Max
Time	9:34	16:41
VO2	1277	2045
VO2/Kg	13	21
RER	0.89	1.10
HR	104	126
VE	26	49
VE/VO2	20	24
PetO2	89	97
Watts	29	126
Speed	2.0	3.0
Elev	3.4	9.9

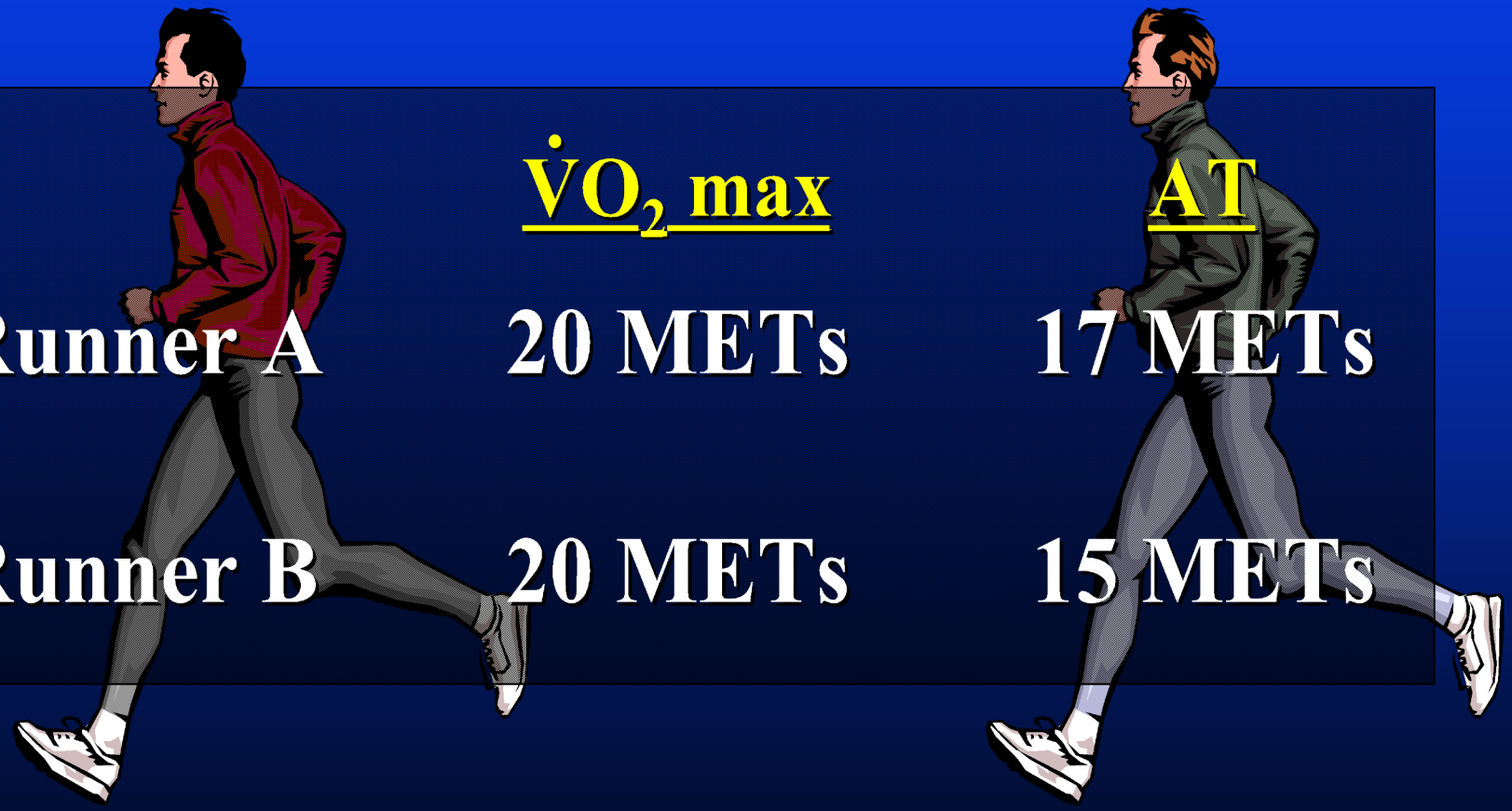
Who Would You Bet On?





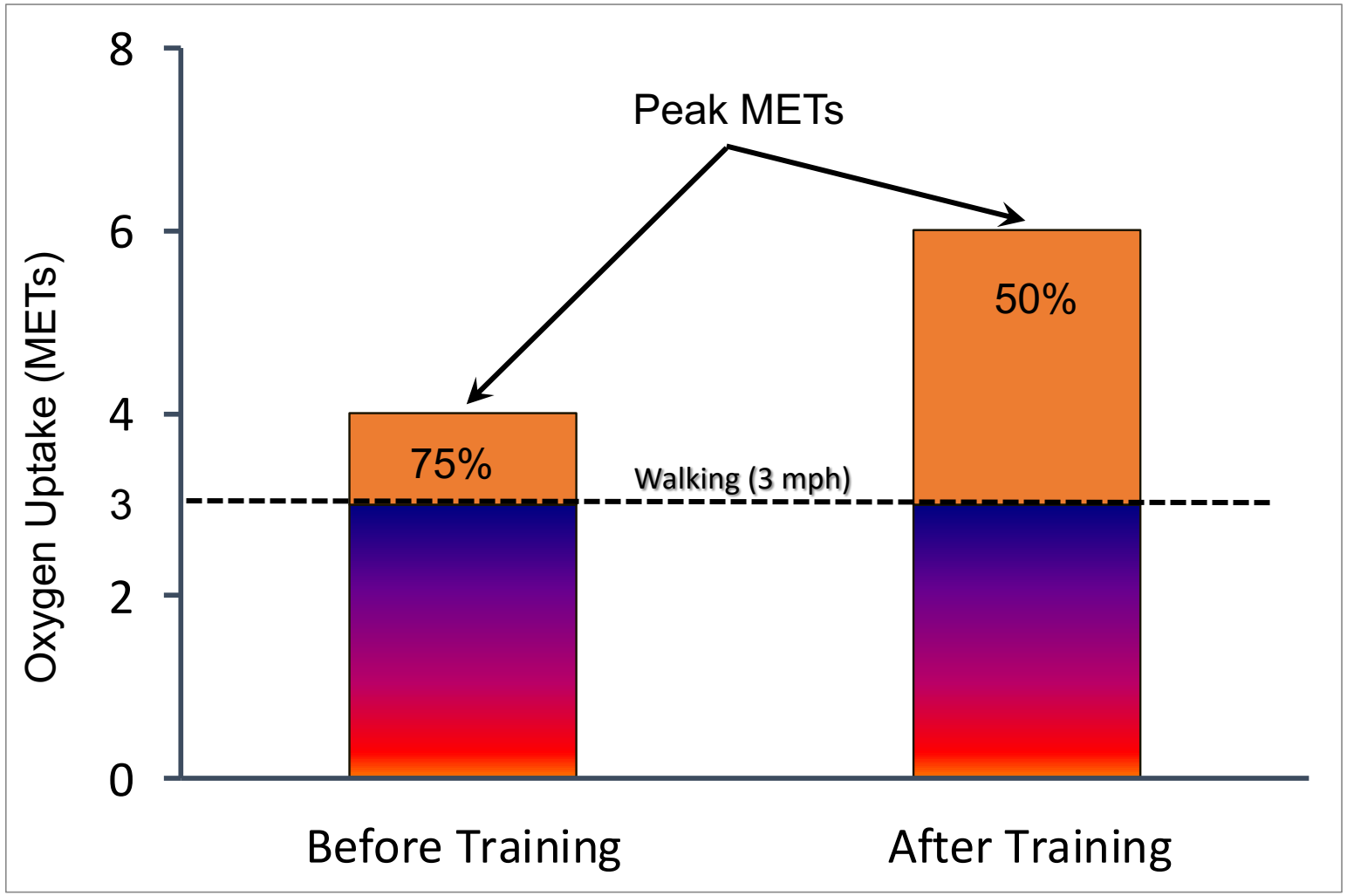
	$\dot{V}O_2$ max
Runner A	20 METs
Runner B	20 METs

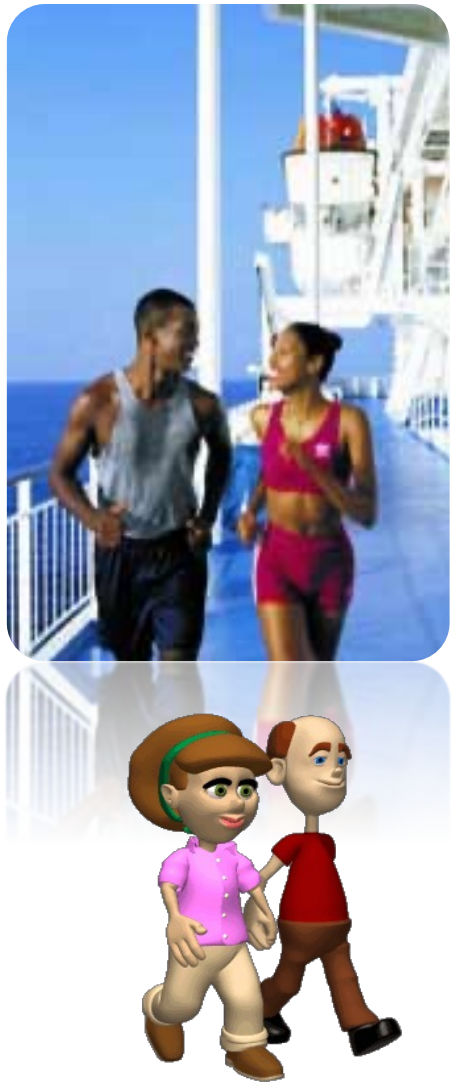
Who Would You Bet On?



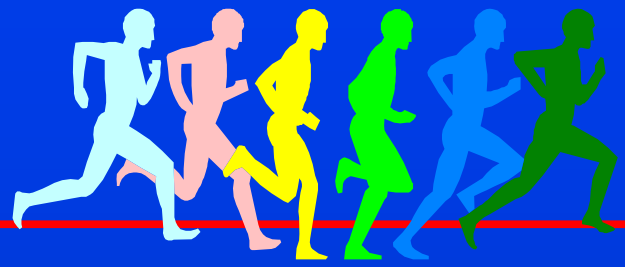
	$\dot{V}O_2$ max	AT
Runner A	20 METs	17 METs
Runner B	20 METs	15 METs

Why Increased Peak METs → ↓ Fatigue





Outline



- Energy systems for exercise
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- **Fitness and Mortality (Seminal Studies)**
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- Clinical Considerations: CPX Testing

MET Capacity: An Underutilized Prognostic Indicator (Men)

Age-adjusted mortality rates in healthy men categorized by level of fitness

Exercise capacity is a stronger predictor of mortality than established risk factors of hypertension, smoking, and diabetes, and stress testing parameters of ST-segment depression, peak HR, or arrhythmias during exercise

Myers, J. Circulation 2003;107:e2-e5

ORIGINAL CONTRIBUTION

Relationship Between Low Cardiorespiratory Fitness and Mortality in Normal-Weight, Overweight, and Obese Men

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James B. Kampert, PhD
Carolyn E. Barlow, MS
Milton Z. Nichaman, MD, ScD
Larry W. Gibbons, MD, MPH
Ralph S. Paffenbarger, Jr, MD, DrPH
Steven N. Blair, PED

Context Recent guidelines for treatment of overweight and obesity include recommendations for risk stratification by disease conditions and cardiovascular disease (CVD) risk factors, but the role of physical inactivity is not prominent in these recommendations.

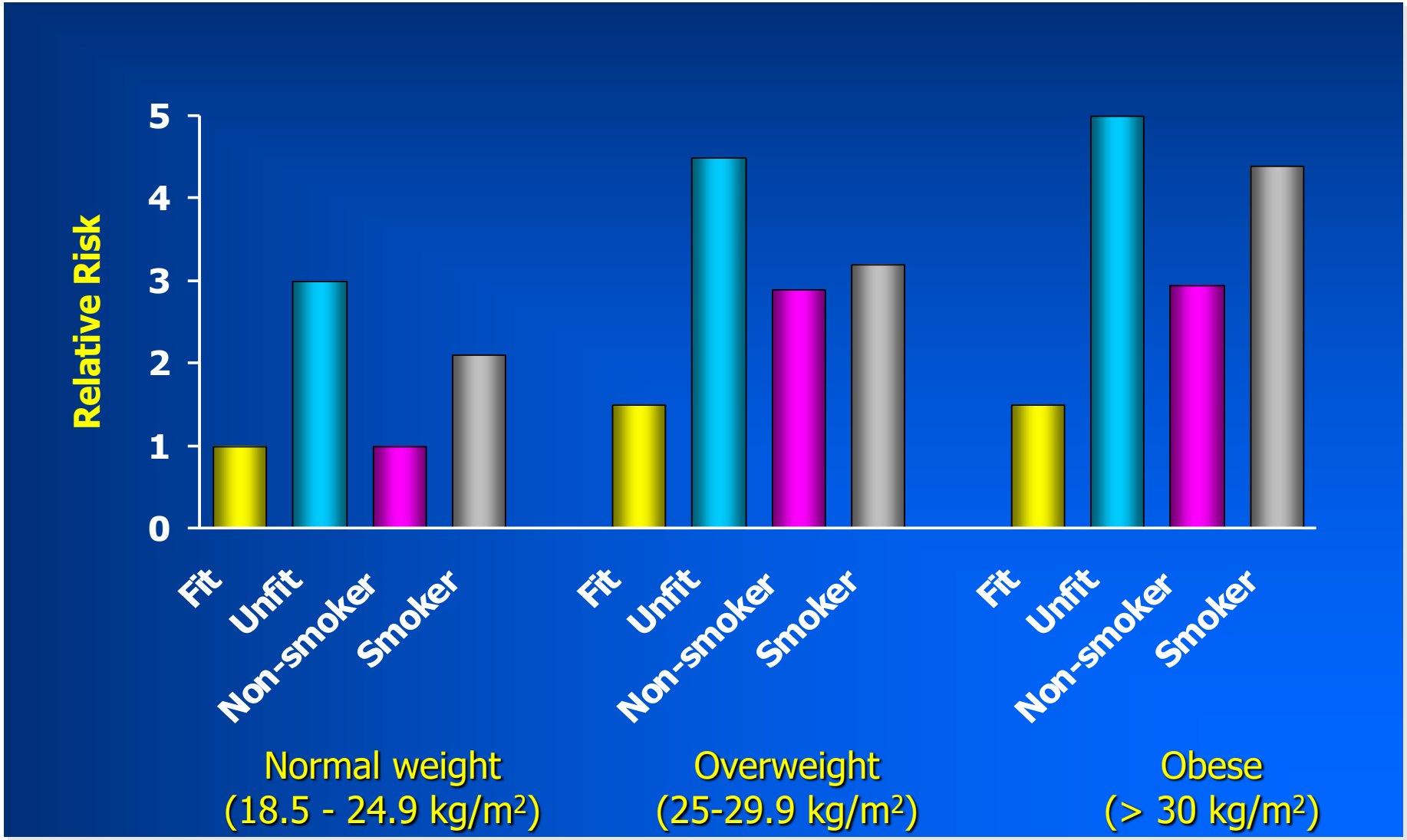
Objective To quantify the influence of low cardiorespiratory fitness, an objective marker of physical inactivity, on CVD and all-cause mortality in normal-weight, overweight, and obese men and compare low fitness with other mortality predictors.

Design Prospective observational data from the Aerobics Center Longitudinal Study.

Setting Preventive medicine clinic in Dallas, Tex.

Participants A total of 25 714 adult men (average age, 43.8 years [SD, 10.1 years])

Low Cardiovascular Fitness Increases Relative Risk of All-Cause Mortality vs. Being Fit



Wei M. et al. JAMA 1999;282:1547

Although physical activity or exercise training may not make all people lean, it appears that an active way of life may have important health benefits, even for those who remain overweight.

Cardiorespiratory Fitness as a Quantitative Predictor of All-Cause Mortality and Cardiovascular Events in Healthy Men and Women

A Meta-analysis

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Hitoshi Shimano, MD, PhD
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Nobuhiro Yamada, MD, PhD
Hirohito Sone, MD, PhD

Context Epidemiological studies have indicated an inverse association between cardiorespiratory fitness (CRF) and coronary heart disease (CHD) or all-cause mortality in healthy participants.

Objective To define quantitative relationships between CRF and CHD events, cardiovascular disease (CVD) events, or all-cause mortality in healthy men and women.

Data Sources and Study Selection A systematic literature search was conducted for observational cohort studies using MEDLINE (1966 to December 31, 2008) and EMBASE (1980 to December 31, 2008). The Medical Subject Headings search terms used included *exercise tolerance, exercise test, exercise/physiology, physical fitness, oxygen consumption, cardiovascular diseases, myocardial ischemia, mortality, mortalities, death, fatality, fatal, incidence,* or *morbidity*. Studies reporting associations of baseline CRF with CHD events, CVD events, or all-cause mortality in healthy participants were included.

Data Extraction Two authors independently extracted relevant data. CRF was estimated as maximal aerobic capacity (MAC) expressed in metabolic equivalent (MET) units. Participants were categorized as low CRF (<7.9 METs), intermediate CRF (7.9-10.8 METs), or high CRF (≥10.9 METs). CHD and CVD were combined into 1 outcome (CHD/CVD). Risk ratios (RRs) for a 1-MET higher level of MAC and for participants with lower vs higher CRF were calculated with a random-effects model.

Data Synthesis Data were obtained from 33 eligible studies (all-cause mortality, 102 980 participants and 6910 cases; CHD/CVD, 84 323 participants and 4485 cases). Pooled RRs of all-cause mortality and CHD/CVD events per 1-MET higher level of MAC (corresponding to 1-km/h higher running/jogging speed) were 0.87 (95% confidence interval [CI], 0.84-0.90) and 0.85 (95% CI, 0.82-0.88), respectively. Compared with participants with high CRF, those with low CRF had an RR for all-cause mortality of 1.70 (95% CI, 1.51-1.92; $P<.001$) and for CHD/CVD events of 1.56 (95% CI, 1.39-1.75; $P<.001$), adjusting for heterogeneity of study design. Compared with participants with intermediate CRF, those with low CRF had an RR for all-cause mortality of 1.40 (95% CI, 1.32-1.48; $P<.001$) and for CHD/CVD events of 1.47 (95% CI, 1.35-1.61; $P<.001$), adjusting for heterogeneity of study design.

Conclusions Better CRF was associated with lower risk of all-cause mortality and CHD/CVD. Participants with a MAC of 7.9 METs or more had substantially lower rates of all-cause mortality and CHD/CVD events compared with those with a MAC of less 7.9 METs.

JAMA. 2009;301(19):2024-2035 www.jama.com

CORONARY HEART DISEASE (CHD) is a major cause of disability and premature death throughout the world.[1] Epidemiological studies have demonstrated an inverse association between physical fitness and the incidence of CHD or all-cause mortality in healthy or asymptomatic participants. Physical fitness is typically expressed as cardiorespiratory fitness (CRF) and is assessed by exercise tolerance testing[2]; however, it is rare for clinicians to consider CRF when evaluating future risk of CHD.[3]

A major reason for lack of consideration of CRF as a marker of CHD risk may be that the quantitative association of CRF for cardiovascular risk is not well established. The degree of risk reduc-

CME available online at www.jamaarchivescme.com and questions on p 2053.

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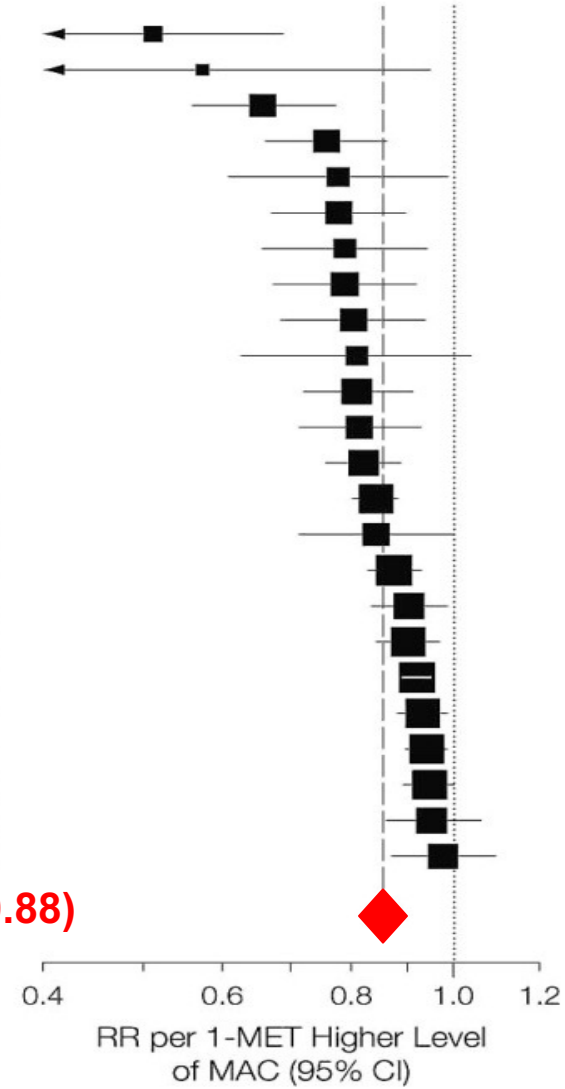
Clinical Review Section Editor: Mary McGrae McDermott, MD, Contributing Editor. We encourage authors to submit papers for consideration as a Clinical Review. Please contact Mary McGrae McDermott, MD, at mdm608@northwestern.edu.

Kodama S et al.
JAMA 2009;301:2024

CHD/CVD

Allen et al,[31] 1980 [women]	1.32	0.51 (0.38-0.68)
Sobolski et al,[52] 1987	0.49	0.57 (0.35-0.94)
Allen et al,[31] 1980 [men]	3.12	0.65 (0.56-0.76)
Bruce et al,[34] 1980	3.66	0.75 (0.65-0.85)
Peters et al,[48] 1983	1.70	0.77 (0.60-0.98)
Arraiz et al,[32] 1992	3.37	0.77 (0.66-0.89)
Miller et al,[6] 2005	2.54	0.78 (0.65-0.94)
Gulati et al,[39] 2005	3.11	0.78 (0.67-0.91)
Rywik et al,[49] 2002	2.98	0.79 (0.68-0.93)
Cumming et al,[35] 1975	1.58	0.80 (0.62-1.03)
Jouven et al,[43] 2005	4.22	0.80 (0.71-0.90)
Sawada and Muto,[51] 1999	3.77	0.81 (0.71-0.92)
Gyntelberg et al,[41] 1980	5.36	0.81 (0.75-0.88)
Mora et al,[46] 2003	6.59	0.83 (0.79-0.87)
Stevens et al,[21] 2002 [women]	2.83	0.83 (0.70-0.99)
Laukkanen et al,[8] 2007	6.28	0.87 (0.82-0.92)
Erriksen et al,[37] 2004	5.32	0.90 (0.83-0.98)
Stevens et al,[22] 2004	5.89	0.90 (0.84-0.96)
Sui et al,[7] 2007 [men]	7.18	0.91 (0.89-0.94)
Stevens et al,[21] 2002 [men]	6.48	0.93 (0.88-0.98)
Slattery and Jacobs,[5] 1988	6.86	0.94 (0.90-0.97)
Balady et al,[33] 2004 [men]	6.43	0.94 (0.89-0.99)
Sui et al,[7] 2007 [women]	4.67	0.94 (0.85-1.05)
Balady et al,[33] 2004 [women]	4.27	0.97 (0.87-1.09)
Overall	**100.00**	**0.85 (0.82-0.88)**

Test for heterogeneity: $I^2 = 74.7\%$; $P < .001$



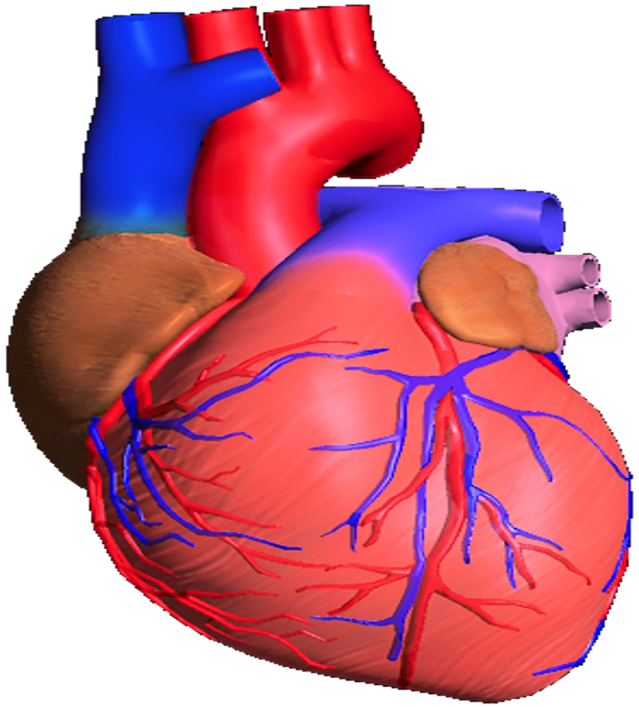
CHD indicates coronary heart disease; CI, confidence interval; CVD, cardiovascular disease; MAC, maximal aerobic capacity; MET, metabolic equivalent; RR, risk ratio. Area of each square is proportional to study weight.

Kodama S et al. JAMA 2009;301:2024

A Memorable Teaching Experience:

My ‘Stupid’ ?

Fitness *versus* Heart Function



Left Ventricular
Ejection Fraction

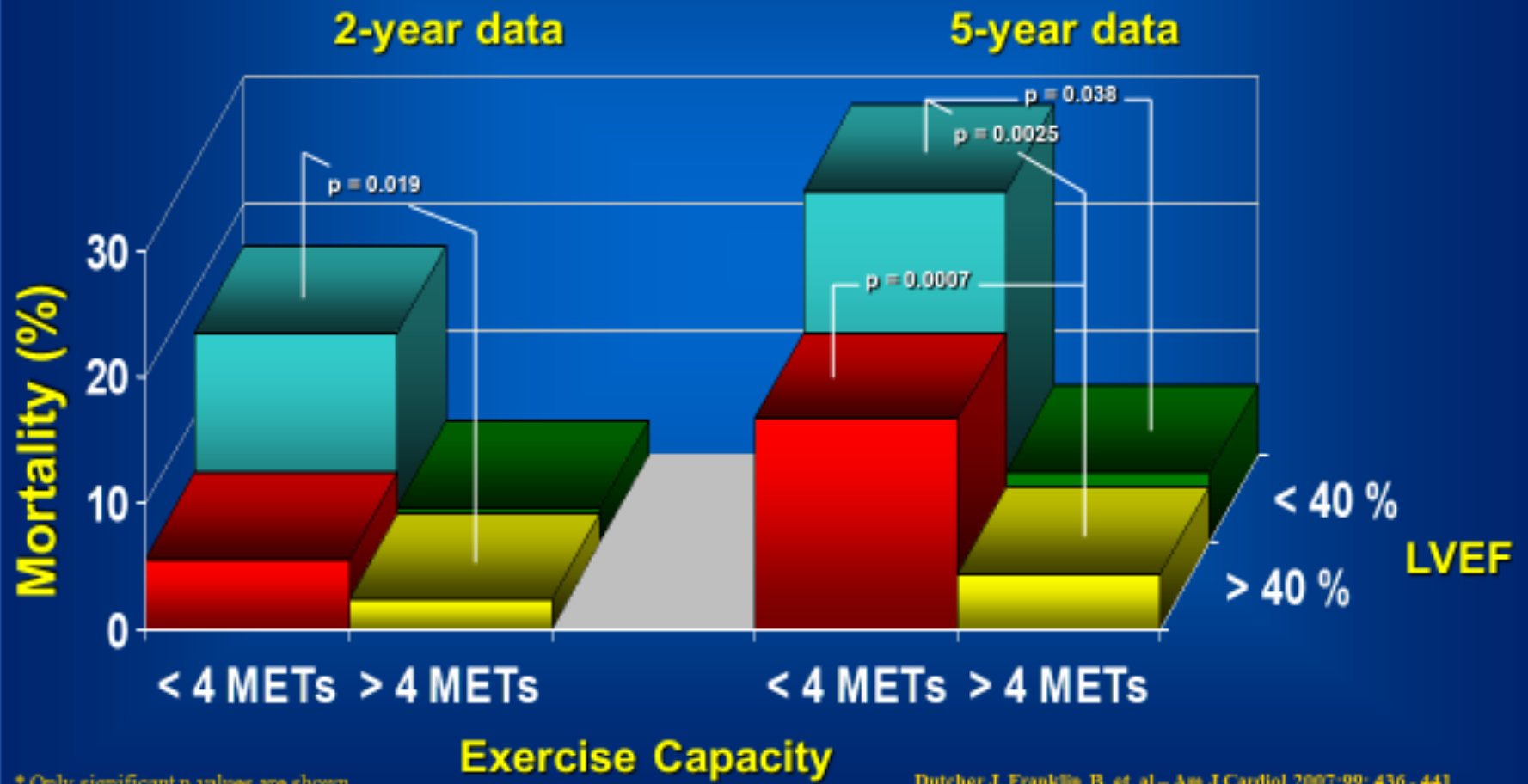
Comparison of Left Ventricular Ejection Fraction and Exercise Capacity as Predictors of Two- and Five-Year Mortality Following Acute Myocardial Infarction

Jacob R. Dutcher, MD*, Joel Kahn, MD, Cindy Grines, MD, and Barry Franklin, PhD

This study evaluated exercise capacity and left ventricular ejection fraction (LVEF) as predictors of long-term mortality in patients with ST-elevation myocardial infarction (STEMI) treated with percutaneous coronary intervention. LVEF is a well-established predictor of mortality in patients with STEMI. Exercise capacity, expressed as milliliters per kilogram per minutes or METs (1 MET = 3.5 ml/kg/min), may also serve as an independent predictor of mortality in this cohort. However, it is unclear whether these variables used together more accurately define mortality risk than either alone. In the Primary Angioplasty in Acute Myocardial Infarction-2 trial, 330 patients with long-term mortality data underwent radionuclide ventriculography at rest and cycle ergometer stress testing 6 weeks after percutaneous coronary intervention for STEMI. We used this database to evaluate the ability of LVEF at rest and exercise capacity to predict 2- and 5-year mortality. Exercise capacity <4 METs was a significant predictor of 5-year mortality (odds ratio [OR] 4.54, p = 0.0016). In contrast, decreased LVEF demonstrated a trend toward higher mortality but was not statistically significant at 2- (OR 2.22, p = 0.22) or 5-year (OR 2.04, p = 0.20) follow-up. When evaluated in combination, there was a statistically significant 2-year mortality risk for those with a decreased LVEF and decreased exercise capacity (OR 6.03, p = 0.018). Exercise capacity was a better predictor of 2- and 5-year mortality than LVEF in patients with STEMI treated with percutaneous intervention. In conclusion, when combined with LVEF, exercise capacity provides independent and additive information regarding long-term prognosis. [redacted]

Am J Cardiol 2007;99:436-441

LVEF And Exercise Capacity As Predictors Of 2- And 5-year Mortality



* Only significant p-values are shown

Dutcher J, Franklin B, et. al – Am J Cardiol 2007;99; 436.- 441 .

VIEWPOINT

Physical Activity and Structured Exercise for Patients With Stable Ischemic Heart Disease

William E. Boden, MD
Barry A. Franklin, PhD
Nanette K. Wenger, MD

EXERCISE WAS RECENTLY DESCRIBED AS "A MIRACLE drug" that can benefit every part of the body and sub-

tion. Anginal symptoms were reduced in both groups, and there was no significant difference in health status between the groups, demonstrating that optimal control of risk factors could favorably affect outcomes. Despite clinical guideline recommendations that, among patients with stable ischemic heart disease, revascularization may be deferred until the effects of optimal medical therapy and lifestyle modification have been

Each 1-MET increase in exercise capacity is associated with an 8% to 35% (average, 16%) reduction in mortality, which compares favorably with the survival benefit conferred by low-dose aspirin, statins, ß-blockers, and angiotensin-converting enzyme inhibitors after acute myocardial infarction.

tured exercise and increased physical activity for patients with stable ischemic heart disease and the need to highlight the poor prognosis associated with being in the least fit, least active cohort (bottom 20%) for the 12 to 13 million US residents who comprise this population.

One of the most puzzling aspects of the medical community's failure to recommend regular exercise for patients with stable ischemic heart disease may be the fundamental simplicity and affordability of this intervention, particularly compared with other widely accepted preventive measures. For instance, the Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation (COURAGE) trial[3] showed no difference in clinical outcomes in patients with stable ischemic heart disease (eg, death, myocardial infarction, hospitalization for unstable angina) during a mean 55-month follow-up between those who underwent percutaneous coronary intervention (PCI) and optimal medical therapy (including both risk-reducing and symptom-reducing therapies) and those treated with optimal medical therapy and lifestyle modifica-

See also p 141.

min), which is equivalent to the energy requirement for basal homeostasis. Multiples of this value are often used to quantify relative levels of energy expenditure. Each 1-MET increase in exercise capacity is associated with an 8% to 35% (median, 16%) reduction in mortality,[8] which compares favorably with the survival benefit conferred by low-dose aspirin, statins, β-blockers, and angiotensin-converting enzyme inhibitors after acute myocardial infarction.

Current guidelines recommend 30 to 60 minutes of moderate-intensity aerobic activity at least 5 days a week for patients with stable ischemic heart disease to augment peak oxygen uptake and modify cardiovascular risk factors, as well as complementary resistance training at least 2 days a week to increase weight-carrying tolerance and skeletal muscle strength.[9] Resistance training also attenuates the rate-

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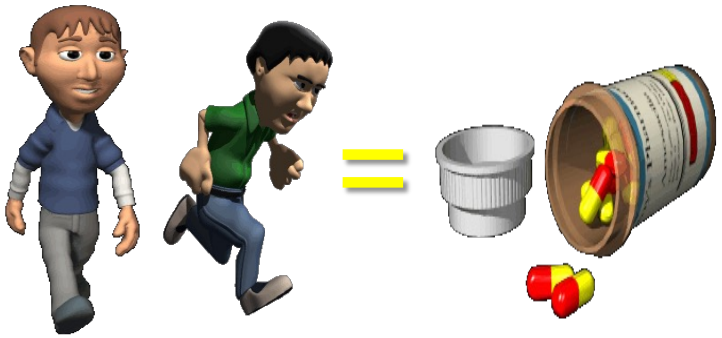
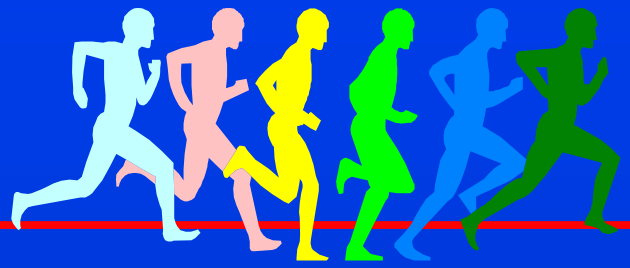
Corresponding Author: William E. Boden, MD, Department of Medicine, Samuel Stratton VA Medical Center, 113 Holland Ave, Albany, NY 12208 (william.boden@va.gov).



JAMA 2013;309(2):143

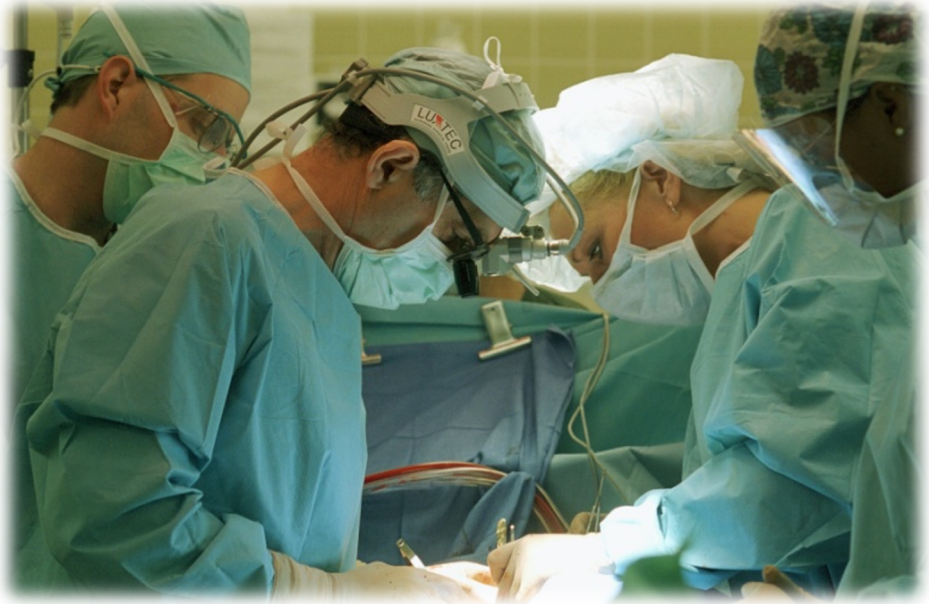
Outline



- Energy systems for exercise
- Acute cardiorespiratory responses ($\dot{V}O_2$ max)
- Metabolic equivalents (METs)
- Anaerobic (Ventilatory) Threshold
- Fitness and Mortality
- **Fitness and Surgical Outcomes**
- Clinical considerations: CPX Testing

Metabolic Equivalents as Pre-Operative Risk Assessment

- One of the strongest indicators of all-cause and cardiovascular mortality is aerobic capacity.
- Reduced cardiorespiratory fitness levels are associated with increased morbidity/mortality after:
  - Bariatric surgery
  - Liver transplantation
  - Noncardiac thoracic surgery
  - Major abdominal surgery



CHEST

Original Research

BARIATRIC SURGERY

Cardiorespiratory Fitness and Short-term Complications After Bariatric Surgery*

Peter A. McCullough, MD, MPH; Michael J. Gallagher, MD; Adam T. deJong, MA; Keisha R. Sandberg, MPH; Justin E. Trivax, MD; Daniel Alexander, DO; Gopi Kasturi, MD; Syed M. A. Jafri; Kevin R. Krause, MD; David L. Chengelis, MD; Jason Moy, MD; and Barry A. Franklin, PhD

Background: **Morbid obesity is associated with reduced functional capacity, multiple comorbidities, and higher overall mortality. The relationship between complications after bariatric surgery and preoperative cardiorespiratory fitness has not been previously studied.**

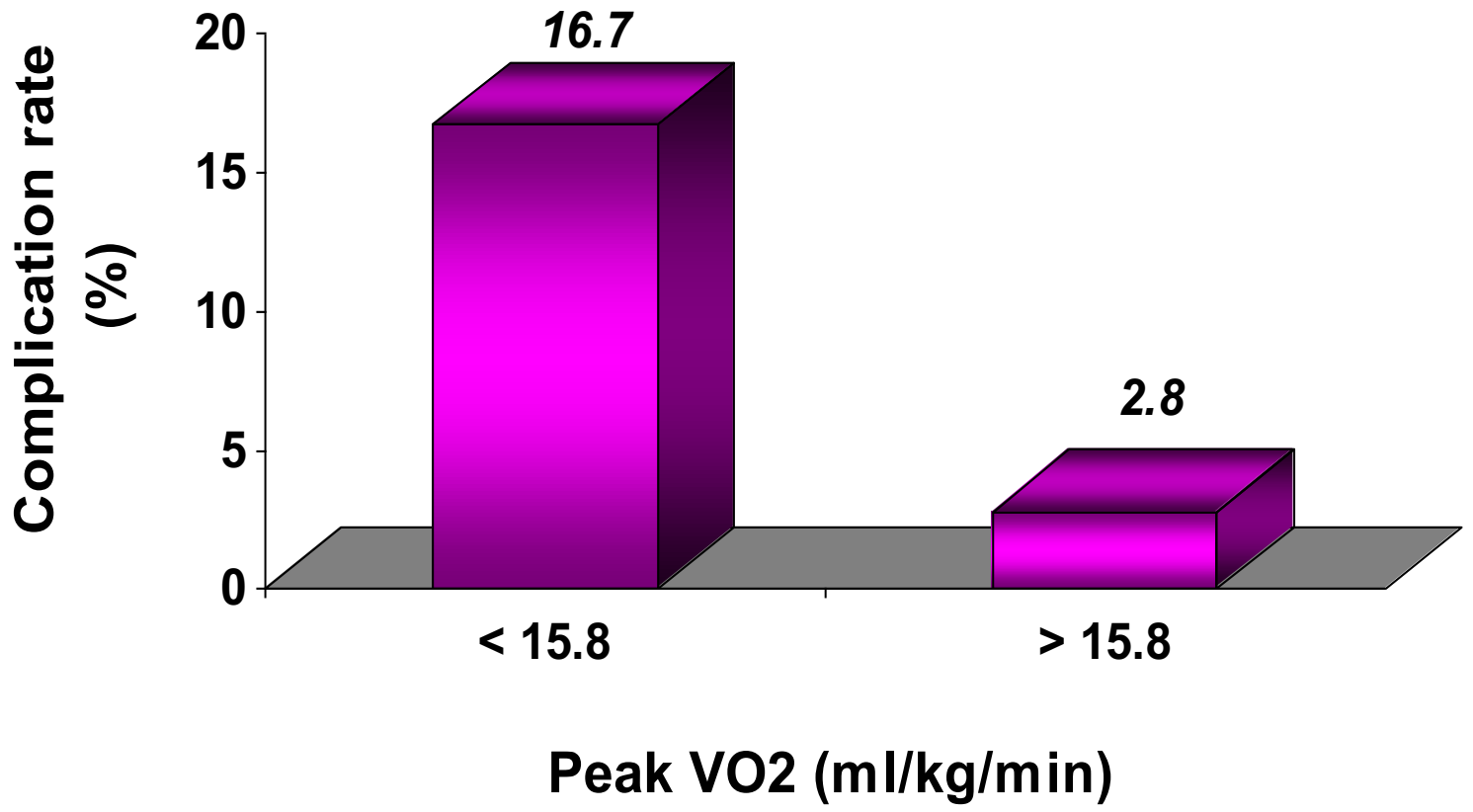
Methods: **We evaluated cardiorespiratory fitness in 109 patients with morbid obesity prior to laparoscopic Roux-en-Y gastric bypass surgery. Charts were abstracted using a case report form by reviewers blinded to the cardiorespiratory evaluation results.**

Results: **The mean age (± SD) was 46.0 ± 10.4 years, and 82 patients (75.2%) were female. The mean body mass index (BMI) was 48.7 ± 7.2 (range, 36.0 to 90.0 kg/m^2). The composite complication rate, defined as death, unstable angina, myocardial infarction, venous thromboembolism, renal failure, or stroke, occurred in 6 of 37 patients (16.6%) and 2 of 72 patients (2.8%) with peak oxygen consumption ($\dot{V}O_2$) levels < 15.8 mL/kg/min or > 15.8 mL/kg/min (lowest tertile), respectively (p = 0.02). Hospital lengths of stay and 30-day readmission rates were highest in the lowest tertile of peak $\dot{V}O_2$ (p = 0.005). There were no complications in those with BMI < 45 kg/m^2 or peak $\dot{V}O_2$ ≥ 15.8 mL/kg/min. Multivariate analysis adjusting for age and gender found peak $\dot{V}O_2$ was a significant predictor of complications: odds ratio, 1.61 (per unit decrease); 95% confidence interval, 1.19 to 2.18 (p = 0.002).**

Conclusions: **Reduced cardiorespiratory fitness levels were associated with increased, short-term complications after bariatric surgery. Cardiorespiratory fitness should be optimized prior to bariatric surgery to potentially reduce postoperative complications.**

(CHEST 2006; 130:517–525)

Cardiorespiratory Fitness and Outcomes after Bariatric Surgery



Death, unstable angina, myocardial infarction, venous thromboembolism, renal failure, or stroke Moy J, Gallagher M de Jong A, Sandberg K, Trivax J, Alexander D, Kasturi G, Jafri S, Krause K, Chengelis D, Franklin B, McCullough P. Obesity Research 2005;13, A14 (54-OR).

Patient population

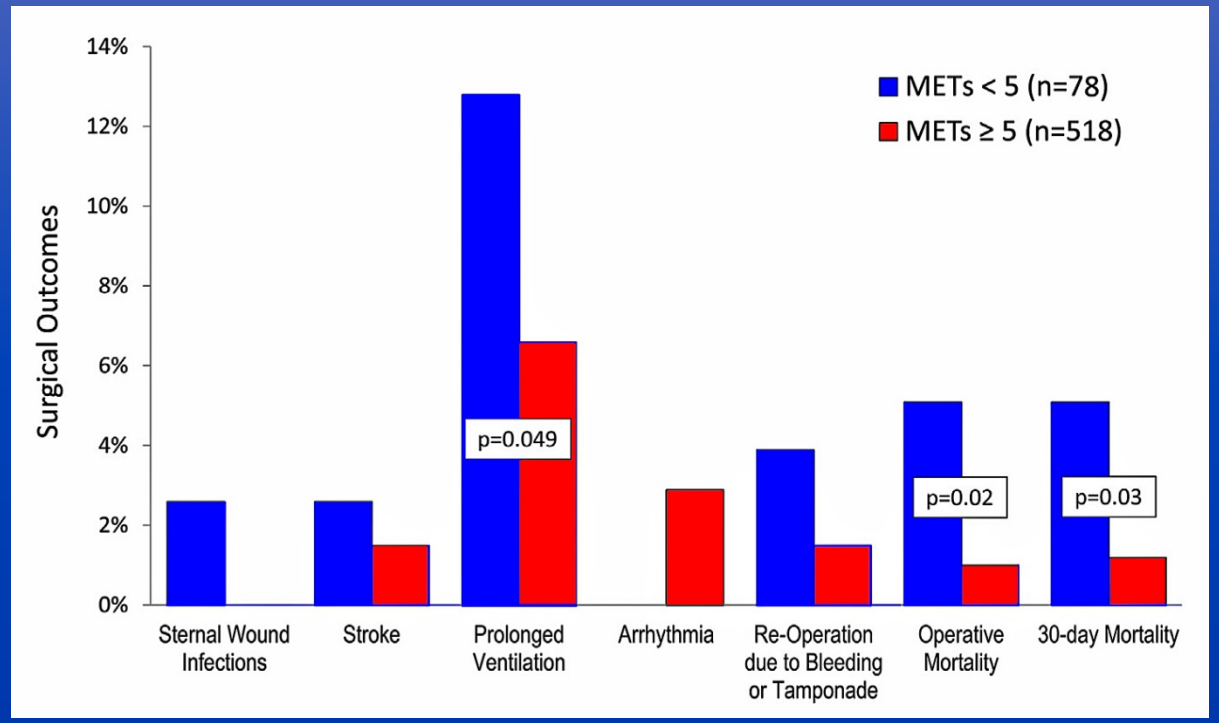
- 596 patients underwent pre-operative exercise stress testing < 90 days prior to their bypass at William Beaumont Hospitals in Royal Oak and Troy, MI campuses, from 2002-2010.

ARTICLE IN PRESS

Effect of Cardiorespiratory Fitness on Short-Term Morbidity

Specifically, low preoperative cardiorespiratory fitness (<5 METs) was associated with higher operative and 30-day mortality after CABG (p<0.05).

from January 2002 to December 2010 at Beaumont Health Systems. Electronic medical

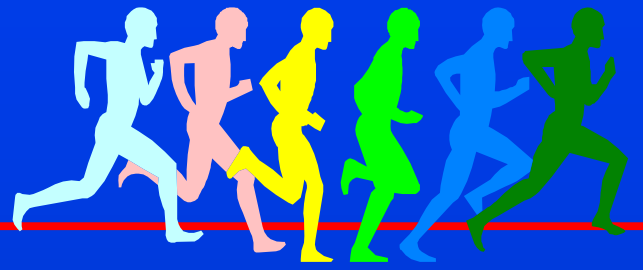


with increased morbidity after abdominal aortic aneurysm

William Beaumont Hospitals in Royal Oak and Troy, Michigan. Patients who underwent simultaneous interven-

Smith JL et al. AJC 2013; 112:1104

Outline



- Energy systems for exercise
- Acute cardiorespiratory responses ($\dot{V}O_2$ max)
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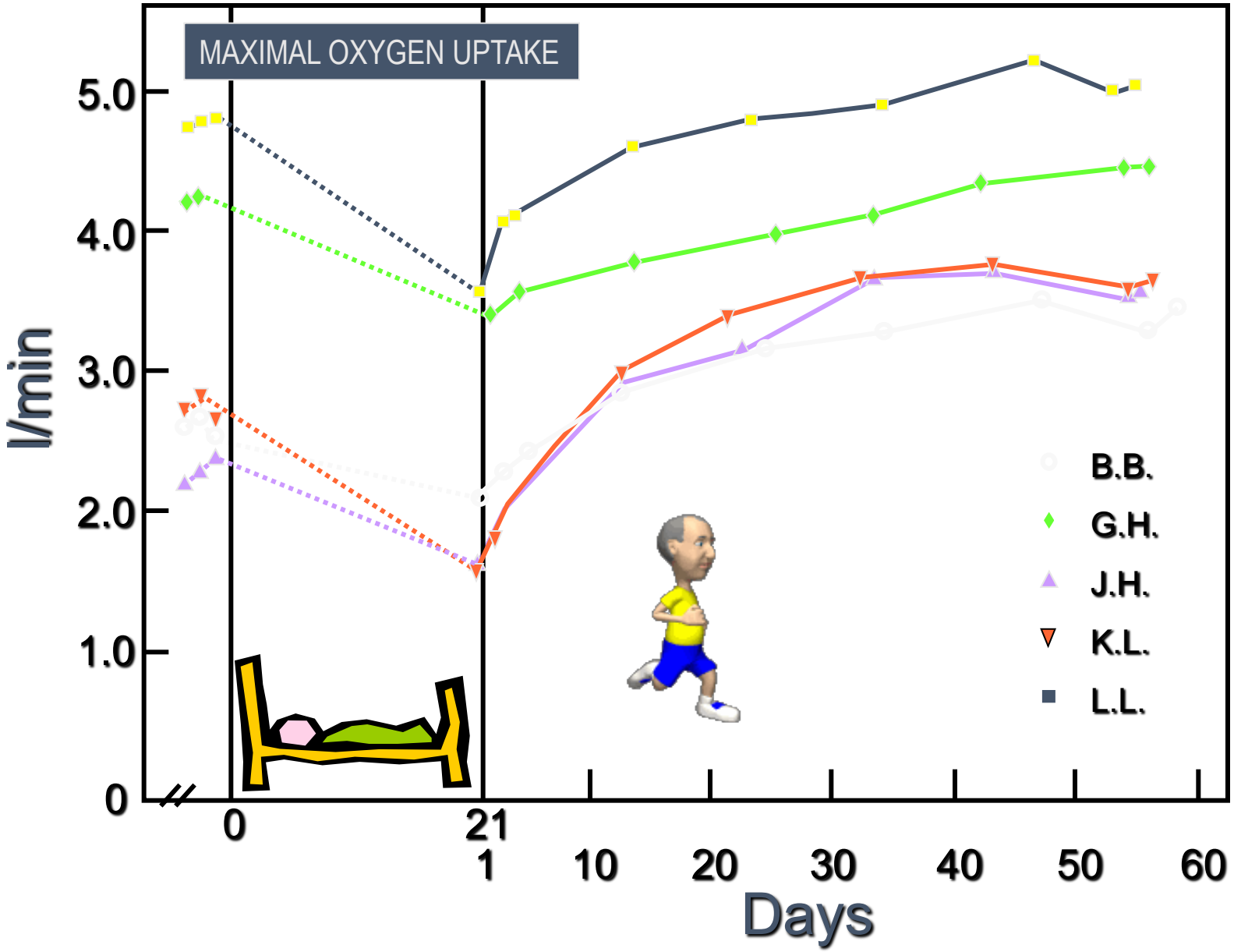
- Fitness and Mortality
- Fitness and Surgical Outcomes
- **Clinical considerations: CPX Testing**

Bed Rest Deconditioning


MAXIMAL OXYGEN UPTAKE
5.0
4.0
3.0
2.0
1.0
0
l/min
0
21
1
10
20
30
40
50
60
Days
B.B.
G.H.
J.H.
K.L.
L.L.

Three weeks of bed rest resulted in a reduction in the maximal oxygen uptake (VO_2 max) of 25 %, equivalent to the decrease in aerobic capacity that normally occurs over 30 years!

Saltin B et al. Circ 1968;38[suppl 7]:1

Mean Changes in Aerobic Capacity ($\dot{V}O_2$ 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

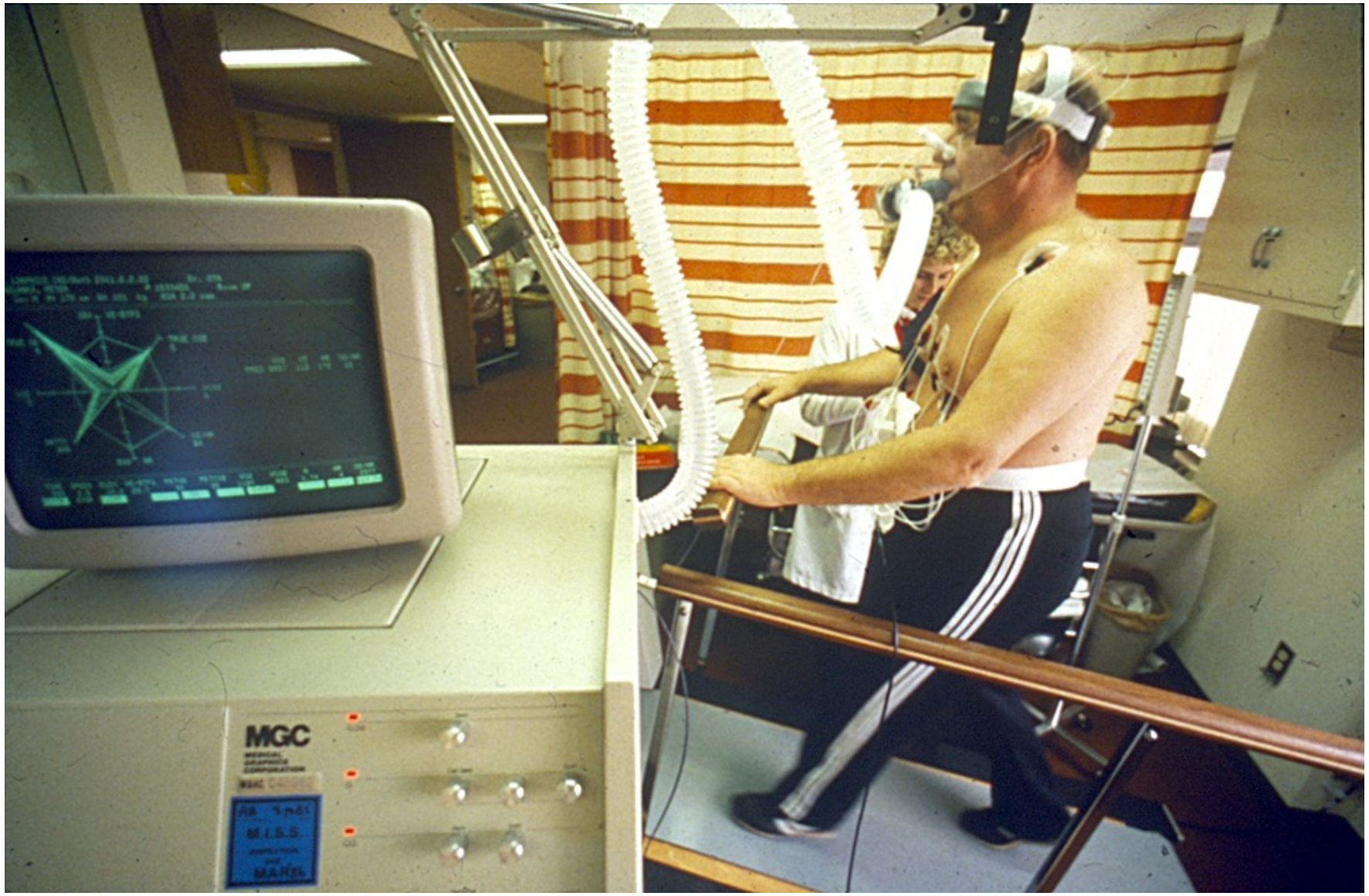
% Δ = percent change

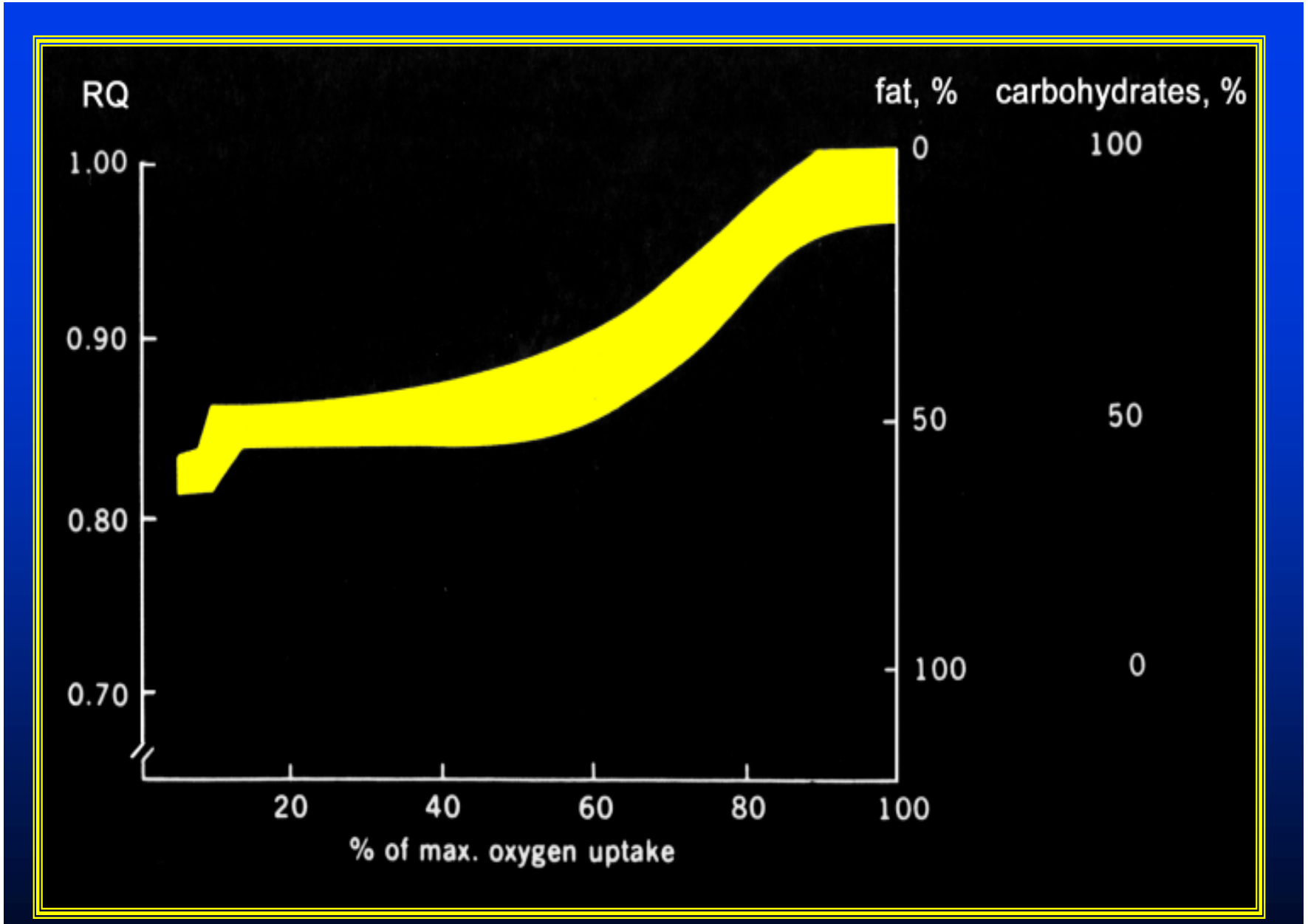
** Convertino VA et al. J Appl Physiol 1982;52:1343-1348*

It appears that deterioration of exercise performance resulting from bed rest may be largely obviated by regular exposure to orthostatic stress, such as intermittent sitting or standing during the hospital confinement period.*

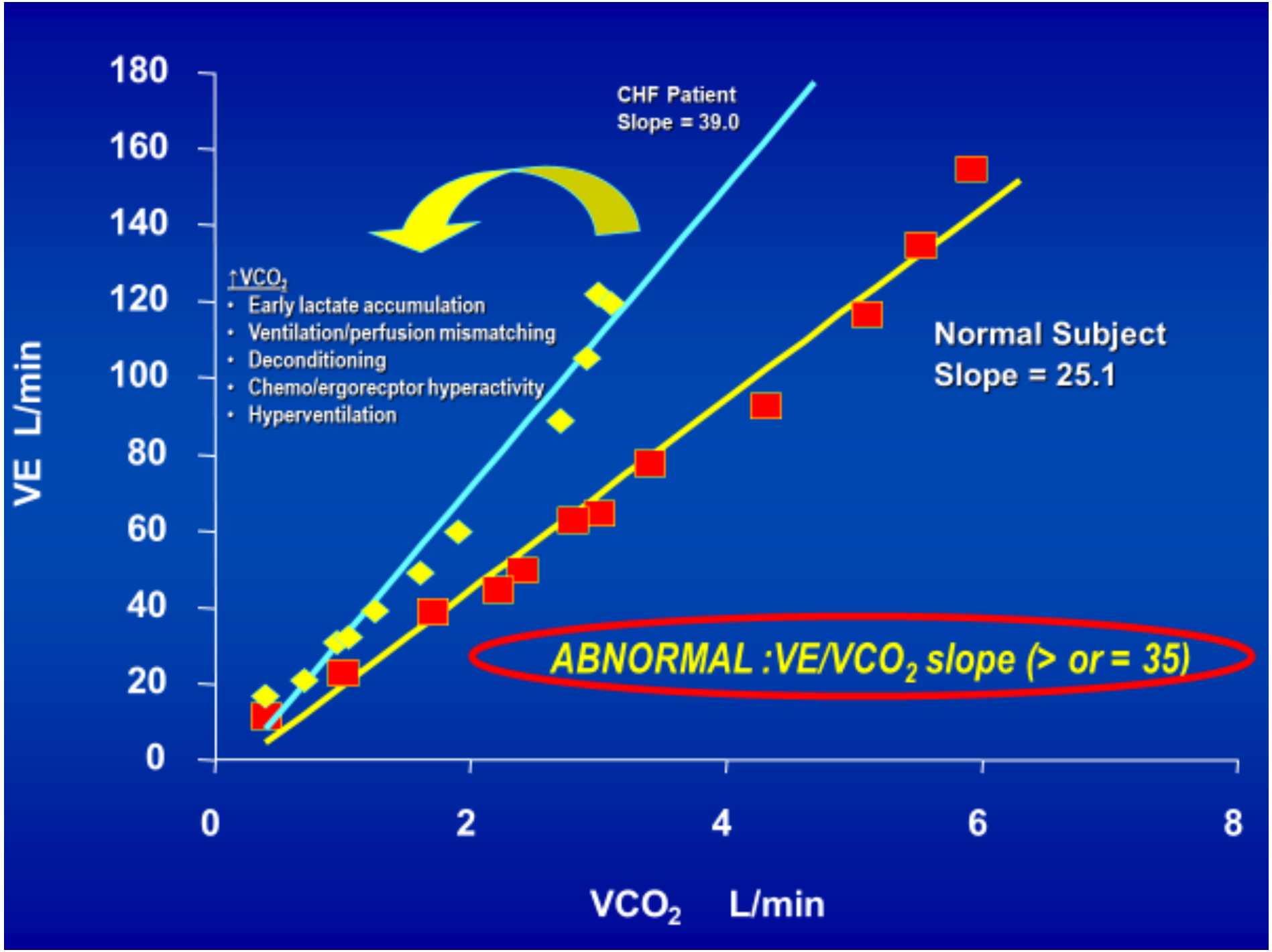
*Convertino VA et al. J. Cardiac Rehabil. 1983;3:660

Cardiopulmonary Exercise Testing (CPX)

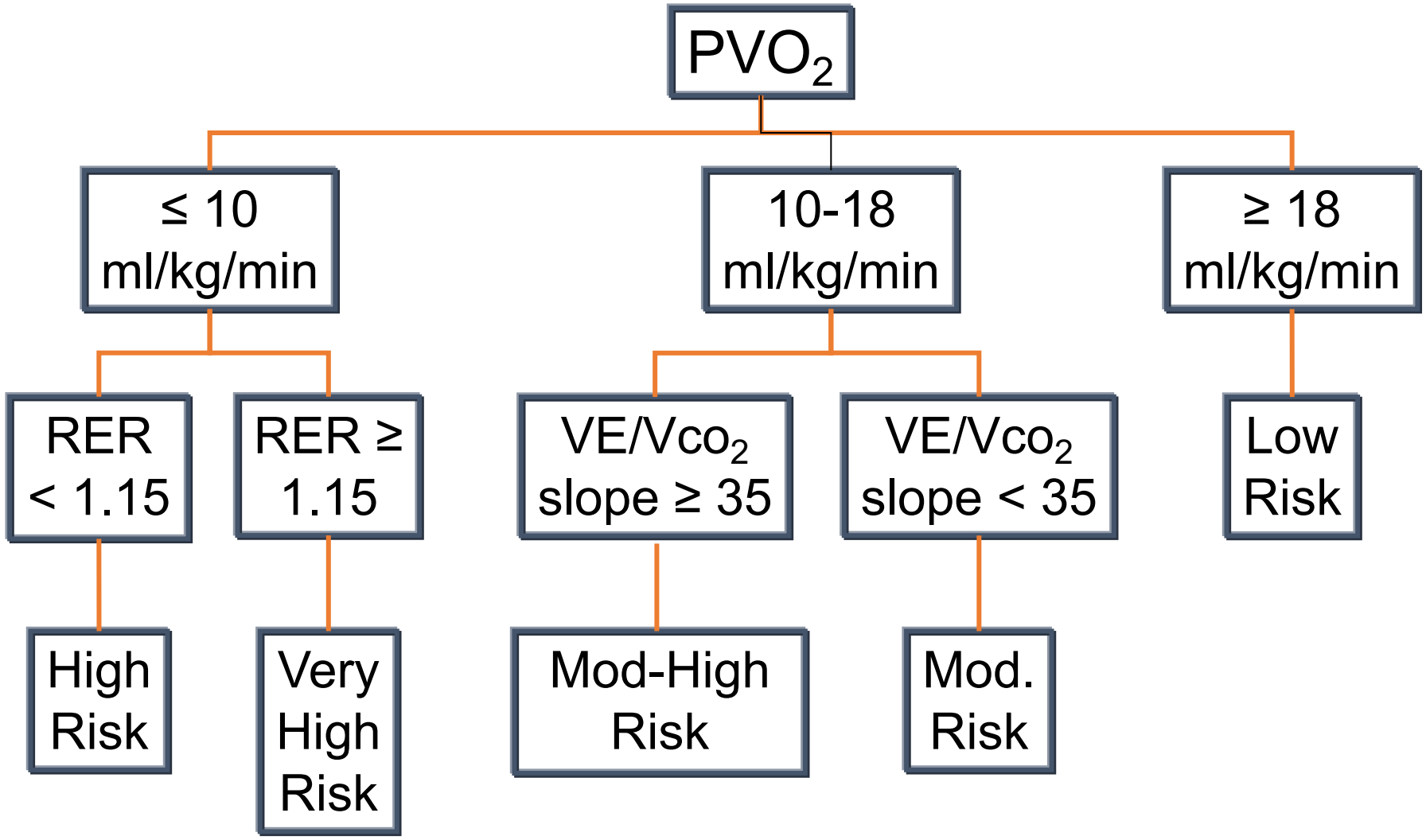




Respiratory Quotient (RQ) or RER: The ‘Lie’ Detector (Values > 1.15-1.20 = Max Effort)


180
160
140
120
100
80
60
40
20
0
VE L/min
0
2
4
6
8
VCO2 L/min
CHF Patient
Slope = 39.0
↑VCO2
• Early lactate accumulation
• Ventilation/perfusion mismatching
• Deconditioning
• Chemo/ergorecptor hyperactivity
• Hyperventilation
Normal Subject
Slope = 25.1
ABNORMAL :VE/VCO2 slope (> or = 35)

Risk Stratification Algorithm



Corra U et al. Chest 2004;126:942

Conclusions

Last 3 slides…

Exercise Physiology: Take Home Messages

- In the normal healthy individual, heart rate increases 2.7x, stroke volume increases 1.4 x, and arterial-venous oxygen difference increases 3x from rest to maximal exercise.
- The anaerobic threshold typically occurs between 50 and 80% of the maximal oxygen consumption.

Exercise Physiology: Take Home Messages

- For persons with and without heart disease, each 1 MET increase in exercise capacity is associated with ~ a 15% reduction in mortality.
- Regardless of body habitus (normal weight, overweight, obese) or risk factor profile, unfit patients are 2 to 3 times more likely to die prematurely in follow-up studies.
- The primary goal is to move clients/patients out of the 'least fit', high risk cohort (< 5 METs); on the other hand, the survival benefits of regular exercise appear to plateau beyond a fitness level > 10 METs.


FINISH