Why should we be concerned regarding Risk of Exercise?

• Physical activity pattern during adulthood/level of fitness are more strongly associated with Heart Disease and all-cause morbidity/mortality than traditional risk markers
• Small investments in activity yield large health outcome benefits
• Higher level of fitness/physical activity are associated with lower health care costs
• Few Americans are physically active enough to gain health benefits
• Not enough is being done to incorporate physical activity into the health care paradigm
Preventable Causes of Death in the US, 1990 vs. 2000

1990:
- Tobacco
- Poor diet/Physical inactivity
- Alcohol consumption
- Microbial agents
- Toxic agents
- Firearms
- Sexual behavior
- Motor vehicles
- Illicit drug use

2000:
- Tobacco
- Poor diet/Physical inactivity
- Alcohol consumption
- Microbial agents (e.g., influenza, pneumonia)
- Toxic agents (e.g., pollutants, asbestos)
- Motor vehicles
- Firearms
- Sexual behavior
- Illicit drug use

Percentage (of all deaths)

References:
* McGinnis JM, Foege WH. JAMA 1993;270(18):2207-12
Where is the Evidence?

♥ Retrospective Epidemiological Studies
♥ Bus drivers, Harvard alumni, SF Longshoremen, ...

♥ Prevalence Epidemiological Studies
♥ Cross-sectional
♥ Bias problem: the sick population is generally more inactive

♥ Longitudinal (Prospective)
♥ Observational (Framingham, Veterans Affairs, etc.) vs. Interventional

♥ Meta-analyses of best Epidemiology
Epi Methods for Quantifying the Exercise Stimulus:

- Physical Activity: job title, questionnaires ... Calories or kiloponds expended ... 
- Physical Fitness: Exercise test result ... METs
Williams, Meta-analysis, MSSE 2001:754
8 fitness cohorts (317,908 person-yrs fu)
30 activity cohorts (>2 million person-yrs fu)
Strength, independence and primacy of the relationship between Fitness and Death
Consistency (Biological Plausibility)

- Animal Models demonstrate that Physical Activity induces changes on both the Heart and the Periphery
  - Wild vs. Domestic animals
  - Increased fibrillatory threshold in dogs
  - Increased coronary flow in pigs
  - Smaller infarcts in rats
The Genetic Factor

MICHAEL
PHELPS
Effects of Chronic Exercise on Animals

- Age-dependent myocardial hypertrophy
- Myocardial histological changes
- Proportional increase in coronary artery size
- Coronary collateral circulation
- Improved cardiac mechanical and metabolic performance
- Favorable changes in skeletal muscle mitochondria and respiratory enzymes
- Myocardial mitochondria and enzyme changes
- Atherosclerosis delay and regression
- Serum cholesterol reduction
Effects of Regular Dynamic Exercise on Normal Hearts

❤ Morphologic changes
   ❤ Larger hearts (cross-sectional and longitudinal)
     ❤ Echo exams show an average increase in LV mass
   ❤ Coronary artery size (parallels mass)

❤ Hemodynamic changes
   ❤ Lower heart rate, systolic BP
   ❤ Greater cardiac output, VO2, exercise capacity, coronary reserve
   ❤ Better cardiac function
     – Faster recovery (including heart rate)

❤ Endothelial Function Protected
Key points:

- <30% of Americans meet the minimal recommendations for physical activity
- *More than one third of Americans report getting no physical activity at all*
- The prevalence of obesity has more than doubled since 1990
- Deaths due to physical inactivity/poor diet may soon exceed tobacco use as the leading cause of preventable death (CDC, 2004)
Incremental Survival Benefit per MET

- 1 MET = resting metabolic rate (3.5 ml O₂/kg/min)
- Exercise capacity commonly expressed in multiples of the resting metabolic rate (adjusted for age and training)
- 1 MET ≈ 2.5% grade on the treadmill at walking speed, 25 watts on cycle ergometer
- 5 METs is upper limit of ADLs
- <5 METs = high risk; >10 METs = low risk
Energy expenditure expressed in kcals

- 1 kcal (calorie) = energy required to increase 1 kg water 1°C
- 30 minutes of walking ≈ 150 kcals
- CDC/ACSM/Surgeon General's Report recommendation is roughly 1,000 kcals/week
- 30 minutes of brisk walking burns the calories in 1 plain donut (185 kcals), 1 hour for a glazed donut
2000 kcal/week:

- Moderate activity (walking) 1 hr/day
- Higher intensity activity, 1 hr, 3-4 times/week
- 6,000 steps/day (pedometer)
- 20 to 25 MET-hours (5 MET activity, 1 hour, 5 times/week)
Roughly 10 million young competitive athletes each year in the US

OVER 200 YOUNG ATHLETES DIE EVERY YEAR IN THE US
Risks of Exercise

Sudden Death

- Exercise-related incidence per year:
  - 1 out of 250,000 children and young adults
  - 1 out of 50,000 adults in the general population
  - 1 out of 200,000 high school/college athletes
  - 1 out of 80,000 to 160,000 man-hours in populations with CAD
- Patients with heart disease are at increased risk
- Regular exercise decreases risk
  - (Siscovick, 1984)
  - (Mittleman, NEJM 1993)
Sudden Death

- > 40 years of age
  - Primarily due to CAD
- < 40 years of age
  - Most common causes: Hypertrophic Cardiomyopathy (approx. 50%), Marfan's Syndrome, coronary artery anomalies
    - Prevalence of HCM in young people is approximately 0.1%
  - Less common causes: viral myocarditis, RV dysplasia, mitral valve prolapse, aortic valve stenosis....

Note: Sudden Death is extremely rare in athletes; for young athletes it is usually due to congenital problems
Sudden Death in Famous Athletes

- Jim Fixx
  - >40, due to CAD
- Reggie Lewis
  - Nonspecific Cardiomyopathy
  - Dilated Cardiomyopathy
- Hank Gathers
  - Congenital Anomaly
- Pete Maravich
  - Congenital Anomaly
- Flo Hyman
  - Dissecting Aortic Aneurysm (Marfan's)

The Maryland Basketball Team inspired NIH research of SCD in athletes--- Registry is very difficult
Screening for Sports Participation

- History of chest pain or syncope -- best signs
  - Syncope during as opposed to post-exercise

- Hypertrophic Cardiomyopathy is very difficult to discern from "athlete's heart"
  - Athletic Heart Syndrome includes many abnormalities that are not dangerous
    ✷ Gallop sounds, increased heart size/movements

 официальнɛ History - current best genetic test

Bethesda Guidelines; European Guidelines … the ECG controversy
ECG Added to Stanford Athletes Annual Pre-participation Exam 2007
Center for Inherited CV Diseases/HCM Clinic


T wave Inversion greater than 2 mm in 3 leads other than V1 and AVR in 21 yo Stanford Female athlete

Pelliccia, A, et al. Outcomes in Athletes with Marked ECG Repolarization Abnormalities. NEJM 2008:358:152-161. Positive predictive value of 36% for this ECG abnormality that occurs in 1% of athletes (immediate diagnosis in 39 and 5 in follow up [out of 129], mostly cardiomyopathies).
T wave Inversion greater than 2 mm in 3 leads other than V1 and AVR in 33 yo 6ft 205 lb 49er
Computer ECG in Stanford Athletes
AHA 12 Point for CV Screening in PPE

Medical history*

Personal history

1. Exertional chest pain/discomfort
2. Unexplained syncope/near syncope†
3. Excessive exertional and unexplained dyspnea/fatigue associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure

Family history

6. Premature death (sudden and unexpected or otherwise) before 50 y of age resulting from heart disease in ≥1 relative
7. Disability from heart disease in a close relative <50 y of age
8. Specific knowledge of certain cardiac conditions in family members: hypertrophic or dilated cardiomyopathy, long-QT syndrome or other ion channelopathies, Marfan syndrome, or clinically important arrhythmias

Physical examination

9. Heart murmur‡
10. Femoral pulses to exclude aortic coarctation
11. Physical stigmata of Marfan syndrome
12. Brachial artery blood pressure (sitting position)§
• Few Americans are physically active enough to gain health benefits
  \( \approx 30\% \) meet the minimal recommendations for activity

• Sedentary lifestyle is a major health problem; increasing physical activity should be a standard part of medical management
  Exercise is discussed between <10 and \( \approx 30\% \) of health care encounters

• Moderate activity associated with 20-40\% improvements in health outcomes
  Physical fitness/physical activity pattern are more powerful markers of risk than commonly appreciated
Summary (2 of 3)

The least fit stand to benefit the most from improving fitness

*As much as half the benefit occurs between the least fit and the next fit category*

- In patients with existing CV disease, rehabilitation programs reduce mortality
  
  *\approx 20 \text{ to } 30 \text{ reductions in CV and all-cause mortality}*

- Incorporation of modest amounts of physical activity results in lower health care costs
  
  *\approx \$1 \text{ per kcal energy expenditure/week}
Historically - Iatrogenic but situation has changed

- Decreased need with shortened hospitalizations
- Realization that activity as important as aerobic fitness
- No definitive randomized trial tho meta-analyses suggestive (but typically so)
- Competition from improved technologies both medical (Statins, troponin, ACS, change in MI definition); PCIs and surgery.
Abdominal Aortic Aneurysm (AAA) Disease: Mechanism, Stratification, and Treatment

Program Director: Ronald L. Dalman MD; Program Co-Director: Lawrence Leung, MD
Project and Core Leaders: Andrew Connolly, MD, PhD, John Cooke, MD, PhD, Michael McConnell, MD,
Jonathan Myers, PhD, Charles Taylor, PhD, Philip Tsao, PhD
Stanford University Schools of Medicine and Engineering

This Specialized Centers for Clinically Oriented Research (SCCOR) P50 Program will identify novel biomarkers to monitor small AAA disease and test the ability of exercise therapy to modify disease progression.

SIGNATURE PROTEIN PROFILES TO IDENTIFY AAAs (Project II)

Background: Serum protein profiling may enable early diagnosis of AAAs, predict expansion, and monitor response to novel medical therapies. In this Project, transcriptional profiling of human AAA tissue, database mining for patterns of protein expression, and serum multikinase assessment of experimental models will be used to develop proteomic profiles of AAA disease.

Figure 1. Heatmap of relative serum protein levels that coincide with progression of experimental atherosclerosis. Real-time PCR confirms vascular production of many of these markers (red indicates increased expression).

Biomarker Profiles of AAA

ApoE(−/−) + AngII
ApoE (−/−) + Saline

Figure 2. A proteoglycan deficient mouse model of AAA disease: Supraphysiological AAA present after 28 day infusion with Angiotensin II (1000ng/kg/min) via an osmotic pump (left, arrow), compared to control mouse aorta (right) after normal saline infusion.

REST AND EXERCISE HEMODYNAMICS IN AAA PROGRESSION (Project III)

Background: Hemodynamic conditions in the infrarenal aorta play an important role in the pathophysiology of AAA disease. Our goal is to quantify abdominal aortic blood flow at rest and during dynamic exercise, and develop and validate computational methods to model blood flow, pressure, and wall motion in patient-specific reproductions of the abdominal aorta using magnetic resonance angiography (MRA).

Figure 3. MR imaging of the aorta during exercise.

Figure 4. An MR angiogram (left) of a patient with a small AAA is used to construct a solid model of the lumen (right) for analyses.

EVALUATION OF EXERCISE THERAPY FOR SMALL AAA (Project IV)

Background: Substantial evidence links sedentary existence and resulting pro-inflammatory aortic conditions to the pathogenesis of AAA disease. Our goal is to test the ability of lower extremity exercise to reduce AAA risk, limit small aneurysm progression, and modify biologic markers of disease.

Recruitment Goals:
- 1,400 small AAA patients will complete exercise history and health history questionnaires, and undergo a blood draw and abdominal ultrasound to correlate risk factors with AAA disease status.
- 1,000 patients with previously defined exercise capacity will undergo aortic imaging to correlate fitness status and exercise capacity with aortic diameter.
- 340 patients with small AAAs will be randomized to exercise or usual activity, and followed over three years with serial imaging to test the ability of exercise therapy to modify disease progression and candidate biomarkers of AAA disease.

Recruitment Locations:
- Stanford University Medical Center
- Palo Alto Veteran’s Administration Health Care System
- Kaiser Permanente of Northern California

SUPPORTING INVESTIGATORS

Francis Blankenberg, MD
Chris Cheng, PhD
Mary Druaney Blomme, Ph.D
Alberto Figueroa, Ph.D
Victor Froechlicher, MD
E. John Harris, Jr, MD
Trevor Hasting, Ph.D
Robert Herffsens, MD
Bradley Hill, MD
Brooke Jeffrey, MD
Timothy Myles, PhD
Balasubramanian Narasimhan, PhD
Richard Olshen, Ph.D
John Paul, Ph.D
Tom Quennerhaus, MD
George Segall, MD
Christopher K. Zarins, MD

CONTACT INFORMATION

For participation in AAA disease studies, please contact Julie White by phone at (650) 498-6039 or by email at jlwithe@stanford.edu.
http://aaastop.stanford.edu

FUNDING SOURCE

This work was funded by the National Heart, Lung, and Blood Institute grant number R01 HL083800.

next

PAUSE = PCI Alternative Using Sustained Exercise
Pre-Sport Physicals

Who: Any and All Athletes
      (High School, Pop Warner, Little League)

When: Thursday May 20th 2010
      Tuesday May 25th 2010
      6:00 - 7:30 pm

Where: BAK Physical Therapy
       705 Oak Grove Avenue
       Menlo Park, CA 94025

WANTED:

MDs to volunteer to help out during our physicals! Please contact Stevan Allen for more info Stevan.Allen@physiocorp.com

BAK Physiotherapy Associates

For more information, call: 650.363.5674
The End
The Stanford/Palo Alto VA Clinical Exercise Physiology Consortium

Euan Ashley MD, PhD,  Frederick E. Dewey, Jonathan Myers PhD, Victor F. Froelicher MD

Stanford University, Palo Alto, CA, Palo Alto VA Health Care System, Palo Alto, CA

The clinical exercise physiology consortium is located at five sites, three at the Palo Alto VA Medical Center (PAVAMC) and two at the Stanford University Campus:
1) Cardiology Department at the VA Hospital (Bldg 101);
2) Exercise Training Unit (PAVAMC, Bldg 51);
3) Spinal Cord Rehabilitation Unit (PAVAMC, Bldg 6);
4) Stanford Sports Medicine Human Performance Laboratory (Arrellaga Recreation Bldg, 531 Galvez Ave, Stanford Campus),
5) Stanford Medical Center Exercise Testing Laboratory and Cardiomyopathy Clinic.

• The Palo Alto VA Health Care System includes the Medical Center in Palo Alto (where three of our sites are located) and satellite clinics in Menlo Park, San Jose, Livermore, Monterey, Stockton, and Modesto, California. The Medical Center is a large combined medical and surgical, inpatient and outpatient VA facility. We are mainly located in the Cardiology Division on the second floor of Building 101. We have a large room with 8 computers used by researchers and a combined exercise testing room divided by a movable partition with complete labs, one for clinical and the other for research testing. Our main offices are located there along with most of our supporting staff. Computer networking is readily available throughout the health care facility with direct access to VA computerized patient record data bases. The Cardiology Division includes rooms dedicated to Echocardiography, Cardiac Catheterization and ECG services. A regular educational lecture series is provided for a broad range of internal medicine and cardiology topics for Stanford students, residents and fellows who rotate through Cardiology.

• The Exercise Training Unit is a large room with multiple exercise training devices and ECG monitoring for up to 8 patients. It is on the first floor of Bldg 51 which is in the south corner of the VA grounds with large windows and ready access to grassy areas and walking paths.

• The Spinal Cord injury Research Laboratory is located in between our two facilities described above and is the site for ongoing VA Rehabilitation Research and Development funded projects involving exercise, risk reduction, and cardiovascular health in spinal cord injury.

• The Stanford Sports Medicine Human Performance Laboratory performs cardiovascular testing for evaluation of Stanford athletes, alumni and community, as well as research in human performance. It is associated with the Stanford Sports Medicine Clinic in the same building. Drs. Myers and Froelicher have provided Cardiology and Exercise Physiology consultation for over 10 years and are part of the Sports Medicine faculty. The lab has been recently opened and contains the latest equipment for the evaluation of athletes including portable VO2 analysis, GPS recorders, Holter monitors and a portable cardiovascular ultrasound device.

• Stanford Medical Center Exercise Laboratory is located in the Stanford Medical Center, a world renowned tertiary care center. Stanford witnessed the birth of heart and lung transplantation and maintains a busy advanced heart failure service. As such, the exercise testing laboratory specializes in cardiopulmonary exercise testing for transplant evaluation and on going management of patients with cardiomyopathy and heart failure, as well as pulmonary hypertension. Stress echocardiography is combined with expired gas analysis to provide sophisticated integrated measurements in certain groups such as those with hypertrophic cardiomyopathy or those with ischemic cardiomyopathy. Serving multiple scientific studies as well as the clinical population of Stanford and nearby centers, the lab interacts closely with other exercise physiology labs in the consortium.

Key Researchers

Dr Euan Ashley is Assistant Professor of Medicine at Stanford University and directs the Hypertrophic Cardiomyopathy Clinic. He graduated in Physiology and Medicine from the University of Glasgow, Scotland, before completing his residency at the John Radcliffe Hospital in Oxford, England. He was awarded the Wellcome trust award to join the clinician-scientist PhD program in Molecular Cardiology at the University of Oxford. Recent publications have dealt with apelin-APJ signaling in heart failure and the ACE gene impact on endurance sports cardiac alterations

Rick Dewey currently is a third year Medical Student at Stanford who was a second place finisher in the Physiology, Pathology, and Pharmacology division of the Young Investigator Awards at the ACC Scientific Sessions in 2006. Recent investigations have centered around the clinical associations and prognostic applications of heart rate patterns and ventricular ectopy associated with exercise. He is also working with Dr. Ashley towards more accurate clinical recognition of Hypertrophic Cardiomyopathy.

Dr. Jonathan Myers’ research focus has been in the areas of exercise testing, training, and epidemiology in patients with coronary artery disease and chronic heart failure. He has extensive experience in the measurement, evaluation, and interpretation of cardiopulmonary exercise test responses, and the application of epidemiology to cardiovascular disease. Dr Myers is an Associate Clinical Professor of Medicine at Stanford and a Career Scientist Award Recipient at the VA Palo Alto HCS.

Dr. Victor Froelicher - After fellowship at the University of Alabama at Birmingham, at the U.S. Air Force School of Aerospace Medicine, he published numerous works related to exercise physiology and early screening for coronary artery disease. While at the UCSD, he was the PI of a VA cooperative multicenter study of exercise testing and angiography called QUEXTA.

The Exercise Consortium current projects include:
1. Providing the exercise testing and training components of the NIH/HLBI study of small aortic aneurysms,
2. Gathering a digital ECG data base on athletes and veterans,
3. Development of algorithms for heart rate variability analysis in response to exercise for predicting prognosis and detecting over training,
4. Demonstration of whether the regression of cardiac hypertrophy during detraining can be used distinguish between hypertrophic cardiomyopathy and the normal response to aerobic training,
5. Follow-up studies of Expired Gas analysis and CHF,
6. Application of the exercise test for the epidemiologic study of patients with cardiovascular disease (VETS or Veterans Exercise Testing Study) with over 10,000 patients enrolled.
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The Exercise Consortium current projects include:
- Providing the exercise testing and training components of the NHLBI study of small aortic aneurysms
- Development of algorithms for heart rate variability analysis in response to exercise for predicting prognosis and detecting over training
- Evaluation of the time course of regression of athletically induced cardiac hypertrophy to aid in the diagnosis of hypertrophic cardiomyopathy
- Exercise physiology of adventure races including the cardiac response to prolonged exercise
- Prognostic indices in heart failure - a combined database of gas analysis exercise tests from Stanford and VA heart failure patients
- Application of the exercise test for the epidemiologic study of patients with cardiovascular disease (VETS or Veterans Exercise Testing Study) with over 10,000 patients enrolled.

Key Researchers:
Dr. Euan Ashley is Assistant Professor of Medicine at Stanford University and directs the Stanford Hypertrophic Cardiomyopathy Center. He graduated in Physiology and Medicine from the University of Glasgow, Scotland, before completing residency and a clinical-scientist PhD program in Molecular Cardiology at the University of Oxford. His interests are in the genetics of cardiomyopathy and recent publications have dealt with spadec-AP signaling in heart failure and the effect of ACE genotype of the cardiovascular response to prolonged exercise.

Rick Dewey is currently a third-year Medical Student at Stanford who was a second-place finisher in the Physiology, Pathology, and Pharmacology division of the Young Investigator Awards at the ACC Scientific Sessions in 2006. Recent investigations have centered around the clinical associations and prospective applications of heart rate patterns and ventricular ectopy associated with exercise. He is also working with Dr. Ashley towards more accurate clinical recognition of Hypertrophic Cardiomyopathy.

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Dr. Victor Froelicher - After fellowship at the University of Alabama at Birmingham, at the U.S. Air Force School of Aerospace Medicine, he published numerous works related to exercise physiology and early screening for coronary artery disease. While in the USAF, he was the chief of a NIH randomized trial of cardiac rehabilitation (PER FEVT). Later he was the PI of a VA cooperative multicenter study of exercise testing and angiography called QUESTA. He has authored over 400 publications.
Hazards of Exercise

- **Gynecologic**—delayed menarche, secondary amenorrhea, oligomenorrhea
- **Endocrinologic**—hypoglycemic (for diabetics)
- **Musculoskeletal**—acute muscle injury, exertional rhabdomyolysis, strains and sprains, arthropathies, fractures
- **Renal**—hematuria, proteinuria
- **Hematologic**—anemia, GI blood loss
- **Thermal**—heat cramps, heat exhaustion, heatstroke, frostbite, hypothermia
Outline

- Introduction to CV Disease
- Cardiac Causes of Death
- Sports and Sudden Death
Cardiac Causes of Death during Exercise

Heart Coronary Artery Disease = ischemia
- due to atherosclerosis, congenital anomalies
  - temporary - Chest pain
  - permanent - MI and possible death
- problem: exercise increases myocardial oxygen requirements

Heart Muscle Disease
- LV cardiomyopathy
  - hypertrophic [non-obstructive (generalized or localized) and obstructive (localized to septum)]
  - dilated due to damage (viral, CAD, alcohol)
- RV dysplasia
- Arrhythmias
Valvular disease = insufficiency/obstruction
- problem: exercise requires an increase in cardiac output

Congenital vascular disorders

Conduction system abnormalities
- problem: electrical system fails

Arrhythmias
- problem: secondary and primary or congenital