Sudden Cardiac Arrest in Athletes and Commotio Cordis

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Sudden Cardiac Death in Athlete

- Historical Perspective
- Epidemiology
- Screening
- Structural Heart Disease
- Syncope
- Performance Enhancing Substances
- AEDs
- Commotio Cordis
- Conclusions
Sudden Cardiac Death in the Athlete
Historical Perspective

• Pheidippides

Ran from Marathon to Athens to announce victory over the Persians.

After running 24 miles (40 km) he dropped dead.
To an athlete dying young

Towns man of a stiller town.

A. E. Hausman 1895
To an Athlete Dying Young

The time you won your town the race
We chaired you through the marketplace
Man and boy stood cheering by
And home we brought you shoulder high

To-day the road all runners come
Shoulder high we bring you home
And set you on your threshold down

Townsman of a stiller town

A. E. Hausman 1895
Sudden Cardiac Death Athletes

Epidemiology

High school and college women 1/769,000

High school and college men 1/133,000
  High school men 0.66/100,000
  College men 1.45/100,000

Males > age 40 1/15,000
  Risk of SCD increases 8-56X with exercise

Sudden Cardiac Death in the Athlete
Deaths by Sport

Sudden Cardiac Death in the Athlete
Cardiac Condition and Race

Sudden Cardiac Death in the Athlete
Preparticipation Screening

AHA Consensus Panel Recommendations for
Preparticipation Athletic Screening

Family History
1. Premature sudden cardiac death
2. Heart disease in surviving relatives less than 50 years old

Personal History
3. Heart murmur
4. Systemic hypertension
5. Fatigue
6. Syncope/near-syncope
7. Excessive/unexplained exertional dyspnea
8. Exertional chest pain

Physical Examination
9. Heart murmur (supine/standing*)
10. Femoral arterial pulses (to exclude coarctation of aorta)
11. Stigmata of Marfan syndrome
12. Brachial blood pressure measurement (sitting)

Maron BJ et al. Circulation; Estes et al. JCE 2001
Sudden Death in the Athlete  
Preparticipation Screening

- No data that screening as recommended is sensitive, specific, cost effective, or useful for reducing the risk of SCD in the athlete
- One study concluded that history and exam would have detected only 3% of cardiac abnormalities that ultimately resulted in sudden death
- Multiple studies have shown the low yield of screening EKG and echocardiograms
- Standardized national Italian screening program
  - History, physical examination, EKG, and ETT
  - Selective use of echo and HM
  - This has reduced the frequency of SCD

Flow diagram illustrating the proposed screening protocol for young competitive athletes

1. young competitive athletes
   - family and personal history, physical examination, 12-lead ECG
     - negative findings: eligible for competition
     - positive findings: further examinations (echo, stress test, 24-h Holter, cardiac MRI, angio/EMB, EPS)
       - no evidence of cardiovascular disease: eligible for competition
       - diagnosis of cardiovascular disease: management according to established protocols
Criteria for a Positive History, Physical Examination, and 12-Lead Electrocardiogram at Pre-participation Screening

**Family History**
Close relative(s) with premature myocardial infarction or sudden death at <50 years
Family history of cardiomyopathy, coronary artery disease, Marfan syndrome, long QT syndrome, severe arrhythmias, or other disabling cardiovascular diseases

**Personal History**
Syncope or near-syncope
Exertional chest pain or discomfort
Shortness of breath or fatigue out of proportion to the degree of physical effort
Palpitations or irregular heartbeat

**Physical Examination**
Musculoskeletal and ocular features suggestive of Marfan syndrome
Diminished and delayed femoral artery pulses
Mid- to late systolic clicks
Abnormal second heart sound (single or widely split and fixed with respiration)
Heart murmurs (systolic grade 2/6 and any diastolic)
Irregular heart rhythm
Brachial blood pressure 140/90 mm Hg on more than 1 reading

**Electrocardiogram**
Left atrial enlargement: negative portion of the P wave in lead V1 0.1 mV in depth and 0.04 s in duration
Right atrial enlargement: peaked P wave in leads II and III or V1 0.25 mV in amplitude
Frontal-plane QRS axis deviation: right +120° or left –30° to –90°
Increased voltage: amplitude of R or S wave in a standard lead 2 mV, S wave in lead V1 or V2 3 mV, or R wave in lead V5 or V6 3 mV
Abnormal Q waves 0.04 s in duration or 25% of the height of the ensuing R wave, or QS pattern in 2 leads
Right or left bundle-branch block with QRS duration 0.12 s
R or R’ wave in lead V1 0.5 mV in amplitude and R:S ratio 1
ST-segment depression or T-wave flattening or inversion in 2 leads
Prolongation of heart rate corrected QT interval >0.44 s in men and >0.46 in women
Premature ventricular beats or more severe ventricular arrhythmia
Supraventricular tachycardia, atrial flutter, or atrial fibrillation
Ventricular preexcitation: short PR interval (<0.12 s) with or without delta wave
First-degree (PR 0.21 s, not shortening with hyperventilation), second-degree, or third-degree atrioventricular block

Carrodo D et al Trends in Sudden Cardiovascular Death in Young Competitive Athletes After Implementation of a Preparticipation Screening Program  JAMA. 2006;296:1593-1601
Trends in Sudden Cardiovascular Death in Young Competitive Athletes After Implementation of a Preparticipation Screening Program

Domenico Corrado, MD, PhD
Cristina Basco, MD, PhD
Andrea Pavei, MD
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Marinello Torre, MD
Corrado Thiene, MD

The majority of young athletes who suddenly die previously have unrecognised cardiovascular disease.1,2 Cardiovascular symptoms have been consistently implicated in the leading cause of cardiac arrest in young competitive athletes with hypertrophic cardiomyopathy accounting for more than one third of all fatal cases in the United States.3,4 and approximately eight percent in cardiac arrest in young competitive athletes with hypertrophic cardiomyopathy for approximately one fourth of all fatal cases in Italy.5,6

Medical evaluation of athletic populations before competition allows the identification of asymptomatic athletes with potentially lethal cardiovascular abnormalities and prevents cardiac deaths due to disqualification of athletes from competitive sports.7,8 Italian law mandates that every participant engaged in competitive sports activity undergo a clinical examination and obtain eligibility.9 Accordingly, a myocardial systematic screening program was launched in Italy in 1982.10,11 This program is performed using an echocardiogram.12 The EFCA has shown to be effective in identifying athletes with hypertrophic cardiomyopathy and in

Conclusions. The incidence of sudden cardiovascular death in young competitive athletes substantially declined in the Veneto region of Italy since the introduction of a national systematic screening. Mortality reduction was predominantly due to the decrease incidence of sudden death from cardiomyopathies that paralleled the increasing identification of athletes with cardiomyopathies at preparticipation screening.

Trends in Sudden Cardiovascular Death in Young Competitive Athletes After Implementation of a Preparticipation Screening Program

During the study period, the annual incidence of sudden cardiovascular death decreased by 89% in screened athletes (P for trend < .001). In contrast, the incidence rate of sudden cardiovascular death did not demonstrate consistent changes over time in unscreened nonathletes.

ECG has a low specificity as a screening test due to the high frequency of changes that occur in association with the normal physiologic adaptations to training

- Sinus bradycardia, 1st degree AV block, LVH, J point and ST elevation, peaked T waves, T wave inversion are common

- Approximately 25% of athletes will have ECG patterns that are sufficiently unusual to warrant additional evaluation

Maron BJ et al. Circulation; Estes et al. JCE 2001
### Cardiovascular Conditions Associated with Sudden Cardiac Death in Athletes

<table>
<thead>
<tr>
<th>Condition</th>
<th>Condition</th>
</tr>
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<tbody>
<tr>
<td>Coronary artery disease</td>
<td>Pulmonary embolus</td>
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<tr>
<td>HCM</td>
<td>Arteriovenous malformation</td>
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<tr>
<td>Wolff-Parkinson-White syndrome</td>
<td>Berry aneurysm</td>
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<tr>
<td>Arrhythmogenic right ventricular dysplasia</td>
<td>Conduction system abnormalities</td>
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<td>Mitral valve prolapse</td>
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<td>Commotio Cordis</td>
<td>Myocardial bridge</td>
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<tr>
<td>Left ventricular hypertrophy</td>
<td>Coronary aneurysm</td>
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<td>Idiopathic ventricular fibrillation</td>
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<tr>
<td></td>
<td>Dilated cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td>Cerebral embolus</td>
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</tbody>
</table>
Causes of SCD in Athletes (<40 years)

- Congenital coronary anomalies (19%)
- HCM (36%)
- Mildly increased cardiac mass (10%)
- Ruptured aorta 5%
- Tunnelled LAD 5%
- Aortic stenosis 4%
- Myocarditis 3%
- Dilated cardiomyopathy 3%
- ARVC 3%
- MVP 2%
- CAD 2%
- Other 6%

Sudden Death in the Athlete
Bethesda and NASPE Guidelines for Participation/Restriction by Arrhythmia

**Supraventricular Arrhythmias**
- No restrictions after successful medical therapy or RFA

**Ventricular Arrhythmias**
- PVC- No restrictions if no SHD
- NSVT- No restrictions if no SHD
- Sustained VT- No restrictions in no SHD and successful RFA

Zipes, JACC, 1994; Estes NAM et al. JCE, 2001
Sudden Death in the Athlete
Guidelines for Athletic Participation By Cardiac Disease

Low intensity participation

CAD
HOCM
ARVD
IDCM
LQTS

No restrictions 3 months after successful therapy

RVOT VT
LVOT VT

Estes NAM et al. JCE, 2001 Zipes 2005
Syncope in the Athlete

- Syncope and presyncope in the athlete present a unique clinical challenge
- While syncope can result from benign, it can recur, cause serious trauma, place others at risk, and portent risk for SCD
- The cause is frequently difficult to define despite comprehensive evaluation
- If no cause for syncope is determined, restriction from athletic may be required
- Risk varies by age, gender, nature of the sport, underlying structural heart disease, and etiology of the syncope
- Current guidelines for restriction are based on expert opinion rather than prospective studies or data

Zipes D et al 36th Bethesda Conference Arrhythmias JACC 2005:45
Goldschlager, N et al Etiologic Considerations in the patient with syncope and an apparently normal heart Arch Int Med 2003:163:151-162
Syncope in the Athlete

Athletes with syncope or near syncope should not participate in sports where there is the likelihood of even a momentary loss of consciousness may be hazardous the cause has been determined and treated, if necessary.

Zipes D et al 36th Bethesda Conference Arrhythmias JACC 2005:45
Syncope in the Athlete
Lessons Learned

- Late access to medical care
- Minimization of symptoms
- Pitfalls of VIP treatment
- Compliance
- MD Shopping
- Communication
“You don’t win the Tour de France on mineral water” (Jacques Anquetil -- 5 time winner)

Cycling deaths
1896 European Competition
1960 Olympics
1967 Tour de France
Common Product Names and Alternative Names for Ephedra

**Supplement Ephedra**

*(Chinese ephedra)*

*(Mongolian ephedra)*

*(American ephedra)*

**Other Names**

**Ephedrine**

**Epitonin**

**Ma huang**

**coa ma huang**

**Mu zei mu huang**

**Natural Ecstasy**

**Pinellia**

**Popotillo**

**Sea grape**

**Sida cordifolia**

**Yellow astringent**

**Yellow horse**

**Zhong ma huang**

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**Adverse Cardiovascular Events Temporarily Associated With Ma Huang, an Herbal Source of Ephedrine**

**David Namensky, MD; Mark S. Line, MD; Matthias K. Homberg, MD; Robert C. Price, BS; Theodore C. Tsimba, MD; Paul J. Wang, MD; and N. A. Mark Estes III, MD**

- **Objective:** To evaluate possible cardiovascular toxic effects associated with use of dietary supplements containing ma huang, an herbal source of ephedrine.
- **Methods:** We reviewed the comprehensive database Adverse Reaction Monitoring System of the Food and Drug Administration, which included clinical records, case reports, and survey reports related to ma huang use. The study outcome was cardiovascular mortality.

**Results:** From 1995 to 1997, 978 cases of possible cardiovascular toxicity were reported to the Food and Drug Administration. In 37 patients (22 women and 15 men) NOS 25 were aged ≤50 years; 53 (16) were reported to have experienced sudden death. Among patients performed in 7 of the 11 patients who experienced sudden death showed a normal heart. In the majority other characteristics A and ephedrine was reported to be within the manufacturers' dosing guidelines.

**Conclusion:** Analysis of the 37 patients indicates the following findings: (1) no known use is temporally related to stroke, myocardial infarction, and sudden death. (2) Unifactorial heart or vascular disease is not a prerequisite for the underlying event adverse event, and (3) the systo-vascular toxic effects associated with ma huang were not limited to women patients. Although the pathogenesis of adverse events of ma huang remains incompletely defined, available observational and circumstantial evidence indicates that use of the substance may be associated with serious medical complications.

For editorial comment, see page 7.

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Adapted from: National Center for Complementary and Alternative Medicine; “Professional’s Handbook of Complementary and Alternative Medicines” (Springhouse, PA; Springhouse, 1999) and online sources.
Sudden Death in the Youth

Performance Enhancing Drugs

- Anabolic Steroids
- Androstenedione
- Ma Huang
- Creatine
- Growth Hormone
- Erythropoeitin
- Diuretics

- Amphetamines
- Cocaine
- Alcohol
- Marijuana
- Tobacco
- Corticosteroids
- Antidepressants

AED Termination of VF
Survival vs. Time to Defibrillation

- Ischemic arrest
- Commotio cordis
- HCM

Survival vs. Time to Defibrillation (minutes)
<table>
<thead>
<tr>
<th>Report</th>
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<th>AED</th>
<th>P Value</th>
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<td>15 *</td>
<td>29 *</td>
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*# of survivors
Effectiveness of Automated External Defibrillators in High Schools in Greater Boston

Hannah England, BA, Caitlin Hoffman, BS, Thomas Hodgman, BA, Sushil Singh, MD, Munther Homoud, MD, Jonathan Weinstock, MD, Mark Link, MD, and N.A. Mark Estes III, MD

A program using a strategy of donating a single automatic external defibrillator to 35 schools in the Boston area resulted in compliance with American Heart Association guidelines on automatic external defibrillator placement and training and 2 successful resuscitations from sudden cardiac arrest. Participating schools indicated a high degree of satisfaction with the program. ©2005 by Excerpta Medica Inc. (Am J Cardiol 2005;95:1484-1486)

Suscitation (CPR) classes taught in the schools. Respondents were asked to rank a series of statements on a scale from 1 to 5, with 1 meaning “completely disagree” and 5 meaning “completely agree.” The questionnaire also included a section concerning the director’s overall levels of satisfaction with the program.

Urban and suburban high schools that participated in the program had 466 to 4,280 students. The average number of schools in the districts was 9.64 ± 7.69, with 10.03 ± 7.74 school buildings. The school sys-
PAD Trial Location of Cardiac Arrest

PAD Investigators The Public Access to Defibrillation Study NEJM 2004;637-645
Task Force 10: Automated External Defibrillators

Robert J. Myerburg, MD, FACC, Chair
N. A. Mark Estes III, MD, FACC, John M. Fontaine, MD, FACC, Mark S. Link, MD, FACC, Douglas P. Zipes, MD, MACC

GENERAL CONSIDERATIONS OF CARDIAC ARREST RISK AMONG ATHLETES

The incidence of out-of-hospital cardiac arrest among the general population of adults is 1 to 2 deaths per 1,000 subjects per year; a figure that represents 50% of all cardiovascular deaths (1). For the adolescent and young adult subgroups, the estimated incidence is 1 per 100,000 per year or less. Available data suggest that among the younger population, competitive athletes account for a disproportionately higher-risk subset, compared to the general population in a comparable age group (2–4). In addition, among the adult population performing conditioning activities in health clubs, the probability of cardiac arrest during exertion appears higher than the expected rate for comparable groups generally, especially among persons who exercise despite being habitually sedentary (5).

Within the subgroup of the population age 35 years and older, coronary heart disease accounts for approximately 80% of all sudden cardiac deaths (SCDs), with the cardiomyopathies accounting for another 10% to 15%. In the younger age groups, hypertrophic cardiomyopathy, anom-
AED Athletic Events- Recommendations

- AEDs should be available at educational facilities that have competitive athletic programs (including intramural sports and conditioning classes), stadiums, arenas, and training sites, with trained responders identified among the permanent staff.

- Devices should be deployed so as to provide a response time of less than 5 min.

- The initial response to a suspected or identified cardiac arrest should be to contact emergency medical services (e.g., 9-1-1), followed immediately by, or concomitant with, initiating CPR and deploying the AED.
Automated External Defibrillators in the Public Domain: Am I Ready to Use One?

N.A. Mark Estes III, MD

1. Activate AED.
2. Apply pads.
3. AED analyzes heart rhythm.

An AED is used on a victim of sudden cardiac arrest. After 911 has been called, the AED is turned on (1). The adhesive pads are then applied (2). The AED automatically analyzes the cardiac rhythm (3). If a life-threatening cardiac rhythm disturbance is present, a voice prompt from the AED advises that a button be pushed to deliver a shock (4).
State and Federal Polices
Legal Protection for Owners, Users, Medical Directors
Court Opinions

As evidence-based medicine has defined the clinical benefits of AED use, public policy, laws, funding programs, and court decisions have served the societal interest of promoting use of AEDs by minimizing legal liability.

The Automated External Defibrillator
Clinical Benefits and Legal Liability

Hannah England, BA
Paul S. Weinberg, JD
N. A. Mark Estes III, MD

Sudden cardiac arrest is the most common cause of death in the United States, accounting for an estimated 350,000 deaths annually, and is a leading cause of disability and health care costs. 1 Life-threatening cardiac arrhythmias such as ventricular tachycardia or ventricular fibrillation usually cause sudden cardiac arrest.2,3 Early defibrillation of ventricular tachycardia or ventricular fibrillation is necessary to resuscitate individuals with cardiac arrest, and survival depends directly on the time to defibrillation. Automated external defibrillators (AEDs) reduce the time to defibrillation and have improved survival rates.4 Athough clinical benefits of AEDs are established, individuals, institutions, and organizations implementing AED programs have faced a seemingly complex and evolving legal framework. However, compliance with relevant regulations minimizes legal risks of AED overdose in use, or malpractice cases.5,6 Health care professionals should be aware of the clinical benefits of AED programs and strategies for risk management.

Evidence Supporting AED Use
In an effort to improve survival from cardiac arrest, the American Heart Association has promoted the Chain of Survival concept, describing a sequence of prehospital and hospital interventions that result in improved survival after sudden cardiac arrest.7,8 These interventions include prompt access to emergency medical services by calling 911, prompt cardiopulmonary resuscitation, early defibrillation when indicated, and early initiation of advanced cardiac care. Early defibrillation has emerged as the most important intervention with survival increasing by 10% with each minute of delay in defibrillation.9

Multiple studies and meta-analyses10,11 have demonstrated that early defibrillation increases survival for individuals with sudden cardiac arrest. Despite this evidence, many communities continue to have poor survival rates because of long response times of emergency personnel and delays in delivering definitive therapy with defibrillation.12 To address these concerns, the concept of public access to defibrillation has been promoted to expand the use of an immediately available defibrillator for minimally trained first responders such as police officers, firefighters, security guards, flight attendants, and trained laypersons.13-24 In some small studies, AEDs were safely and successfully operated by such guardians who performed defibrillation in 90 seconds in a simulated resuscitation. In the same study, trained paramedics performed defibrillation in 67 seconds.25 Another small study of AED use in airports, 13 of 16 individuals with cardiac arrest due to ventricular fibrillation were alive and neurologically intact at 1 year26 and 6 of the 11 successful resuscitations were travelers who did not have formal AED training.27

The Public Access Defibrillation Trial demonstrated that trained laypersons using AEDs safely and effectively provide early defibrillation.28 In this prospective randomized trial, 993 communities were randomized to cardiopulmonary resuscitation training with response by emergency personnel or trained laypersons using an AED. Survival in the AED groups was nearly 26% greater of 129 cardiac arrests in the cardiopulmonary resuscitation plus AED communities, 29 patients survived. Of the 129 cardiac arrests in the communities trained only in cardiopulmonary resuscitation, 13 survived. In 21.5 months of follow-up, there were no adverse events related to AED use. No patient received an inappropriate shock or failed to receive a needed shock. The Public Access Defibrillation Trial demonstrated that training and equipping laypersons within a structured response system increases the number of survivors after out-of-hospital cardiac arrest in public locations, and that trained laypersons can use the AED safely and effectively.29 Based on their proven benefit, AEDs are increasingly being used in public and private locations.30

Federal Policies and Legal Considerations
Federal laws provide the framework for limiting liability for AED ownership, oversight, and use.31-33 Until recently, expansion of AED programs has been hampered by largely unfounded concerns regarding legal liability.34-36 To address liability concerns, states and federal Good Samaritan legislation has been developed specifically to protect responders using AEDs.34,36 Good Samaritan legislation refers to statutes that provide immunity from claims

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AEDs in the NCAA

% Institutions with AEDs

- Div IA: 97%
- Div IAA: 88%
- Div IAAA: 84%
- Div II: 77%
- Div III: 81%
Almost all young athletes dying suddenly have underlying heart disease—males are at greater risk than females. In older patients underlying CAD is the most common cause.

Current screening techniques lack sensitivity and specificity for detecting athletes at risk for sudden death. Evaluation of standardized screening programs with tracking of long-term outcomes is needed.

Syncope in an individual with heart disease indicates a high risk for SCD. Comprehensive evaluation is warranted in the athlete with syncope.
Sudden Cardiac Death in Athlete

Conclusions

- Performance enhancing substances are widely used and may contribute to risk of death in athletes. Educational and enforcement programs are needed for performance enhancing and illicit drugs.

- AEDs may be effective in acute treatment of ventricular arrhythmias in athletes. Further studies are needed to assess the efficacy and cost of making AEDs available at all athletic events.

- Further basic, clinical and epidemiologic research is needed to develop cost-effective strategies to predict and prevent SCD in the athlete.
Case Report

- 14 year old healthy boy.
- During football he is hit in anterior chest.
- Patient immediately states that he is dizzy and then loses consciousness.
- CPR begins within 1 minute, and EMS arrives at 6 minutes.
Initial Electrocardiogram
Definitions and Background

**Commotio Cordis**

- Concussion of the heart
- *Commotio cordis*-disturbed or agitated heart motion (*Latin*)
- Chinese Martial Art Dim Mak (*death touch*) precordial blow results in sudden death in one’s opponent
- Sudden death from chest wall impact
- At autopsy, no pathologic abnormalities.
Collapse and Arrhythmias

- Instantaneous collapse in one half
- In the others a brief period of consciousness with lightheadedness
- Initial rhythms are generally ventricular fibrillation
- Post resuscitation ECGs show ST elevations
Commotio
Demographic Profile 217 Cases

Maron, Link, Estes 2009
Animal Studies-Our Protocol

- Juvenile anesthetized male swine placed prone in a sling.
- Baseball propelled at 30 mph.
- Release of the object was timed so that the impact could be adjusted according to the cardiac cycle.
T-wave Impact

Impact

Lead 1

Lead 2

Lead 3

AVR

AVL

AVF

LV (mmHg)

0

120

Time in Seconds

AN EXPERIMENTAL MODEL OF SUDDEN DEATH DUE TO LOW-ENERGY CHEST WALL IMPACT (COMMISSUROCARDIAE)

Mark S. Lee, M.D., Peter H. West, M.D., Nantia G. Paneras, M.D., Samuel Rabinovitz, M.D.,
James E. Usdin, M.D., Max Young, M.D., Mark A. Vintenburg, B.S., Brian A. Vintenburg, B.S.,
Gerard A. Minei, M.D., Barry J. Martin, M.D., and N.A. Minei, M.D.
Lacrosse Chest Wall Protection

Maron, Link, Estes  in press 2009
Possible Variables Important in Commotio Cordis

- Timing (examined)
- Energy of impact (examined)
- Location of impact (examined)
- Hardness of impact object (examined)
- LVP in commotio (examined)
- Role of ANS (examined)
- Role of $K_{ATP}$ Channel (examined)
- Efficacy of chest wall protectors/ safety balls
- Efficacy of defibrillation (examined)
Left Ventricular Pressure In Commotio Model

![Graph showing Left Ventricular Pressure over time with a spike at the impact point.](image)
Energy: Peak LVP/VF

Predicted Probability of VF by Peak LV Pressure

VF Probability

Peak LV Pressure (in mm Hg)
T-wave Impact

Link, Circulation, 1999
Commotio AEDs

Link et al
JACC 2002
Commotio AEDs

Graph showing survival rates over time in ventricular fibrillation.

Link et al
JCE 2002
Commotio Cordis

Maron and Estes NEJM 2010
Commotio Cordis Conclusions

- In humans, the clinical scenario is produced by low energy chest wall impact in an area overlying the heart
- Young males are the most susceptible to commotio cordis
- The initial rhythm, when documented, is most often ventricular fibrillation
- Survivors have had ST elevation on EKGs
Commotio Cordis Conclusions

- Low energy trauma during a 30 ms window on the upslope of the T-wave causes ventricular fibrillation in juvenile swine
- The VF produced is immediate and not preceded by PVC’s, ST elevations or other EKG abnormalities
- The site of impact must directly overlie the cardiac silhouette
- Changes in LV pressure may mediate VF
Commotio Cordis Conclusions

- Activation of the $\mathbf{K^+_\text{ATP}}$ channel may be the cause of the ventricular fibrillation seen with chest wall impact
- Softer than standard baseballs (safety balls) decrease the incidence of commotio cordis
- Currently available chest wall protectors are inadequate to protect against sudden death
- Immediate defibrillation can result in improved survival
- Further research is needed to identify methods of reducing the risk of death from commotio cordis
Sudden Cardiac Arrest in Athletes and Commotio Cordis

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