

6. Myerburg RJ. Sudden cardiac death: exploring the limits of our knowledge. *J Cardiovasc Electrophysiol* 2001;12:369-81.
7. Cobb LA, Weaver WD, Fahrenbruch CE, Hallstrom AP, Copass MK. Community-based interventions for sudden cardiac death: impact, limitations, and changes. *Circulation* 1992;85:198-102.
8. Mosesso VN Jr., Davis EA, Auble TE, Paris PM, Yealy DM. Use of automated external defibrillators by police officers for treatment of out-of-hospital cardiac arrest. *Ann Emerg Med* 1998;32:200-7.
9. White RD, Hankins DG, Bugliosi TF. Seven years' experience with early defibrillation by police and paramedics in an emergency medical services system. *Resuscitation* 1998;39:145-51.
10. Kette F, Sbrojavacca R, Rellini G, et al. Epidemiology and survival rate of out-of-hospital cardiac arrest in north-east Italy: the F.A.C.S. study: Friuli Venezia Giulia Cardiac Arrest Cooperative Study. *Resuscitation* 1998;36:153-9.
11. Newman MM, Mosesso VN Jr., Ornato JP, et al. Law Enforcement Agency Defibrillation (LEA-D): position statement and best practices recommendations from the National Center for Early Defibrillation. *Prehosp Emerg Care* 2002;6:346-7.
12. Myerburg RJ, Fenster J, Velez M, et al. Impact of community-wide police car deployment of automated external defibrillators on survival from out-of-hospital cardiac arrest. *Circulation* 2002;106:1058-64.
13. Capucci A, Aschieri D, Piepoli MF, Bardy GH, Iconomu E, Arvedi M. Tripling survival from sudden cardiac arrest via early defibrillation without traditional education in cardiopulmonary resuscitation. *Circulation* 2002;106:1065-70.
14. O'Rourke MF, Donaldson E, Geddes JS. An airline cardiac arrest program. *Circulation* 1997;96:2849-53.
15. Page RL, Joglar JA, Kowal RC, et al. Use of automated external defibrillators by a U.S. airline. *N Engl J Med* 2000;343:1210-6.
16. Valenzuela TD, Roe DJ, Nichol G, Clark LL, Spaite DW, Hardman RG. Outcomes of rapid defibrillation by security officers after cardiac arrest in casinos. *N Engl J Med* 2000;343:1206-9.
17. Caffrey SL, Willoughby PJ, Pepe PE, Becker LB. Public use of automated external defibrillators. *N Engl J Med* 2002;347:1242-7.
18. Zipes DP. President's page: the neighborhood health watch program: Save A Victim Everywhere (SAVE). *J Am Coll Cardiol* 2001;37:2004-5.
19. Holmberg M, Holmberg S, Herlitz J. Incidence, duration and survival of ventricular fibrillation in out-of-hospital cardiac arrest patients in Sweden. *Resuscitation* 2000;44:7-17.
20. Nichol G, Hallstrom AP, Kerber R, et al. American Heart Association report on the Second Public Access Defibrillation Conference, April 17-19, 1997. *Circulation* 1998;97:1309-14.
21. Marengo JP, Wang PJ, Link MS, Homoud MK, Estes NA III. Improving survival from sudden cardiac arrest: the role of the automated external defibrillator. *JAMA* 2001;285:1193-200.
22. Hazinski MF, Markenson D, Neish S, et al. Response to cardiac arrest and selected life-threatening medical emergencies: the medical emergency response plan for schools: a statement for healthcare providers, policymakers, school administrators, and community leaders. *Circulation* 2004;109:278-91.
23. Wik L, Hansen TB, Fylling F, et al. Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation: a randomized trial. *JAMA* 2003;289:1389-95.

#### Appendix 1. Author Relationships With Industry and Others

Name	Consultant	Research Grant	Scientific Advisory Board	Stock Holder	Expert Witness Testimony
Dr. N. A. Mark Estes III	<ul style="list-style-type: none"> <li>• Guidant</li> <li>• Medtronic</li> </ul>	<ul style="list-style-type: none"> <li>• Guidant</li> <li>• Medtronic</li> </ul>	<ul style="list-style-type: none"> <li>• Guidant (Executive Committee)</li> </ul>	None	None
Dr. John M. Fontaine	None	None	None	None	None
Dr. Mark S. Link	None	<ul style="list-style-type: none"> <li>• Guidant</li> <li>• Medtronic</li> </ul>	None	None	None
Dr. Robert J. Myerburg	<ul style="list-style-type: none"> <li>• Guidant</li> <li>• Procter &amp; Gamble</li> </ul>	None	<ul style="list-style-type: none"> <li>• Procter &amp; Gamble</li> <li>• Reliant Pharmaceutical</li> </ul>	None	<ul style="list-style-type: none"> <li>• 2000, Defense, Lewis vs. Mudge</li> <li>• 2002, Defense, Weiner vs. Vitello</li> <li>• 2005, Defense, Ephedra Multi-District Litigation</li> </ul>
Dr. Douglas P. Zipes	<ul style="list-style-type: none"> <li>• Cardiofocus</li> <li>• Janssen</li> <li>• Medtronic</li> </ul>	<ul style="list-style-type: none"> <li>• Medtronic</li> </ul>	<ul style="list-style-type: none"> <li>• Medtronic</li> </ul>	<ul style="list-style-type: none"> <li>• MVMD</li> </ul>	<ul style="list-style-type: none"> <li>• 1996, Defense, Knapp vs. Northwestern</li> </ul>

## Task Force 11: Commotio Cordis

Barry J. Maron, MD, FACC, *Chair*

N. A. Mark Estes III, MD, FACC, Mark S. Link, MD, FACC

### GENERAL CONSIDERATIONS

Sudden and unexpected deaths of young athletes are most frequently the consequence of unsuspected cardiovascular diseases (1). However, participants in organized sports are also subject to another risk for sudden death that occurs in the

absence of cardiovascular disease—namely, blunt, non-penetrating, and usually innocent-appearing chest blows, triggering ventricular fibrillation unassociated with structural damage to the ribs, sternum, or heart itself (*commotio cordis*) (2,3). Although the precise incidence during competitive

and recreational sports is unknown, commotio cordis events may be a more frequent cause of sudden death than previously believed, and also more common than many of the cardiovascular diseases that cause these catastrophes (1).

Knowledge of the demographics and clinical profile of commotio cordis is based largely on information from the U.S. Registry (Minneapolis, Minnesota) (2,3). Precordial blows that cause commotio cordis usually are not perceived to be unusual for the sport or activity involved, nor of sufficient magnitude to trigger arrhythmic sudden death. Although reported at a wide range of ages (3 months to 50 years), commotio cordis has a predilection for children and adolescents (mean age 13 years) probably because the young characteristically have narrow, pliable chest walls that facilitate transmission of energy from the chest impact to the myocardium.

Commotio cordis occurs in a wide variety of sports, but most commonly youth baseball (and softball), ice hockey, football, and lacrosse, with death often caused by projectiles that are implements of the competition. Although blows are typically of low energy, projectiles may strike the precordium with a range of velocities—paradoxically, and most commonly, with only modest force such as a pitched baseball striking a batter at 30 to 40 mph, but also with high-velocity blows from hockey pucks or lacrosse balls (up to an estimated 90 mph), and frequently with bodily contact to the precordium such as by karate blows or collisions between outfielders tracking a baseball in-flight. Collapse can be instantaneous or preceded by brief periods of consciousness and physical activity. Despite a structurally normal heart, survival from commotio cordis is uncommon (i.e., only 15%). However, survival from commotio cordis has been reported with increasing frequency associated with prompt cardiopulmonary resuscitation and defibrillation (3). Survivors of commotio cordis should undergo a complete cardiac evaluation including electrocardiogram (ECG), Holter ambulatory monitoring, echocardiogram, and possibly cardiac catheterization to exclude underlying structural cardiac abnormalities.

Also, many deaths from commotio cordis occur around the home or on the playground in informal activities related or unrelated to recreational sports (often involving close relatives) in which the chest impact is delivered in an innocent fashion; for example, such events have occurred as a result of light blows during playful “shadow boxing” or as a remedy for hiccups (3). Unfortunately, some commotio cordis events have even triggered criminal convictions for manslaughter or murder (4).

A swine model that replicates commotio cordis has provided important insights into the mechanisms responsible for the devastating electrophysiologic consequences of these precordial blows (5–7). Determinants of ventricular fibrillation following a chest blow include impact delivered at a wide range of velocities directly over the heart, and timing within a narrow 15-to-30-ms window just prior to the T-wave peak during the vulnerable phase of repolariza-

tion (representing only 1% of the cardiac cycle) (5–8). The requirement for such an exquisite confluence of circumstances may largely explain the uncommon occurrence of commotio cordis.

In addition, spontaneously aborted commotio cordis events may occasionally result from blows sustained during the QRS complex (depolarization), which trigger transient complete heart block or nonsustained polymorphic ventricular tachycardia in the animal model (5). Basic cellular mechanisms responsible for commotio cordis are incompletely understood, although selective activation of  $K^+$  ATP channels may play a pivotal role (9).

Several strategies for prevention of commotio cordis events, including innovations in the design of sports equipment, have been considered. Softer-than-normal (“safety”) baseballs reduce risk for ventricular fibrillation under laboratory conditions (5,7). Although such projectiles do not provide absolute protection from sudden death on the baseball field (3), nevertheless there is sufficient reason to encourage the use of such baseballs in organized play at appropriate ages (10). Chest barriers with proven efficacy for youth sports (e.g., baseball, lacrosse, and hockey) have not yet been developed, and many of the commercially available products offer no or only incomplete protection against provoked arrhythmias (3,11). The continued reports of commotio cordis events during organized and recreational sports emphasize the importance of more timely resuscitative efforts, including immediate access to automated external defibrillators (AEDs) (12,13), and also development of preventive strategies including design of effective chest barriers (11).

#### *Recommendations:*

- 1. Age-appropriate safety baseballs are recommended for use in children up to 13 years of age.**
- 2. Although chest wall protectors may prevent traumatic injury in goalies and baseball catchers, insufficient evidence is available to recommend universal use of commercially available chest barriers for all participants in sports, specifically to prevent commotio cordis events.**
- 3. AEDs should be available within 5 min after participant collapse at sporting events.**
- 4. Survivors of a commotio cordis with ventricular fibrillation (or a presumed aborted event without documented ventricular fibrillation) should undergo a thorough cardiac evaluation, including at least 12-lead ECG, ambulatory Holter monitoring, and echocardiogram. Standard electrophysiologic testing and an implantable cardioverter-defibrillator are not usually recommended.**
- 5. Because data are lacking with regard to the susceptibility for recurrent events, eligibility for returning to competitive sports in survivors is at present a decision left to individual clinical judgment.**

**TASK FORCE 11 REFERENCES**

1. Maron BJ. Sudden death in young athletes. *N Engl J Med* 2003;349:1064-75.
2. Maron BJ, Poliac LC, Kaplan JA, Mueller FO. Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. *N Engl J Med* 1995;333:337-42.
3. Maron BJ, Gohman TE, Kyle SB, Estes NAM III, Link MS. Clinical profile and spectrum of commotio cordis. *JAMA* 2002;287:1142-6.
4. Maron BJ, Mitten MJ, Greene BC. Criminal consequences of commotio cordis. *Am J Cardiol* 2002;89:210-3.
5. Link MS, Wang PJ, Pandian NG, et al. An experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). *N Engl J Med* 1998;338:1805-11.
6. Link MS, Maron BJ, VanderBrink BA, et al. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. *J Am Coll Cardiol* 2001;37:649-54.
7. Link MS, Maron BJ, Wang PJ, Pandian NG, VanderBrink BA, Estes NAM III. Reduced risk of sudden death from chest wall blows (commotio cordis) with safety baseballs. *Pediatrics* 2002;109:873-7.
8. Link MS, Maron BJ, Wang PJ. Upper and lower limits of vulnerability to sudden arrhythmic death with chest wall impact (commotio cordis). *J Am Coll Cardiol* 2003;41:99-104.
9. Link MS, Wang PJ, VanderBrink BA, et al. Selective activation of the K(+)(ATP) channel is a mechanism by which sudden death is produced by low-energy chest-wall impact (commotio cordis). *Circulation* 1999;100:413-8.
10. Kyle SB. Youth Baseball Protective Equipment Project Final Report. United States Consumer Product Safety Commission. Washington, DC: 1996.
11. Weinstock J, Maron BJ, Song C, Mane PP, Estes NAM III, Link MS. Commercially available chest wall protectors fail to prevent ventricular fibrillation induced by chest wall impact (commotio cordis) (abstr). *Heart Rhythm* 2004;1:692.
12. Strasburger JF, Maron BJ. Images in clinical medicine: commotio cordis. *N Engl J Med* 2002;347:1248.
13. Link MS, Maron BJ, Stickney RE, et al. Automated external defibrillator arrhythmia detection in a model of cardiac arrest due to commotio cordis. *J Cardiovasc Electrophysiol* 2003;14:83-7.

**Appendix 1. Author Relationships With Industry and Others**

Name	Consultant	Research Grant	Scientific Advisory Board	Expert Witness Testimony
Dr. N. A. Mark Estes III	<ul style="list-style-type: none"> <li>• Guidant</li> <li>• Medtronic</li> </ul>	<ul style="list-style-type: none"> <li>• Guidant</li> <li>• Medtronic</li> </ul>	<ul style="list-style-type: none"> <li>• Guidant (Executive Committee)</li> </ul>	None
Dr. Mark S. Link	None	<ul style="list-style-type: none"> <li>• Guidant</li> <li>• Medtronic</li> </ul>	None	None
Dr. Barry J. Maron	None	<ul style="list-style-type: none"> <li>• Medtronic</li> </ul>	None	<ul style="list-style-type: none"> <li>• 1996, Defense, Knapp vs. Northwestern</li> </ul>

## Task Force 12: Legal Aspects of the 36th Bethesda Conference Recommendations

Matthew J. Mitten, JD, *Chair*  
 Barry J. Maron, MD, FACC, Douglas P. Zipes, MD, MACC

**GENERAL CONSIDERATIONS**

In 1994, when the 26th Bethesda Conference recommendations were formulated, no court had yet considered whether an athlete with a cardiovascular abnormality could be involuntarily excluded from a competitive sport if physicians disagreed in their participation recommendations (1-3). However, new data have subsequently become available, and several highly visible cases involving the sudden deaths of elite competitive athletes (4,5) have brought medical-legal and liability considerations into prominent focus. A 1996 lawsuit brought by a student-athlete claiming the legal right to play intercollegiate basketball contrary to a university team physician's medical recommendation has established a developing legal framework for medical decisions regarding the eligibility or disqualification of trained athletes with cardiovascular disease to participate in competitive sports. In this case, Knapp vs. Northwestern University (6,7), a federal appellate court recognized the appropriateness of a physician's reliance on current consensus medical guidelines when making a participation recommendation for an athlete with a cardiovas-

cular abnormality. Consequently, judicial precedent now provides some guidance regarding the role of the present 36th Bethesda Conference recommendations in resolving legal issues relating to athletic participation disputes.

In the Knapp case, the court upheld Northwestern University's legal right to accept its team physician's recommendation, which was consistent with the then-current 26th Bethesda Conference guidelines, to medically disqualify a student-athlete from playing college basketball (6). As a high school senior, Nicholas Knapp suffered sudden cardiac arrest while playing in an informal basketball game, which required cardiopulmonary resuscitation and defibrillation to restore sinus rhythm. Thereafter, he had a cardioverter-defibrillator (ICD) implanted. He resumed playing recreational basketball without any subsequent cardiovascular events, and three cardiologists medically cleared him to play college basketball.

Knapp had received a full athletic scholarship at Northwestern University to play intercollegiate basketball. While Northwestern honored Knapp's scholarship, it barred him