Cardiovascular disease may be responsible for a quarter of diving fatalities, but there are few studies on the cardiovascular complications of this activity. In contrast, there is a rich literature on land-based, exercise-related cardiac events. These studies document that exercise can increase the risk of acute cardiac events, but that absolute risk is small for healthy individuals. There are no proven strategies to reduce exercise-related cardiac events and consequently no proven strategies that could be confidently applied to diving. Nevertheless, requiring a pre-diving medical evaluation and clearance for those with known cardiac disease, training dive personnel to elicit possible cardiac prodromal symptoms, and frequent emergency training for diving supervisors are prudent approaches to this problem.

Introduction
Cardiovascular disease (CVD) has been identified as a possible contributing event to 156 of 590 (26 percent) diving-related fatalities (Denoble, Caruso et al. 2008). There are few studies on the cardiovascular complications of diving, however, and many of those available deal with issues related to patent foramen ovale and systemic emboli.

A PubMed search performed in February 2010 using the terms “diving” and “cardiac events” yielded 25 publications of which only one (Denoble, Caruso et al. 2008) was directly relevant. In contrast, a search using the terms “exercise” and “cardiac events” yielded 3,051 publications. Consequently, the present manuscript will discuss nondiving exercise-related cardiovascular complications in an attempt to suggest strategies that may be useful in reducing diving-related cardiac events. This extrapolation of exercise-related cardiovascular events to diving is necessary given the paucity of data on cardiac events during diving, but it should be recognized that diving includes additional cardiovascular stressors such as cold exposure, changes in pulmonary compliance and vascular pressures, and centralization of blood volumes that do not attend routine exercise.

Habitual physical activity has repetitively been associated with a reduced incidence of atherosclerotic cardiovascular disease events (Powell et al. 1987; Fletcher et al. 1996; Lee, Paffenbarger 2001; Thompson, Buchner, et al. 2003). Nevertheless, vigorous physical activity can acutely, albeit transiently, increase the risk of acute myocardial infarction (AMI) (Giri et al. 1999; Mittleman et al. 1993) and sudden cardiac death (SCD) in young (Corrado et al. 2003) and older (Siscovick et al. 1984; Albert et al. 2000) susceptible individuals.

Exercise-related cardiac events are usually defined as those occurring during, or within one hour of the cessation of sports participation in young subjects or vigorous physical exertion in older individuals (Rai, Thompson 2009).

Vigorous exercise is usually defined as exercise requiring ≥6 metabolic equivalents (METs). Six METs is approximately equal to an oxygen uptake ($VO_2$) of 21 ml/kg/minute or the energy required for activities such as jogging. This is an absolute...
workrate, however, and the cardiovascular stress of exercise is more closely related to the VO₂ requirements of exercise relative to the individual’s maximal exercise capacity. Consequently, exercise workrates under 6 METs may still place considerable stress on the cardiovascular systems of unfit and older individuals.

**Causes of Exertion-Related Cardiovascular Events**

Exercise-associated acute cardiac events require some pathological substrate and do not occur in normal hearts. This substrate may be structural, such as hypertrophic cardiomyopathy or acute coronary thrombosis, or cellular, such as inherited ion channel disease. The causes of exercise-related deaths are different in young individuals, defined as ages younger than 30 to 40 years (Van Camp et al. 1995; Maron, Shirani et al. 1996; Corrado et al. 1990), and older subjects. The pathological findings in young individuals suffering an exercise-related cardiac death are primarily inherited or congenital cardiovascular abnormalities (Table 1) (Van Camp et al. 1995; Maron, Shirani et al. 1996; Corrado et al. 1990). Myocarditis is also associated with exercise-related deaths in young individuals. Ventricular fibrillation is assumed to be the immediate cause of death except in Marfan syndrome, where aortic rupture is responsible.

Coronary artery disease (CAD) is the cause of most exercise-related cardiac events in older individuals (Thompson, Stern, et al. 1979; Burke, Farb, et al. 1999). Acute coronary artery plaque rupture or erosion producing thrombotic occlusion is the proximate cause of these events in previously asymptomatic individuals (Burke et al. 1999), whereas either plaque disruption or an arrhythmia produced by ischemia or myocardial scar is the presumed proximate cause of such events in individuals with previously diagnosed CAD (Cobb, Weaver 1986).

The mechanism by which vigorous exercise destabilizes coronary plaques is not defined, but increased arterial wall stress from increases in heart rate and blood pressure, exercise-induced coronary artery spasm in diseased artery segments (Gordon et al. 1989) and increased flexing of atherosclerotic epicardial coronary arteries (Black, Black, Gensini 1975) have been suggested as etiologic factors.

**The Frequency of Exercise-Related Acute Cardiovascular Events**

The frequency of exercise-related cardiovascular events varies with the prevalence of diagnosed or occult CVD in the study population. A 27-year registry of young U.S. athletes who died or survived cardiac arrest identified only 1,339 cases of which 1,049 were attributed to definite (n=690) or probable (n=359) cardiac disease (Maron, Doerer et al. 2009). The majority (61 percent) of events occurred in the final collection period between 1994 and 2002 and averaged only 66 cardiovascular deaths per year, or an estimated 0.6 deaths per 100,000 person-years (1 death per year per 166,666 athletes). Only 11 percent of these events occurred in women.

Others using some of the same data sources have published similar death rates for young U.S. athletes of only one per 133,000 men and 769,000 women, respectively (Van Camp et al. 1995). These later estimates include all sports-related nontraumatic deaths and are not restricted to cardiovascular events. These numbers are extremely low and consistent with the general safety of athletic participation in young individuals.

The incidence of cardiac death associated with vigorous physical exertion is considerably higher in adults, especially adult males, given their much higher prevalence of occult CAD. We estimated an incidence of only one death per 396,000 person-hours of jogging, or one death per year for every 7,620 joggers.
The explanation for the lower rates of exercise SCD in adult women is probably related to the delayed development of CAD in women and less participation in vigorous exercise among older females.

Both studies have wide confidence limits because the rates were calculated using only 10 (Thompson et al. 1982) and nine (Siscovick et al. 1984) exercise-related deaths. All victims in both studies were men. These studies were also performed almost 30 years ago, but more recent studies confirm the low absolute risk of exercise-related SCD and the low rate among women. The absolute incidence of sudden death during and up to 30 minutes after vigorous exertion in the Physicians’ Health Study was only one death per 1.51 million episodes of exertion (Albert et al. 2000). In the Nurses’ Health Study there was only one death per 36.5 million hours of moderate to vigorous exertion (Whang et al. 2006). The explanation for the lower rates of exercise SCD in adult women is probably related to the delayed development of CAD in women and less participation in vigorous exercise among older females.
Vigorous exercise can also precipitate AMI (Willich et al. 1993; Mittleman et al. 1993; Giri et al. 1999), but the absolute incidence of exercise-related MI in the general population has not to our knowledge been carefully determined. Approximately 11 percent of individuals with AMI treated with primary angioplasty suffered an exertion-related event (Giri et al. 1999). Also, using the observation that exercise MI is 6.75 more frequent than exercise-related SCD (Siscovick et al. 1991) and confidence limits for SCD (Thompson et al. 1982), one can estimate that the 95 percent confidence limits for an exercise-related MI is one per year for every 593 to 3,852 apparently healthy middle-aged joggers. This number should be viewed cautiously, however, given that the SCD rate is based on only 10 cases (Thompson et al. 1982).

The incidence of exercise-related cardiovascular complications is considerably higher among those patients participating in supervised exercise-based cardiac rehabilitation programs (Table 2). An analysis of four reports estimates one cardiac arrest per 116,906 patient-hours, one myocardial infarction per 219,970 patient-hours, one fatality per 752,365 patient-hours and one major complication per 81,670 patient-hours of rehab participation (Van Camp, Peterson 1986; Digenio et al. 1991; Franklin et al. 1998; Vongvanich, Paul-Labrador, Merz 1996). This fatality rate applies only to medically supervised programs that are equipped to handle emergencies, since the death rate would be sixfold higher without the successful management of cardiac arrest. Cardiac rehabilitation patients also undergo a medical evaluation that often includes exercise stress testing, and they are under staff supervision. Consequently, it is likely that the rate of events, and especially fatal events, is considerably higher among patients with CAD exercising in unsupervised situations.

Table 2: Summary of contemporary exercise-based cardiac rehabilitation program complication rates

<table>
<thead>
<tr>
<th>Investigator</th>
<th>Year</th>
<th>Hours</th>
<th>Cardiac arrest</th>
<th>Myocardial infarction</th>
<th>Fatal events</th>
<th>Major complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Van Camp/Peterson (1986)</td>
<td>1980-1984</td>
<td>2,351,916</td>
<td>1/111,996†</td>
<td>1/293,990</td>
<td>1/783,972</td>
<td>1/81,101</td>
</tr>
<tr>
<td>Digenio et al. (1991)</td>
<td>1982-1988</td>
<td>480,000</td>
<td>1/120,000‡</td>
<td></td>
<td>1/160,000</td>
<td>1/120,000</td>
</tr>
<tr>
<td>Average</td>
<td></td>
<td>1/116,906</td>
<td>1/219,970</td>
<td>1/752,365</td>
<td>1/81,670</td>
<td></td>
</tr>
</tbody>
</table>

* MI and cardiac arrest; †Fatal 14%; §Fatal 75%; $Fatal 0%. Reproduced with permission (Thompson et al. 2007).

This discussion on the incidence of exercise-related events has several messages important to diving. The high putative rate of nonfatal exercise-related AMI provided above is of concern, because some of these nonfatal events could become a “disabling event,” leading to a fatality because of pain or panic during submersion. Indeed, among 590 diving fatalities, 159 — or 27 percent — had a possible cardiac event leading to disability and ultimately drowning (Denoble, Pollock et al. 2008). Also, the group most at risk for an exercise-related event are by far those with established CVD. Nevertheless, the cardiac complication rate of diving seems very low. Using the reported seven-year diving fatality rate of 16.4 per 100,000 persons (Denoble, Pollock et al. 2008) and the estimate that 26 percent are related to cardiac disease (Denoble, Caruso et al. 2008), one can calculate an annual rate of only 0.6 per 100,000 divers.
It is also important to note that several of the above studies have documented a decrease in both AMI (Willich et al. 1993; Mittleman et al. 1993; Giri et al. 1999) and SCD (Siscovick et al. 1984) in the most habitually active subjects, meaning that exercise-related cardiac events are more frequent among those performing unaccustomed physical exertion.

**Strategies to Reduce Exercise-Related Cardiovascular Events**

A variety of strategies have been suggested to prevent exercise-related acute cardiovascular events (Thompson et al. 2007), but none have been sufficiently studied to evaluate their efficacy or to be required practice for health providers. Also, any prevention strategy must consider the rarity of exercise-related cardiac events in ostensibly healthy individuals and the cost of falsely positive screening strategies when these are applied to low-risk populations.

The medical screening of young athletes prior to organized sports participation is endorsed by multiple organizations including the American Heart Association (AHA) (Maron, Thompson et al. 1996; Maron et al. 1998; Maron et al. 2007) and the study group on sports cardiology of the European Society of Cardiology (Corrado et al. 2005). There is considerable and sometimes vitriolic (Myerburg, Vetter 2007) debate as to what constitutes effective screening. The European group recommends routine echocardiograms (ECGs) as part of the screening paradigm, whereas the AHA does not. A recent cost-effectiveness analysis of both approaches favored ECG use (Wheeler et al. 2010) but will probably not be readily accepted by both sides since it estimated the annual death rates in young athletes as 1 death per 42,000 participants, a value higher than most other estimates’ symptoms prior to death.

Preparticipation screening and the restriction of higher-risk subjects could also be applied to adults. Most exercise-related cardiac events in adults are due to atherosclerotic CVD (ASCVD). The population risk of ASCVD can be predicted using tools such as the Framingham Risk Score, which predicts the 10-year risk of SCD and AMI. Potential participants with a specific score could be identified and excluded. The problem with this approach is that ASCVD is prevalent among lower-risk subjects. Also, extremely high-risk subjects are only a small part of the total population. Consequently, the largest absolute number of acute events occurs not in the highest-risk subjects but in the moderate- and lower-risk groups. Excluding the highest-risk group would likely have little effect on the total number of deaths.

Many physicians routinely perform preparticipation exercise testing in adults prior to vigorous exercise. Both the American College of Cardiology/American Heart Association (ACC/AHA) Guidelines on Exercise Testing (Gibbons et al. 2003) and the American College of Sports Medicine (ACSM) (ACSM 2005) recommend exercise testing for adults with increased risk of CVD, generally based on age or the presence of two or more CVD risk factors. Both groups recommend such testing in persons with diabetes primarily because diabetics may not experience angina induced by exercise. In contrast, the U.S. Preventive Services Task Force (USPSTF 2004) states that there is insufficient evidence to determine the benefits and harm of exercise stress testing prior to an exercise training program (USPSTF 2004). Furthermore, a recent decision analysis recommended against exercise testing at all levels of risk because the numbers of exercise-related deaths prevented by exercise testing were fewer than the deaths produced by medical intervention (Lahav, Leshno, Brezis 2009).
In addition to such concerns, there are two largely underappreciated limitations to routine exercise testing. First, a positive exercise test result, by either ECG or imaging criteria, requires a flow-limiting coronary lesion. Most acute cardiac events in previously asymptomatic subjects are due to vulnerable plaque disruption, and so a normal exercise test does not exclude a nonflow-limiting, vulnerable plaque. Second, a positive exercise test in asymptomatic subjects is a better predictor of subsequent angina than of the events of most concern, AMI and SCD (McHenry et al. 1984). This is possibly because asymptomatic, flow-limiting lesions prompt the development of collateral vessels that limit the severity of any acute cardiac event.

Exclusion of subjects with established cardiovascular disease from sports participation or activities such as diving would likely be the single most effective technique to decrease exercise-related cardiac events. A careful case-controlled study identified a prior history of cardiovascular disease as the single most discriminating factor differentiating the 57 cases of exercise-related cardiac events from the controls (Van Teeffelen et al. 2009). The odds ratio in multivariate analysis suggests that those with prior CVD had a 32-fold higher (95 percent CI=7 to 143) risk of suffering a cardiac event. Since the risk of an exercise-related cardiac event is so much higher in patients with diagnosed CVD, excluding such participants, or requiring more extensive testing in this group, may reduce cardiac complications.

Educating patients, exercise attendants and physicians about possible cardiac prodromal symptoms and their need for prompt evaluation has been advocated to reduce exercise-related cardiac events. In the case-controlled study mentioned above (Van Teeffelen et al. 2009), fatigue or flulike symptoms over the past month increased the risk of a cardiac event by 12- and 13-fold respectively (95 percent CIs: 1.2-118 and 1.4-131). Among adults who died from CAD during exertion, 50 percent of joggers (Thompson et al. 1979), 75 percent of squash players (Northcote, Flannigan, Ballantyne 1986) and 81 percent of distance runners (Noakes, Opie, Rose 1984) had probable cardiac symptoms prior to death (Table 3). Many of these symptoms were assumed to be gastrointestinal in origin and were not reported to medical personnel.

### Table 3: Prodromal symptoms reported by 45 subjects within one week of their sudden death

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Number of reports</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest pain/angina</td>
<td>15</td>
</tr>
<tr>
<td>Increasing fatigue</td>
<td>12</td>
</tr>
<tr>
<td>Indigestion/heartburn/gastrointestinal symptoms</td>
<td>10</td>
</tr>
<tr>
<td>Excessive breathlessness</td>
<td>6</td>
</tr>
<tr>
<td>Ear or neck pain</td>
<td>5</td>
</tr>
<tr>
<td>Vague malaise</td>
<td>5</td>
</tr>
<tr>
<td>Upper respiratory tract infection</td>
<td>4</td>
</tr>
<tr>
<td>Dizziness/palpitations</td>
<td>3</td>
</tr>
<tr>
<td>Severe headache</td>
<td>2</td>
</tr>
</tbody>
</table>

Note: Adapted from Northcote, Flannigan, Ballantyne 1986 and reproduced with permission (Thompson et al. 2007).
Also, among 159 diving fatalities attributed to cardiac disease, 10 percent of the victims had reported dyspnea, fatigue, chest pain, distress or some illness prior to their fatal dive (Denoble, Caruso et al. 2008). The specificity of such complaints is probably poor, given the prevalence of nonspecific complaints in the general population. Nevertheless, training exercise professionals to solicit new complaints and to refer subjects for appropriate evaluation seems prudent but, as with most of these interventions, is untested.

There is excellent evidence that training for cardiovascular emergencies reduces exercise-related cardiac deaths. This is demonstrated by the observation that the rates of cardiac arrest in cardiac rehabilitation programs is considerably lower than the fatality rate. Prompt resuscitation is not readily applicable to diving because most deaths occur during the dive, but it is reasonable that diving facilities be encouraged to establish and maintain emergency plans, periodic drills and training to address cardiac emergencies.

**Suggested Approach to the Problem of Cardiac Events with Diving**

Any requirements of diving participants should be cognizant of the rarity of exercise-related events in asymptomatic individuals, the poor predictive accuracy of most cardiac testing techniques and the absence of data that any method, except medical supervision, actually improves prognosis. Nevertheless, it does seem prudent to do the following:

1. Require medical clearance for individuals with known cardiac disease since this is the highest-risk subgroup. The group requiring medical clearance could be expanded to those providing positive responses to questionnaires designed for preparticipation evaluation, but this would greatly increase the participant burden without documented benefit.

2. Train diving personnel to elicit possible cardiac prodromal symptoms, and restrict those individuals with such symptoms until cleared by a medical professional.

3. Require cardiac emergency training, and scheduled drills for diving supervisors.

**References**


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