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evaluation of screening guidelines. A recent study by Roberts and Stovitz (1) reported a remarkably low rate of sudden cardiac death (SCD) in Minnesota high school athletes on the basis of analysis of catastrophic insurance claims. The low rate of SCD was linked to a statewide pre-participation screening process involving a standardized history and physical examination conducted every 3 years (1). However, critical examination of the study methodology raises important concerns about the validity of the study findings and conclusions. This letter draws attention to major flaws and presents an alternative incidence of sudden cardiac arrest (SCA) in Minnesota high school athletes on the basis of a search of public media reports.

The authors searched catastrophic insurance claims over a 19-year period (1993 to 2012) to identify cases of SCD during high school athletic practices and games. The reported incidence of SCD in Minnesota high school athletes was 0.24 per 100,000 athlete-years over 19 years and 0.11 per 100,000 athlete-years over the last decade (1). The methodology is analogous to a widely cited 1998 study that reported an incidence of SCD in Minnesota high school athletes of 0.46 per 100,000 athlete-years (2). The study does not include cases resulting in death outside of an official high school–sponsored sporting event such as unofficial training sessions or participation with club/select teams. The report briefly mentions cases of SCA in which the persons were saved but implies they were not athletes or at least the cases did not happen during school-sponsored events.

The authors associate a standardized pre-participation evaluation with the low rate of SCD, a conclusion further promoted by a corresponding editorial (3). However, the study provides no data as to the results of the screening evaluations performed or the cardiac conditions identified. If the rate of cardiovascular events in Minnesota high school athletes is truly as low as reported, one possible conclusion is that no cardiovascular screening of any type is actually needed.

We conducted a review of public media reports by searching the Parent Heart Watch database over the last 10 years of the study period in which the authors report only a single case of SCD. The Parent Heart Watch database tracks cases of SCA through systematic Internet search protocols. Each case was reviewed to assess the circumstances of the event and confirm participation on a Minnesota high school athletic team.

Between 2003 and 2012, public media reports identified 13 cases of SCA in Minnesota high school athletes (all in males), including 6 cases of SCD and 7 cases of SCA in student athletes who survived. Roberts and Stovitz (1) document 917,069 unduplicated high school athletes in Minnesota from 2003 to 2012. Thus, the incidence of all SCA in Minnesota high school athletes is 1.4 per 100,000 athlete-years, and the incidence of SCD 0.65 per 100,000 athlete-years. Of the 6 deaths documented in media reports, only 1 would have been eligible for death benefits from an insurance claim, yet 4 of 6 deaths occurred during sports participation. An alarming 46% of cases (6 of 13) occurred in boys' basketball. The incidence of SCA in Minnesota high school boys' basketball is ~4.7 per 100,000 athlete-years.

Search of catastrophic insurance claims is not an accurate method to conduct death surveillance in athletes. The Minnesota pre-participation evaluation did not prevent at minimum 13 cases of SCA between 2003 and 2012. There remains no evidence that a screening program on the basis of history and physical examination alone is effective in identifying athletes with at-risk conditions or in preventing SCA. Reports examining only death rates will underestimate the incidence of life-threatening cardiovascular events and falsely assume that current screening strategies are effective. To suggest that catastrophic insurance claims are a reliable measure of incidence in support of current screening strategies is unsubstantiated. Although many questions and challenges to more intensive cardiovascular screening in athletes exist, we must recognize that scientific limitations and misinterpretations have perpetuated an underestimate of SCA in athletes and perhaps impeded progress toward the evaluation and implementation of more effective preventive programs.

***Jonathan A. Drezner, MD†**
Kimberly G. Harmon, MD†
Joseph C. Marek, MD‡

*Department of Family Medicine
Box 354060
University of Washington
Seattle, Washington 98195
E-mail: jdrezner@uw.edu

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From the †Department of Family Medicine, University of Washington, Seattle, Washington; and the ‡Midwest Heart Foundation, Oakbrook Terrace, Illinois

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Letters to the Editor

Ankle-Brachial Index in Patients With Nonvalvular Atrial Fibrillation

Violi et al. (1) are to be commended for their large study on the prevalence of subclinical peripheral artery disease (PAD) among patients with nonvalvular atrial fibrillation. Indeed, the ankle-brachial index (ABI) enables the detection of a substantial subset of individuals with asymptomatic (or with atypical symptoms of) PAD in diverse populations, and beyond its diagnostic interest, a low ABI is predictive for stroke, as highlighted recently in a meta-analysis (2). Violi et al. (1) reported an even higher than expected 21% prevalence of PAD detected by an ABI $\leq$0.90, almost doubling the proportion of patients with "vascular disease" as defined in the $CHADS_2$-VASc (congestive heart failure [or left ventricular systolic dysfunction]; hypertension [blood pressure consistently >140/90 mm Hg or on hypertension medication]; age $\geq$75 years; diabetes mellitus; previous stroke, transient ischemic attack, or thromboembolism; vascular disease [e.g., peripheral artery disease, myocardial infarction, aortic plaque]; age 65 to 74 years; sex

category [male or female]) score (i.e., myocardial infarction, complex aortic plaque, and PAD), advocated by the European Society of Cardiology guidelines on the management of atrial fibrillation, to assess the risk of stroke (3). However, we think that prior to proposing the measurement of ABI in patients with nonvalvular atrial fibrillation, several issues should be discussed.

First, the main interest of the $CHADS_2$-VASc score is to determine whether oral anticoagulant therapy (OAC) is necessary. The European Society of Cardiology guidelines recommend OAC when $CHADS_2$-VASc is $\geq$2 and prefer OAC to aspirin when the score is 1 (3). Hence, the practical interest of ABI measurement would be limited to those with a $CHADS_2$-VASc score of 0 or 1 in order to detect "missed" cases of PAD, which would lead to increasing the score by 1 point and ultimately to revising the anticoagulation strategy. In their study, Violi et al. (1) report high rates of diabetes, hypertension, and history of myocardial infarction or stroke among those with an ABI $\leq$0.90. All those variables lead to a higher $CHADS_2$-VASc score, so that the prevalence of ABI $\leq$0.90 among those with a current $CHADS_2$-VASc score at 0 or 1 should be reported to clarify its incremental value to change anticoagulation strategy in these low-risk patients.

Second, 10% of the study population had an ABI >1.40, and these patients have apparently not been adequately taken into consideration, because almost one-half of these patients do have underlying PAD, although the definite diagnosis would need further tests as the ABI measurement is impeded by calcified arteries (4).

Finally, the accuracy of the ABI measurement in case of irregular rhythm is unknown. It has been shown that the measurement of arm blood pressure in this situation is associated with considerable intra- and interobserver variability, and it is plausible to wonder at similar poor results when making the ratio of pressures measured in several limbs (5). The only way to moderate the level of inaccuracy is to advocate systematically repeated measurements and to avoid taking the crucial decision of OAC on the basis of a sole measurement of the ABI.

Once these issues are addressed, we agree with Violi et al. that a prospective study is necessary to assess the ability of the ABI to reclassify low-risk patients and increase the $CHADS_2$-VASc score discrimination index to predict stroke events in case of nonvalvular atrial fibrillation. Ultimately, a trial would be necessary to clarify the interest of OAC in patients with both low $CHADS_2$-VASc score and ABI.

*Victor Aboyans, MD, PhD
Philippe Lacroix, MD
Najmeddine Echahidi, MD
Dania Mohty, MD, PhD

*Department of Cardiology
Dupuytren University Hospital
2 Martin Luther King Avenue
Limoges 87042
France
E-mail: vaboyans@live.fr

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Reply

Ankle-Brachial Index in Patients With Nonvalvular Atrial Fibrillation

We thank Dr. Aboyans and colleagues for their comments on our paper (1) in which we reported a 21% prevalence of low ($\leq$0.90) ankle-brachial index (ABI) in a population suffering from nonvalvular atrial fibrillation. The inclusion of low ABI in the definition of vascular disease within the CHA_2DS_2-VASc (congestive heart failure [or left ventricular systolic dysfunction]; hypertension [blood pressure consistently >140/90 mm Hg or on hypertension medication]; age $\geq$75 years; diabetes mellitus; previous stroke, transient ischemic attack, or thromboembolism; vascular disease [e.g., peripheral artery disease, myocardial infarction, aortic plaque]; age 65 to 74 years; sex category [i.e., female]) score substantially modified the prevalence of vascular disease, which increased from 17.3% to 33%. This had a particular impact on the CHA_2DS_2-VASc score subclasses between 0 and 1, whereby the inclusion of a low ABI in the score resulted in a potential risk upgrading.

Thus, 9.5% of patients in classes 0 and 1 had a low ABI; of these, 20% and 80% were CHA_2DS_2-VASc classes 0 and 1, respectively. Among patients classified as CHA_2DS_2-VASc score 0, smoking was the only risk factor in 29% of patients; among those classified as CHA_2DS_2-VASc score 1, 43 (18%) were women, 34 (14%) aged from 65 to 74 years, 149 (63%) were hypertensive, 5 (2%) were diabetic, and 6 (3%) had vascular components of the "classic" CHA_2DS_2-VASc score.

The presence of an ABI $\geq$1.40 can be detected in atherosclerotic patients, particularly in those with diabetes and this usually reflects tibial artery calcification and is a predictor of cardiovascular disease and total mortality (2). An ABI $\geq$1.40 was detected in 10% of our nonvalvular atrial fibrillation patients. When patients with ABI $\geq$1.40 were compared with those with ABI >0.90 to 1.39 (Table 1), we found that women were more prevalent in the group of patients with normal ABI; conversely arterial hypertension and diabetes mellitus were more frequent in patients with ABI $\geq$1.40. Although the definite diagnosis of PAD would need further tests as the ABI measurement is confounded by calcified arteries, we will consider the possibility to evaluate the predictive power of this parameter in future survival analyses.

We planned this study in 2010 using the Doppler method for the determination of the ABI and organized a training meeting to reduce inaccuracy level. Training included demonstration of performance of an ABI in nonvalvular atrial