appropriate. The Student *t*-test or Mann-Whitney *U* test was used for the comparison of continuous variables, as appropriate. Data have been analyzed according to the intention-to-treat principle. Two-sided tests were used, and a p value <0.025 (using the Bonferroni adjustment, alpha level/number of comparison: 0.05/2) was the statistical significance level. MACE curves using the Kaplan-Meier methods were produced for treatment group and compared by the log-rank test. The software SPSS version 17.0 (SPSS Italia, Florence, Italy) was used for all statistical analyses.

One-year FU was complete in all but 5 of 240 patients (2 patients in the adenosine group, 1 patient in the nitroprusside group, and 2 patients in the placebo group). As in the main study, demographics, cardiovascular risk factors, ischemic times, Killip classes and discharge therapy did not differ significantly among treatment groups (p = NS, data not shown). Of note, compliance with therapy at FU was nearly 90% for all drugs and was similar among treatment groups (p = NS). Baseline end-diastolic and end-systolic left ventricular volumes were similar between adenosine-treated patients (105 ± 19 ml and 62 ± 15 ml, respectively) or nitroprusside-treated patients (107 ± 21 ml and 64 ± 16 ml, respectively) and saline-treated patients (104 ± 20 ml and 65 ± 15 ml, respectively), p = 0.59, p = 0.42, p = 0.48, p = 0.68, respectively), with no significant differences between adenosine- and nitroprusside-treated patients (p = 0.57 and p = 0.61, respectively). However, at 1-year echocardiographic FU, the left ventricular remodeling rate occurred less frequently in the adenosine-treated patients as compared with the placebo-treated patients (6% vs. 23%, p = 0.006), whereas it was similar between nitroprusside- and placebo-treated patients (18% vs. 23%, p = 0.43), and tended to be less frequent in the adenosine-treated patients as compared with the nitroprusside-treated patients (p = 0.07). The MACE rate at 1-year FU was significantly lower in the adenosine group (2 deaths, 1 myocardial infarction, 6 target-lesion revascularizations, 1 heart failure) as compared with the placebo group (4 deaths, 5 myocardial infarctions, 7 target-lesion revascularizations, 7 heart failures) (13% vs. 31%, p = 0.01), whereas it was similar between the nitroprusside (3 deaths, 4 myocardial infarctions, 7 target–lesion revascularizations, 3 heart failures) and the placebo groups (21% vs. 31%, p = 0.21) and between the adenosine and the nitroprusside groups (p = 0.21). Of note, hospitalization for heart failure tended to occur less frequently in the adenosine group as compared with the placebo group (1% vs. 9%, p = 0.06). Kaplan-Meier curves for MACE-free survival according to treatment allocation are shown in Figure 1.

In this study, we showed that high-dose intracoronary adenosine administration in patients with STEMI undergoing primary PCI is associated with improved clinical outcomes after 1 year, mainly because of a reduction in hospitalization for heart failure and less left ventricular negative remodeling (with a statistical power of 99% for adenosine vs. saline and 95% for nitroprusside vs. saline). Because this effect was borderline after 1 month (2), it suggests that the beneficial effects of adenosine on clinical outcomes need more time in order to be demonstrated, as adenosine may have an impact on the rate of left ventricular remodeling (4) after STEMI. The adenosine-related improvements of left ventricular remodeling and clinical outcome are probably due to its effects on MVO and infarct size. Indeed, both reductions of MVO and infarct size have been related to improved clinical outcome (1). Multiple cardioprotective effects of adenosine have been reported, including its potent direct vasodilatory effect on the coronary microcirculation, its anti-inflammatory properties against neutrophils, and its ability to inhibit platelet aggregation, to stimulate angiogenesis, and to mimic ischemic pre-conditioning, thus probably accounting for the beneficial effects observed in this trial (5). In particular, on the basis of these effects, left ventricular remodeling improvement may be related to a better healing process of a smaller infarct area in adenosine-treated patients (suggested also by the smaller enzymatic infarct size observed in the main study for adenosine-treated patients). This study is not powered, however, to demonstrate a mortality benefit, thus further studies should test larger populations for the impact of intracoronary adenosine after thrombus aspiration in STEMI on survival.

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Incidence of Sudden Cardiac Arrest in Minnesota High School Student Athletes

The Limitations of Catastrophic Insurance Claims

To the Editor: An accurate understanding of the incidence of cardiovascular events in athletes is central to the development and

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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.

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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