percent of these patients reported that symptoms of infection started within one week prior to the MI onset. Using the case-crossover analysis, the odds ratio of MI for infections one day prior to MI onset was 2.4 (95% CI: 1.7–3.4), compared to the seventh day prior to the onset.

Conclusion: Although external control data are not available, the finding that 17% of patients in this large database report an infection in the week prior to MI onset is compatible with the possibility that infection triggers MI. This finding coincides with recent studies linking infection and inflammation to atherosclerosis, supports the need for controlled studies of infection as a trigger.

1055-72 Cocaine Use as a Trigger of Acute Myocardial Infarction

M.A. Mittleman, D. Mintzer, M. Maclure, G.H. Tofler, J.B. Sherwood, J.E. Muller. *Beth Israel Deaconess Medical Center, Boston, MA; University of Kentucky Medical Center, Lexington, KY, USA*

Although anecdotal reports indicate that cocaine use can trigger acute myocardial infarction (MI) onset, there have been no controlled studies of the magnitude of the increased risk, nor the duration of the hazard period. We therefore collected data in the Onset Study to rigorously evaluate this association.

Between August 1989 and September 1996 we interviewed 3,946 patients with acute MI at 45 medical centers across the United States. Patients were interviewed an average of 4 days following MI onset. A self-matched case-crossover approach was used to evaluate the relative risk of MI onset following cocaine use.

Of the 3,946 patients interviewed, 38 (0.9%) reported cocaine use in the year prior to the onset of MI, and 9 reported use within the 60 minutes before the onset of their MI symptoms. Cocaine users were more likely to be male (87% vs 67%, $p = 0.01$), younger (44 ± 8 vs 61 ± 13 years, $p < 0.001$) and non-white (61% vs 11%, $p < 0.001$) compared with non-users. The risk of MI onset was elevated 23.7 fold (95% CI, 8.5 to 66.2) in the 60 minutes following cocaine use and rapidly returned to baseline beyond the first hour.

Conclusion: Cocaine use is associated with a large abrupt increase in the risk of acute MI in subjects who are otherwise at relatively low risk. Drug education campaigns ought to include information regarding the magnitude of this risk. This finding also suggests that studying the pathophysiologic changes produced by cocaine may provide insights into the mechanisms of triggering.

1055-73 Difficult to Control Neurally Mediated Syncope: Is it Familial

R. Ashraf, N.N. Kadri, T.T. Hee, G.B. Schaefer, S.M. Mohiuddin. *Creighton University, Omaha, Nebraska*

Familial tendency in difficult to control (DTC) neurally mediated syncope (NMS) has been described in sporadic forms. However pattern of inheritance, if present, has not yet been identified. Therefore, the charts of 35 patients (pt's) with DTC-NMS who were enrolled in a clinical study were reviewed retrospectively. DTC-NMS is defined as a pt with recurrent NMS that occurs at least once monthly, has a positive head up tilt that reproduced symptoms, and unresponsive to conventional therapy. 26/35 available and consenting pts were contacted by telephone to inquire the detailed family history of such disorder. The pedigrees of kindreds were constructed and analyzed by a clinical geneticist (GS).

Results: 14/26 pts with syncope/presyncope (average of 10 episodes/month) failed conventional treatment and had a family history of such disorder were identified. Ten pts had no family history of DTC-NMS up to three generations. One pt died and the other was adopted. Two pts were related as mother and son. 13 pedigrees were constructed. There were 46 affected kindreds with nearly equal male to female ratio (22/24 respectively). 33/46 affected individuals were among first degree relatives. Male to male transmission was noted in one pedigree, suggesting that this is not an X-linked trait. There was one instance of an affected mother transmitting the condition to two sons with different fathers. Incomplete penetrance (transmissions of the trait from a non-affected obligate gene carrier) was noted in three pedigrees.

Conclusion: Genetic analysis of the pedigrees from 13 kindreds suggests autosomal dominant inheritance with incomplete penetrance in patients with DTC-NMS.

1055-74 Safety Baseballs Reduce Ventricular Fibrillation and EKG Changes in a Biological Model of Commotio Cordis, Sudden Death From Low Energy Chest Wall Impact

M.S. Link, P.J. Wang, N. Pandian, M.-Y. Lee, B. Vanderbrink, E. Avelar, B.J. Maron, N.A.M. Estes, III. *Tufts-New England Medical Center, Boston, MA, USA*

Background: Commotio cordis is sudden death resulting from a strike to the chest with a low energy object (typically a baseball). The etiology is thought to be ventricular fibrillation (VF). There is uncertainty about whether softer than standard (safety) baseballs will reduce the risk of sudden death in these young athletes.

Methods: In a juvenile swine model, VF could be reproducibly induced by 30 mph baseball strikes occurring 15 to 30 ms prior to the peak of the T-wave. We impacted 24 animals during this vulnerable period of the cardiac cycle with up to 3 strikes with either a standard baseball or a safety baseball (designed for players aged 5 to 7 years).

Results: Significantly fewer episodes of VF were seen in the animals impacted with a safety baseball ($p = 0.03$). In the 12 animals impacted with a safety ball there were 2 episodes of VF with 27 strikes. In the 12 animals impacted with a standard baseball there were 8 episodes of VF with 23 strikes. In addition, there were significantly fewer episodes of ST elevation, and bundle branch block with a safety ball.

	Standard ball	Safety ball	P-value
Ventricular Fibrillation	8/23 (35%)	2/27 (7%)	0.03
Heart block	3/15 (20%)	1/25 (4%)	0.10
ST elevation	8/15 (53%)	4/25 (16%)	0.03
Bundle branch block	4/15 (27%)	0/25 (0%)	0.03

Conclusion: Safety baseballs decrease the risk of ventricular fibrillation in a swine model of low energy chest wall impact. These findings emphasize potential methods of reducing sudden death in the young athlete.

1055-75 Is Preparticipation Screening for Cardiovascular Disease Adequate in United States High Schools?

D.W. Glover, B.J. Maron. *St. Luke's Hospital, Kansas City, MO; Minneapolis Heart Institute Foundation, Minneapolis, MN, USA*

Background: Sudden death in young student-athletes due to unsuspected cardiovascular (CV) disease has heightened public interest in preparticipation screening.

Methods: To understand the limitations of screening for detecting important CV lesions, 51 state high school jurisdictions were contacted to determine their guidelines for implementation of screening.

Results: Of the 51, 9 (18%) have no recommended history/physical questionnaire to guide examiners. Of the remaining 42 states, only 7 (17%) had adequate forms when measured against 1996 American Heart Association guidelines. History forms showed relevant items were present in ~60% e.g., prior heart disease, murmur, dyspnea/chest pain, familial heart disease, or prior sports exclusion. Physical exam forms also showed high omission rates: ~20% had murmurs, irregular rhythm, blood pressure, Marfan stigmata. All states recommend physicians perform screening; however, 16 permit nurses/physician assistants and 11 provide for chiropractors.

Conclusions: Athletic screening currently in place in U.S. high schools to detect CV disease: 1) is highly dependent on history/physical exam forms that are frequently abbreviated/inadequate; 2) is implemented by various health care workers with different levels of expertise; 3) is severely limited in its power to detect lethal CV lesions. These observations represent an impetus to change/optimize athletic screening process.

1055-76 Reducing Exercise-related Sudden Cardiac Death Rates Among Recruits by Prevention of Exertional Heat Illness

J.A. Kark, J.W. Gardner, F.T. Ward. *Howard University Hospital, Washington, DC; Uniformed Services University of the Health Sciences, Bethesda, MD, USA*

Background: Two thirds of exercise-related death (ERD) of recruits are without preexisting disase: one third unexplained sudden cardiac death (U-SCD) and one third fatal exertional heat illness (EHI). Speculating that unrecognized EHI might cause U-SCD, deaths might be prevented by adjusting exercise intensity, rest cycles, and water intake hourly to the on-site wet bulb globe temperature index.

Methods: To test the effect of this intervention, we enumerated recruits, surveyed training practices, determined etiology for 96 ERDs from autopsy protocols, clinical records, eyewitness accounts, toxicology, and