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Editorial

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Sudden Cardiac Death is not Caused by Ischemia

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Sudden cardiac death (SCD) is a major health problem with upwards of 500,000 cardiovascular deaths in the United States due to this problem each year. The SCD patients referred to here are those apparently healthy persons who drop dead suddenly without experiencing any symptoms or able to call for help [1]. The cause is usually sudden onset of ventricular fibrillation (VF). Survival is very poor with most studies showing about 15% of successfully resuscitated patients leaving the hospital neurologically intact.

The presumed cause of this sudden collapse is myocardial ischemia because the majority of these patients have coronary atherosclerosis on post mortem examination [1]. However, ischemia takes time to develop and SCD occurs too quickly to be caused by ischemia [2]. In addition many patients do not go on to show evidence of AMI if they are resuscitated, and rarely is there evidence of AMI on post mortem exam [3]. Acute obstructive lesions in the coronary arteries are rare. Holter monitor exams worn by patients who suffer sudden onset of VF frequently show premature ventricular contractions (PVC's) and an increase in heart rate, but not ischemia immediately before the onset of VF [4].

Therefore, there is little evidence to support the proposition that SCD is caused by ischemia. There are no blood tests that are specific for ischemia. Elevated troponins indicate myocardial injury, but the cause of the injury is not necessarily ischemia. All of our tests for ischemia are indirect including the treadmill, nuclear scans, ECG's, etc and there is no way to state positively that this injury was caused by ischemia. There are other mechanisms to consider. Every day thousands of balloons are inflated in the coronary arteries creating ischemia on ECG monitoring during cardiac catheterization [5]. VF is a rare event during these procedures, and if it does occur it is immediately cardioverted and the cardiac catheterization procedure is not stopped, but is continued, often without a reoccurrence of further episodes. Specifically, whatever caused the VF has disappeared because VF does not reoccur.

SCD is caused by a disturbance of the conduction system (CS) of the heart and not by a disturbance of the coronary circulation. The CS is extremely stable, day in and day out, beating approximately 100,000 times per day under the various stresses of life in all types of situations. The CS is resistant to ischemia and injury associated with myocardial infarctions, even very large infarctions of the anterior ventricular wall. This is due primarily to its location in the left ventricular subendocardium [6]. The blood supply to the subendocardium comes from the left ventricular cavity. A potent insult is required to disturb the normal function of the CS and produce sudden VF. If the cause is not ischemia, what is the causative agent or process?

Atheromas are basically caseous abscesses with caseation defined as necrotic tissue with a high fat content. All necrotic tissue, including atheromas, contain potent toxins that can injure tissues, including the CS. These toxins include products of lipid metabolism, acids generated as part of cell necrosis, inflammatory cytokines, immune complexes, reactive oxygen species and hydroxyapatite [7]. These toxins are probably similar in potency to absolute alcohol used in the treatment of hypertrophic cardiomyopathy, which damages the CS producing various forms of heart block [8]. Plaque ruptures and the discharge of plaque toxins are of many types. Some are superficial erosions, while others are deep fissures into the necrotic core and others are ruptured plaques, including those that "shell out" completely. A shelled out plaque is probably the most dangerous because of the large amount toxins that is discharged into the coronary circulation [7].

In summary there is no direct evidence to prove SCD is caused by ischemia, other than these patients have coronary atherosclerosis of varying degrees. A potent toxin that circulates to the CS is the most plausible explanation and is most consistent with the observations outlined above. Immediate chest compression on in such a patient, before defibrillation, has been shown to improve the results of defibrillation. In theory, this initial compression of the heart may wash out some of the toxins and facilitate defibrillation. A toxic cause with quick wash out of the toxin from the CS could explain why VF does not immediately recur when cardioverted. Various toxins could be identified by sampling in the coronary sinus during cardiac catheterization or during the treatment of an acute coronary event. If the true toxins were known, perhaps antidotes could be administered immediately to SCD patients in an effort to neutralize these toxins as quickly as possible, and reduce or avoid the fracture of the coronary arteries associated with traditional CPR [9]. Further research will be necessary to fully define this problem and begin to make real inroads on the incidence and management of patients suffering SCD.

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