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HEART OF THE MATTER

Preventing a Tim Russert Event

am sure that many of you have been asked the same question that I have this summer. It typically occurred at a cocktail party, when a 50-something woman,

upon learning that I am a cardiologist, sidled up and motioned toward a portly gentleman hovering over the appetizers. "That's my husband. He saw his doctor last week and was told that his blood pressure was a little high, but that he shouldn't worry. They'll check him again next year. What should he do to prevent a heart attack like Tim Russert's?'

Mr. Russert's untimely death sent shivers through

millions of middle-aged men and their families. Here was a guy seemingly getting the best preventive care, yet he died from an acute myocardial infarction. Despite our efforts, sudden death remains the most common outcome of heart disease. It is estimated that at least a third of all heart attacks lead to sudden death, often the first expression of coronary heart disease. After resuscitation, patients often admit to symptoms they attributed to anything but a myocardial infarction.

BY SIDNEY

GOLDSTEIN, M.D.

Sudden death can occur as the first expression of coronary heart disease, in the setting of known coronary heart disease or in patients with advanced heart failure. Early evaluation, either by stress or by electrophysiologic testing or more sophisticated imaging with fast CT or MRI, can help in identifying patients at increased risk, but it provides little help in establishing timing of the mortality event.

It is likely that Mr. Russert's event occurred as a result of a plaque rupture and thrombus formation. The development of that plaque can be modified with statin

therapy—although little is known about the effect of statins on sudden death.

Acceleration of plaque formation can occur with a variety of stimuli, including cigarette smoking, hypertension, and diabetes. The aggressive treatment of hypertension—a major step on the road to acute MI and heart failure—is critical. I am convinced that calcium entry blockers, particularly dihydropyridines, are the most

effective treatment of hypertension. But since most patients with hypertension require more than one drug, β -blockers also are essential for preventing the long-term mortality and morbidity of hypertension, which include sudden death and heart failure. These drugs mitigate the adrenergic surge that occurs with an acute MI or ischemic stress. However, I admit that achieving adequate treatment of hypertension is one of the most difficult therapeutic challenges I face.

Sudden death as an expression of heart failure probably is related to a complex relation between interstitial fibrosis and increased circulating catecholamines leading to the development of micro reentry circuits that then degenerate into ventricular fibrillation. In patients with heart failure and those who have experienced a MI, β blockers significantly decrease the risk of

It is therefore distressing that in studies of implantable cardioverter defibrillators in heart failure, β -blockers are underutilized. Implantable cardioverter defibrillators do play an important role in the prevention of sudden death in heart failure, but as noted in previous columns, we still do not have a good understanding of which patients are best suited for ICD use.

Lastly, a comment about aspirin. Widely used in the general population, therapy with this ubiquitous drug has a paradoxical effect, as reported in the Physicians' Health Study (N. Engl. J. Med. 1989;321:129-35), by decreasing MI mortality but increasing sudden death. Guidance for aspirin therapy for primary prevention has been based in part on Framingham risk scores (N. Engl. J. Med. 2002;346:1468-74). In my opinion, aspirin has benefit in high-risk patients with ischemic heart disease and particularly in those patients who have experienced an acute coronary syndrome. But for primary prevention, the evidence is not very supportive for its use.

This leads us to the continued quandary posed by my cocktail party questioner. Unfortunately, it is almost impossible to predict the timing of sudden death, but we have been successful in decreasing the likelihood of experiencing an event in the unknown future. Its occurrence is a little like playing Russian roulette: Sometimes, the gun is loaded.

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Adverse Event **Reports Persist**

Interaction from page 1

Although the precise mechanism is not known, it is related to amiodarone's inhibition of the cytochrome P450 3A4 (CYP3A4) enzyme, which also metabolizes simvastatin, a HMG-CoA reductase inhibitor, according to

The FDA is advising that health care professionals consider prescribing another statin in patients who need more than 20 mg of simvastatin to reach their lipid goals and are either taking amiodarone or starting amiodarone

The alert also applies to the combination drug products that contain simvastatin: simvastatin and ezetimibe (marketed as Vytorin) and simvastatin and extended-release niacin (Simcor). Simvastatin alone is marketed as Zocor and is also available in generic formulations. Amiodarone, an antiarrhythmic drug approved for controlling lifethreatening recurrent ventricular arrhythmias, is marketed as Cordarone and Pacerone.

The prescribing information for amiodarone is also being revised to include this information. The FDA does not have any data on how changes in the amiodarone dose in patients also taking simvastatin affect their risk of rhabdomyolysis.

The full alert is available at www. fda.gov/medwatch/safety/2008/safety 08.htm#Simvastatin.

Adverse events associated with these drugs can be reported to the FDA's MedWatch program at 800-332-1088 or www.fda.gov/MedWatch/report.htm.



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