Alcohol and premature mortality: a causal and preventive factor

In most developed countries, heavy alcohol consumption is second only to cigarette smoking as the leading avoidable cause of all premature deaths and all cancer deaths[1]. Heavy alcohol consumption also contributes to premature death from traffic accidents, cirrhosis, violent crime, suicide, and industrial accidents. Alcoholism leads to acute toxicities, as well as chronic psychoses and a wide range of morbid conditions that make alcohol abuse a leading cause of admission to chronic care facilities. In addition, heavy drinkers are among those at highest risk of dying from cardiovascular disease.

Against this backdrop of clear harm, however, there is some evidence that consumption of small-to-moderate amounts of alcohol may confer protection against the development of coronary heart disease. Many epidemiological studies of primary and, more recently, secondary prevention have shown a J-shaped association between amount of alcohol consumed and total mortality. The benefits of light-to-moderate alcohol consumption appear to be mediated largely by a decrease in coronary mortality. This is most likely due to the antiatherogenic effects of alcohol-related increases in high density lipoprotein cholesterol[2] (HDL-C) but other mechanisms, including antithrombotic effects, have also been postulated.

Basic research has provided plausible mechanisms for benefits of small-to-moderate alcohol consumption. Furthermore, epidemiological studies — both case-control and, more recently, large prospective cohort studies — have consistently shown benefits on cardiovascular mortality among individuals who self-select to consume small-to-moderate amounts of alcohol. The fact that many different investigations using very different design strategies tend to reach a similar conclusion adds to the credibility that small amounts of alcohol, rather than other characteristics of the individuals who self-select to consume alcohol, decrease risks of coronary heart disease[3]. At the same time, basic research and epidemiological studies have also demonstrated that alcohol consumption, even in small amounts, can have some deleterious consequences, such as the development of cerebral haemorrhage, hypertension, and breast cancer.

In a recent large, prospective cohort study of secondary prevention[4] we found that men with previous myocardial infarction who consumed small-to-moderate amounts of alcohol had lower total mortality than men who never or rarely drank alcohol. While some have suggested that cardiovascular benefits are confined to those who drink red wine, it is far more plausible, based on the totality of evidence, that benefits accrue to those who consume beer, whiskey, or wine[5]. If red wine drinkers really do derive some extra benefit compared with those who drink beer, whiskey, or white wine, this may be due to other constituents in red wine or even a social class effect.

The complexity of alcohol’s metabolic, physiological, and psychological effects may preclude the general acceptance of policy statements for its use in either primary or secondary prevention of cardiovascular disease. Nonetheless, it is also reasonable for individual health care providers who may wish to do so to suggest to appropriate patients that they consider drinking small amounts of alcohol daily, but only as an adjunct — not as an alternative — to managing other coronary risks. Unfortunately, most patients might prefer prescription of alcohol to prescription of harmful lifestyles, such as the avoidance of smoking, an unhealthy diet, obesity, and physical inactivity[6]. But avoiding these harmful lifestyles has been proven beyond a reasonable doubt to reduce cardiovascular disease, and would yield far greater benefits than daily consumption of small amounts of alcohol, even in the absence of any deleterious consequences stemming from small-to-moderate alcohol use.

In conclusion, the totality of evidence suggests that the difference between consuming small amounts of alcohol each day and consuming larger amounts may be the difference between preventing and causing premature death.

C. H. HENNEKENS
University of Miami,
Miami, Florida, U.S.A.
Implantable cardioverter-defibrillators are clearly beneficial in patients who have suffered aborted sudden cardiac death or symptomatic and sustained ventricular tachycardia in the presence of impaired left ventricular dysfunction. For this patient cohort, implantable cardioverter-defibrillator implantation is now the treatment of first choice and unequivocally superior to antiarrhythmic drug treatment, as has been shown in AVID[1], CIDS[2] and CASH[3].

The role of implantable cardioverters in primary prevention of arrhythmic death has been much more controversial. It is well known that patients with non-sustained ventricular tachycardia, in the presence of coronary artery disease and especially after myocardial infarction, are at higher risk of sudden death. Until recently, however, it was unclear whether antiarrhythmic therapy provides any benefit. Some trials studying antiarrhythmic agents in post-myocardial infarction patients have even shown an increased mortality. The role of arrhythmia treatment, however, became clearer when the results of MADIT[4] (Multicenter Automated Defibrillator Trial) were announced at the NASPE meeting in May 1996. This trial, the results of which were eventually published at the end of 1996, included patients with coronary artery disease and depressed left ventricular function remote from myocardial infarction. These patients presented with asymptomatic (or only mildly symptomatic) non-sustained ventricular tachycardia with a mean length of nine beats. Patients were randomized to implantable cardioverter-defibrillator therapy or conventional treatment if a ventricular tachycardia was inducible during electrophysiological study and could not be suppressed by procainamide. MADIT was the first randomized study to demonstrate a benefit from implantable cardioverter-defibrillator implantation. In fact, it showed a more than 50% reduction in total mortality in the implantable cardioverter-defibrillator group.

In the meantime, the inclusion criteria for MADIT is now a Class 1 ACC/AHA implantable cardioverter-defibrillator indication[5]. However, MADIT was heavily criticized for several points, among them an imbalance in the beta-blocker treatment in favour of the implantable cardioverter-defibrillator group. For this reason, and because other trials failed to show a benefit from prophylactic implantable cardioverter-defibrillator implantation, the results of MADIT have not readily been adopted into everyday clinical practice and MADIT has led only to a very modest increase in implantable cardioverter-defibrillator implants.

At the last meeting of the American College of Cardiology (ACC), Dr Alfred Buxton from the Temple University School of Medicine in Philadelphia, U.S.A. presented the results of the MUSTT trial (Multicenter Unsustained Tachycardia Trial) which was conducted in the United States and Canada[6]. MUSTT was not designed to test implantable cardioverter-defibrillator therapy vs drug therapy but to determine whether antiarrhythmic therapy, guided by electrophysiology, can decrease the risk of arrhythmic death and cardiac arrest in patients with non-sustained ventricular tachycardia, left ventricular dysfunction (ejection fraction <40%), and coronary artery disease. The patients included in MUSTT were similar to MADIT patients, but presented with markedly shorter runs of non-sustained ventricular tachycardia. About 35% of the enrolled 2202 patients had inducible ventricular tachycardia. Of these, 704 were randomized to conservative

References